CHAPTER 3
ORAL DISEASES, INFECTIONS AND CRANIOFACIAL DISORDERS
(6 CE Hours)

Learning objectives
- Explain new research and findings regarding the connection between oral health and health.
- Learn about the six major dental diseases.
- Discuss the connection between heart disease, diabetes and oral infections.
- Review some effective disease preventative measures.
- Learn about craniofacial disorders.
- List factors affecting future dental health care practices.

Introduction
The realization that oral health can have a significant impact on the overall health and well-being of the nation’s population has become a major issue in the world of science and research. Realizing the gains that have been made in disease prevention, while acknowledging that there are populations that suffer disproportionately from oral health problems, the purpose of this course is to help “define, describe and evaluate the interaction between oral health and health and well-being [quality of life], through the life span in the context of changes in society.” Key elements to be addressed are the determinants of health and disease, with a primary focus on prevention and “producing health” rather than “restoring health”; a description of the burden of oral diseases and disorders in the nation; and the evidence for actions to improve oral health to be taken across the life span.

Oral health
The mouth includes not only the teeth and the gums (gingiva) and their supporting tissues, but also the hard and soft palate, the mucosal lining of the mouth and throat, the tongue, the lips, the salivary glands, the chewing muscles, and the upper and lower jaws. Equally important are the branches of the nervous, immune and vascular systems that animate, protect and nourish the oral tissues, as well as provide connections to the brain and the rest of the body. The genetic patterning of development in utero further reveals the intimate relationship of the oral tissues to the developing brain and to the tissues of the face and head that surround the mouth, structures whose location is captured in the word craniofacial.

A key element to discuss is that oral health means much more than healthy teeth. It means being free of chronic oral-facial pain conditions, oral and pharyngeal (throat) cancers, oral soft tissue lesions, birth defects such as cleft lip and palate, and scores of other diseases and disorders that affect the oral, dental and craniofacial tissues, collectively known as the craniofacial complex. These are tissues whose functions we often take for granted, yet they represent the very essence of our humanity. They allow us to speak and smile; sigh and kiss; smell, taste, touch, chew and swallow; cry out in pain; and convey a world of feelings and emotions through facial expressions.

They also provide protection against microbial infections and environmental insults. The craniofacial tissues also provide a useful means to understanding organs and systems in less accessible parts of the body. The salivary glands are a model of other exocrine glands, and an analysis of saliva can provide telltale clues of overall health or disease. The jawbones and their joint function like other musculoskeletal parts. The nervous system apparatus underlying facial pain has its counterpart in nerves elsewhere in the body. A thorough oral examination can detect signs of nutritional deficiencies as well as a number of systemic diseases, including microbial infections, immune disorders, injuries and some cancers. Indeed, the phrase the mouth is a mirror has been used to illustrate the wealth of information that can be derived from examining oral tissues.

New research is pointing to associations between chronic oral infections and heart and lung diseases, stroke and low-birth-weight, premature births. Associations between periodontal disease and diabetes have long been noted. Scientific reports assess these associations and explore mechanisms that might explain the oral-systemic disease connections.

The broadened meaning of oral health parallels the broadened meaning of health. In 1948, the World Health Organization expanded the definition of health to mean “a complete state of physical, mental and social well-being, and not just the absence of infirmity.” It follows that oral health must also include well-being. Just as we now understand that nature and nurture are inextricably linked, and mind and body are both expressions of our human biology, so, too, we must recognize that oral health and general health are inseparable. We ignore signs and symptoms of oral disease and dysfunction to our detriment.

Consequently, a second element of the course is that oral health is integral to general health. You cannot be healthy without oral health. Oral health and general health should not be interpreted as separate entities. Oral health is a critical component of health and must be included in the provision of health care and the design of community programs.

The wider meanings of oral and health in no way diminish the relevance and importance of the two leading dental diseases, caries and the periodontal diseases. They remain common and widespread, affecting nearly everyone at some point in the life span. What has changed is what we can do about them.

Researchers in the 1930s discovered that people living in communities with naturally fluoridated water supplies had fewer dental caries than people drinking unfluoridated water. But not until the end of World War II were the investigators able to design and implement the community clinical trials that confirmed their observations and launch a better approach to the problem of dental caries: prevention. Soon after, adjusting the fluoride content of community water supplies was pursued as an important public health measure to prevent dental caries.

Although this measure has not been fully implemented, the results have been dramatic. Dental caries began to decline in the 1950s among children who grew up in fluoridated cities, and by the late 1970s, decline was evident for many Americans. The application of science to improve diagnostic, treatment and prevention strategies has saved billions of dollars per year in the nation’s annual health bill. Even more significant, the result is that far fewer people are edentulous (toothless) today than a generation ago.

The theme of prevention gained momentum as pioneering investigators and practitioners in the 1950s and 1960s showed that not only dental caries but also periodontal diseases are bacterial infections. The researchers demonstrated that the infections could be prevented by increasing host resistance to disease and reducing or eliminating the suspected microbial pathogens in the oral cavity. The applications of research discoveries have resulted in continuing improvements in the oral health of Americans, new approaches to the prevention and treatment of dental diseases, and the growth of the science.

The significant role that scientists, dentists, dental hygienists and other health professionals have played in the prevention of oral disease and disability leads to a third theme of this course: safe and effective disease prevention measures exist that everyone can adopt to improve oral health and prevent disease. These measures include daily oral hygiene procedures and other lifestyle behaviors, community programs such as community water fluoridation and tobacco cessation programs, and provider-based interventions such as the placement of dental sealants and examinations for common oral and pharyngeal cancers.

General health risk factors, such as tobacco use and poor dietary practices, also affect oral and craniofacial health. The evidence for an association between tobacco use and oral diseases has been clearly delineated in almost every surgeon general’s report on tobacco since 1964, and the oral effects of nutrition and diet are presented in the surgeon general’s report on nutrition (1988). All the dental professions can play a role in reducing the burden of disease in America by calling attention to these and other risk factors and suggesting appropriate actions.

Clearly, promoting health and preventing diseases are concepts the American people have taken to heart. As a nation, we hope to eliminate disparities in health and prevent oral diseases, cancer, birth defects, AIDS and other devastating infections; mental illness and suicide; and the chronic diseases of aging. To live well into old age free of pain and infirmity and with a high quality of life is the American dream.

Scientists today take that dream seriously in researching the intricacies of the craniofacial complex. They are using an ever-growing array of sophisticated analytic tools and imaging systems to study normal function and diagnose disease. They are completing the mapping and sequencing
of human, animal, microbial and plant genomes, the better to understand the complexities of human development, aging and pathological processes. They are growing cell lines, synthesizing molecules and using a new generation of biomaterials to revolutionize tissue repair and regeneration. More than ever before, they are working in multidisciplinary teams to bring new knowledge and expertise to the goal of understanding complex human diseases and disorders.

**Oral diseases and disorders in and of themselves affect health and well-being throughout life.**

The burden of oral problems is extensive and may be particularly severe in vulnerable populations. It includes the common dental diseases and other oral infections such as cold sores and candidiasis that can occur at any stage of life, as well as birth defects in infancy and the chronic facial pain conditions and oral cancers seen in later years. Many of these conditions and their treatments may undermine self-image and self-esteem, discourage normal social interaction, cause other health problems, and lead to chronic stress and depression as well as incur great financial cost. They may also interfere with vital functions such as breathing, food selection, eating, swallowing and speaking and with activities of daily living such as work, school and family interactions.

**Safe and effective measures exist to prevent the most common dental diseases – dental caries and periodontal diseases.** Community water fluoridation is safe and effective in preventing dental caries in both children and adults. Water fluoridation benefits all residents served by community water supplies regardless of their social or economic status. Professional and individual measures, including the use of fluoride mouth rinses, gels, dentifrices and dietary supplements and the application of dental sealants, are additional means of preventing dental caries. Gingivitis can be prevented by good personal oral hygiene practices, including brushing and flossing.

**Lifestyle behaviors that affect general health such as tobacco use, excessive alcohol use and poor dietary choices affect oral and craniofacial health as well.**

These individual behaviors are associated with increased risk for craniofacial birth defects, oral and pharyngeal cancers, periodontal disease, dental caries and candidiasis, among other oral health problems. Opportunities exist to expand the oral disease prevention and health promotion knowledge and practices of the public through community programs and in health care settings. All health care providers can play a role in promoting healthy lifestyles by incorporating tobacco cessation programs, nutritional counseling and other health promotion efforts into their practices.

**There are profound and consequential oral health disparities within the U.S. population.** Disparities for various oral conditions may relate to income, age, sex, race or ethnicity, or medical status. Although common dental diseases are preventable, not all members of society are informed about or able to avail themselves of appropriate oral-health-promoting measures. Similarly, not all health providers may be aware of the services needed to improve oral health. In addition, oral health care is not fully integrated into many care programs. Social, economic and cultural factors and changing population demographics affect how health services are delivered and used, and how people care for themselves. Reducing disparities requires wide-ranging approaches that target populations at highest risk for specific oral diseases and involves improving access to existing care. One approach includes making dental insurance more available to Americans. Public coverage for dental care is minimal for adults, and programs for children have not reached the many eligible beneficiaries.

**The mouth reflects general health and well-being.** The mouth is a readily accessible and visible part of the body and provides health care providers and individuals with a window on their general health status. As the gateway of the body, the mouth senses and responds to the external world and at the same time reflects what is happening deep inside the body. The mouth may show signs of nutritional deficiencies and serve as an early warning system for diseases such as HIV infection and other immune system problems. The mouth can also show signs of general infection and stress. As the number of substances that can be reliably measured in saliva increases, it may well become the diagnostic fluid of choice, enabling the diagnosis of specific disease as well as the measurement of the concentration of a variety of drugs, hormones and other molecules of interest. Cells and fluids in the mouth may also be used for genetic analysis to help uncover risks for disease and predict outcomes of medical treatments.

**Oral diseases and conditions are associated with other health problems.** Oral infections can be the source of systemic infections in people with weakened immune systems, and oral signs and symptoms often are part of a general health condition. Associations between chronic oral infections and other health problems, including diabetes, heart disease, and adverse pregnancy outcomes, have also been reported. Ongoing research may uncover mechanisms that strengthen the current findings and explain these relationships.

**Touch, temperature and pain**

The mouth also contains large numbers of nerve endings, similar to those found elsewhere in the body, that are sensitive to touch (mechanoreceptors), hot and cold temperatures (thermoreceptors) and pain (nociceptors). The dense concentration of these receptors in the facial skin, joints, muscle and oral soft tissues, relayed to an image of the body mapped onto the sensory cortex of the brain, accounts for the finesse with which we can discriminate the qualities and precise location of these sensations. In particular, the periodontal ligament, which anchors the teeth in the jaws, is a tactiley sensitive tissue providing important feedback with regard to mastication and dental occlusion. As a test of this sensibility, a human hair placed between the tips of the fingers will rarely be sufficient to stimulate the nerve endings, but the same hair placed between the lips or incisors will instantly be felt.

Pain and thermal sensitivity in the teeth are transmitted through nerve endings in the pulp. Because the pulp is in a narrow canal composed of connective tissue, blood vessels and nerves and surrounded by hard tissue, any infection or inflammation that would normally cause tissue to swell creates pressure on the pulpal nerves. That pressure, along with bacterial or immune system products that stimulate the nerve endings, produces the severe pain of pulpal infections.

Neuroscientists have long studied oral-facial pain, not only because of its importance in oral disease, but also because it provides an accessible model of pain elsewhere in the body. These investigations have greatly enriched our understanding of the basic mechanisms of pain perception and modulation. They have helped delineate the complex pathways and multiple transmitters that convey pain signals to the brain and spinal cord, as well as the mechanisms and molecules that can modulate and inhibit nociceptive input. These studies have also exploited new brain-imaging techniques to confirm the wide distribution of pain pathways and relay centers in the cerebral hemispheres and cerebellum.

This research has generated new approaches to the control of acute and chronic pain. These approaches include the use of nonsteroidal, anti-inflammatory drugs and long-acting local anesthetics for acute oral and dental pain, and the use of more potent drugs, drug combinations and other kinds of therapies to treat chronic pain.

Researchers have emphasized the importance of adequate pain control in patients with chronic pain conditions. Otherwise, the constant barrage of signals can effect long-term changes in the brain that actually worsen the pain (producing hyperalgesia) and cause normally nonpainful stimuli to be perceived as painful (a condition called allodynia). Unrelieved chronic pain may also suppress the immune system.

Recently, investigators discovered a link between certain taste sensations, pain and temperature. Their findings indicate that capsaicin, the ingredient that makes hot peppers taste hot, binds to a receptor on the surface of nociceptors that also responds to noxious heat. The researchers have cloned the gene for the capsaicin receptor (called vanilloid receptor 1); they believe it is involved in several chronic pain conditions, especially those where inflammation plays a role, such as viral and diabetic neuropathy, rheumatoid arthritis and oral mucositis pain caused by cancer chemotherapy or radiation.

There is evidence that the prevalence of a number of pain conditions varies by gender and that men and women respond differently to different analgesic drugs. These findings
have prompted studies aimed at determining whether there are sex differences in pain anatomy and neurochemistry and whether (and how) nociception is affected by sex hormones.

**Speech**

Human speech and language are the faculties that most distinguish us from other higher primates; they are also the links that bind people together in diverse social groups and cultures.

Central to speech are laryngeal mechanisms involving the vocal cords. Equally critical are the respiratory system, the pharynx and the nasal and oral cavities. The tongue is the most important structure of the peripheral speech mechanisms, working in conjunction with the lips, teeth and palate to produce a rich repertoire of sounds.

Abnormalities in oral structures, from missing or malformed teeth and malocclusion to cleft lip and palate, can seriously affect articulation. The movements of speech are orchestrated by brain centers that coordinate the muscles of mastication, facial expression and jaw movements.

Hearing impairments can also affect speech. To learn to speak, children must be able to hear others and monitor the feedback from their own voices. Congenital deafness and the serious hearing defects associated with some craniofacial syndromes can severely compromise speech acquisition.

**The oral cavity**

The mouth is the gateway to the body, performing dozens of functions that place high demands on its unique hard and soft tissues. The point of entry is the lips, which open into the oral cavity. The cheeks form the sides of the cavity, and the palate, which separates the mouth from the nose above and the pharynx (throat) behind. The anterior palate is hard, formed by underlying bone, and serves as a shield against trauma to the face and head. The posterior palate is soft, composed of muscles and connective tissue that blend into the walls of the pharynx. Hanging from the rear of the soft palate is the uvula, a mass of muscle and connective tissue. Under the tongue is the floor of the mouth, composed primarily of muscle and salivary glands. The paired tonsils and adenoids, important components of the immune system, lie at the sides of the palate and within the nasopharynx, respectively.

The pharynx opens into channels leading either to the lungs for respiration or the esophagus for further digestion and passage to the stomach. This is a point of vulnerability: Should food or some other obstruction lodge in the airway, it could lead to death by asphyxiation.

Externally, the oral cavity is bounded by the maxilla (the upper jaw bone), attached to the cranium, and the mandible (the lower jaw), attached to the temporal bone of the skull by the temporomandibular joint.

**Oral invaders**

As the gateway to the body, the mouth is challenged by a constant barrage of invaders – bacteria, viruses, parasites, fungi. Thus infectious diseases, notably dental caries and periodontal diseases, predominate among the ills that can compromise oral health. Injuries take their toll as well, with the face and head particularly vulnerable to sports injuries, motor vehicle crashes, violence and abuse. Less common but very serious are oral and pharyngeal cancers, with a five-year survival rate of hardly better than 50 percent. Birth defects and developmental disorders frequently affect the craniofacial complex. These appear most commonly as isolated cases of cleft lip or palate, but clefting or other craniofacial defects can also be part of complex hereditary diseases or syndromes. Additionally, acute and chronic pain can affect the oral-facial region, particularly in and around the temporomandibular (jaw) joint, and accounts for a disproportionate amount of all types of pain that drive individuals to seek health care.

Many systemic diseases such as diabetes, arthritis, osteoporosis and AIDS as well as therapies for systemic diseases can directly or indirectly compromise oral tissues. The World Health Organization’s International Classification of Diseases and Stomatology currently lists more than 120 specific diseases, distributed in 10 or more classes that have manifestations in the oral cavity.

**Dental and periodontal infections**

The most common oral diseases are dental caries and the periodontal diseases. Individuals are vulnerable to dental caries throughout life, with 85 percent of adults aged 18 and older affected. Periodontal diseases are most often seen in maturity, with the majority of adults experiencing some signs and symptoms by the mid-30s. Certain rare forms of periodontal disease affect young people. The major oral health success story of the past half century is that both caries and periodontal diseases can be prevented by a combination of individual, professional, and community measures.

**Dental caries**

The word caries derives from the Latin for rotten, and many cultures early on posited a tooth worm as the cause of this rottenness. By the 20th century, caries came to describe the condition of having holes in the teeth – cavities. This description, although not incorrect, is misleading. In actuality, a cavity is a late manifestation of a bacterial infection.

The bacteria colonizing the mouth are known as the oral flora. They form a complex community that adheres to tooth surfaces in a gelatinous mat, or biofilm, commonly called dental plaque. A cariogenic biofilm at a single tooth site may contain one-half billion bacteria, of which species of mutans streptococci are critical components. These bacteria are able to ferment sugars and other carbohydrates to form lactic and other acids. Repeated cycles of acid generation can result in the microscopic dissolution of minerals in tooth enamel and the formation of an opaque white or brown spot under the enamel surface. Frequency of carbohydrate consumption, physical characteristics of food (e.g., stickiness), and timing of food intake also play a role.

The essential role of bacteria in caries initiation was established in landmark experiments in the 1950s. Investigators observed that germ-free animals fed high-sugar diets remained caries-free until the introduction of mutans streptococci (a particular group of bacterial strains having a number of common characteristics and which adhere tightly to the tooth). Later experiments demonstrated the transmissibility of the bacteria from mother to litter and from caries-infected to uninfected cage-mates. Species of Lactobacillus, Actinomyces and other acid-producing streptococci within the plaque may also contribute to the process.

If the caries infection in enamel goes unchecked, the acid dissolution can advance to form a cavity that can extend through the dentin (the component of the tooth located under the enamel) to the pulp tissue, which is rich in nerves and blood vessels. The resulting toothache can be severe and often is accompanied by sensitivity to temperature and sweets. Treatment requires endodontic (root canal) therapy. If untreated, the pulp infection can lead to abscess, destruction of bone, and spread of the infection via the bloodstream.

Dental caries can occur at any age after teeth erupt. Particularly damaging forms can begin early, when developing primary teeth are especially vulnerable. This type of dental caries is called early childhood caries (ECC). Some six out of 10 children in the United States have one or more decayed or filled primary teeth by age 5. ECC may occur in children who are given pacifying bottles of juice, milk or formula to drink during the day or overnight. The sugar contents pool around the upper front teeth, mix with cariogenic bacteria and give rise to rapidly progressing destruction. Other risk factors for ECC include arrested development of tooth enamel, chronic illness, altered salivary composition and volume (resulting from the use of certain medications or malnourishment), mouth breathing and blockage of saliva flow in a bottle-fed infant.

Although there have been continuing reductions in dental caries in permanent teeth among children and adolescents over the past few decades, caries prevalence in the primary dentition may have stabilized or increased slightly in some population groups. Reductions in caries in permanent teeth also have been proportionately greater on the smooth surfaces rather than on the pit-and-fissure surfaces characteristic of chewing surfaces. The gingival tissues tend to recede over time, exposing the tooth root to cariogenic bacteria that can cause root caries. An important risk factor for root caries in older people is the use of medications that inhibit salivary flow, leading to dry mouth (xerostomia).

Saliva contains components that can directly attack cariogenic bacteria, and it is also rich in calcium and phosphates that help to remineralize tooth enamel. Demineralization of enamel occurs when pH levels fall as a result of acid production by bacteria. It can be reversed...
at early stages if the local environment can counteract acid production, restoring pH to neutral levels. Remineralization can occur through the replacement of lost mineral (calcium and phosphates) from the stores in saliva. Fluoride in saliva and dental plaque and the buffering capacity of saliva also contribute to this process. Indeed, it is now believed that fluoride exerts its chief caries-preventive effect by facilitating remineralization. Several studies have demonstrated that remineralization results in an increase in tooth hardness and mineral content, rendering the tooth surface more resistant to subsequent acid attack. Overt caries lesions develop when there is insufficient time for remineralization between periods of acidogenesis or when the saliva production is compromised. Over 400 medications list dry mouth as a side effect, notably some antidepressants, antipsychotics, antihistamines, decongestants, antihypertensives, diuretics and anti-Parkinsonian drugs. The effects of xerostomia may be particularly severe in cancer patients receiving radiation to the head or neck because the rays can destroy salivary gland tissue rather than simply inhibiting salivary secretion.

The professional application of dental sealants (plastic films coated onto the chewing surfaces of teeth) is an important caries-preventive measure that complements the use of fluorides. The films prevent decay from developing in the pits and fissures of teeth, channels that are often inaccessible to brushing and where fluoride may be less effective.

The rate of caries progression through enamel is relatively slow and may be slower in patients who have received regular fluoride treatment or who consume fluoridated water. Because a large percentage of enamel lesions remain unchanged over periods of three to four years, and because progression rates through dentin are comparatively slow, the application of infection control and monitoring procedures to assess caries risk status, lesion activity status, evidence of lesion arrest and evidence of lesion remineralization over extended periods of time is recommended. Experts believe that the earlier mutants streptococci are acquired in infancy, the higher the caries risk. Most studies indicate that infants are infected before their first birthday, around the time the first incisors emerge. However, one study found the median age of acquisition to be 26 months, coinciding with the emergence of the primary molars. DNA fingerprinting has demonstrated that the source of transmission is usually the mother.

It is not clear why some individuals are more susceptible and others more resistant to caries. Genetic differences in the structure and biochemistry of enamel proteins and crystals as well as variations in the quality and quantity of saliva and in immune defense mechanisms are among the factors under study. Analysis of mutants streptococci genomes may also shed light, indicating which species are particularly virulent and which genes contribute to that virulence.

Even the most protective genetic endowment and developmental milieu are unlikely to confer resistance to decay in the absence of positive personal behaviors. These include sound dietary habits and good oral hygiene, including the use of fluorides, and seeking professional care. There are indications, however, that some destructive oral habits are on the rise, such as the use of smokeless (spit) tobacco products by teenage boys. Although the chief concern here lies in the long-term risk for oral cancers, spit tobacco that contains high levels of sugar is also associated with increased levels of decay of both crown and root surfaces.

Periodontal diseases

Like dental caries, the periodontal diseases are infections caused by bacteria in the biofilm (dental plaque) that forms on oral surfaces. The basic division in the periodontal diseases is between gingivitis, which affects the gums, and periodontitis, which may involve all of the soft tissue and bone supporting the teeth. Gingivitis and milder forms of periodontitis are common in adults. The percentage of individuals with moderate to severe periodontitis, in which the destruction of supporting tissue can cause the tooth to loosen and fall out, increases with age.

Signs and symptoms

In the early stages, periodontitis has very few symptoms, and in many individuals the disease has progressed significantly before they seek treatment. Symptoms may include the following:

- Redness or bleeding of gums while brushing teeth, using dental floss or biting into hard food (such as apples) (though this may occur even in gingivitis, where there is no attachment loss).
- Gum swelling that recurs.
- Halitosis, or bad breath, and a persistent metallic taste in the mouth.
- Gingival recession, resulting in apparent lengthening of teeth. (This may also be caused by heavy-handed brushing or with a stiff toothbrush.)
- Deep pockets between the teeth and the gums (pockets are sites where the attachment has been gradually destroyed by collagen-destroying enzymes, known as collagenases).
- Loose teeth, in the later stages (though this may occur for other reasons as well).
- Patients should realize that the gingival inflammation and bone destruction are largely painless. Hence, people may wrongly assume that painless bleeding after teeth cleaning is insignificant, although this may be a symptom of progressing periodontitis in that patient.

Certain factors increase the risk for periodontal disease:

- Smoking.
- Diabetes.
- Poor oral hygiene.
- Stress.
- Heredity.
- Crooked teeth.
- Underlying immunodeficiencies – e.g., AIDS.
- Fillings that have become defective.
- Taking medications that cause dry mouth.
- Bridges that no longer fit properly.
- Female hormonal changes, such as with pregnancy or the use of oral contraceptives.

Prevention

Daily oral hygiene measures to prevent periodontal disease include:

- Brushing properly on a regular basis (at least twice daily), with the patient attempting to direct the toothbrush bristles underneath the gumline, to help disrupt the bacterial growth and formation of subgingival plaque.
- Flossing daily and using interdental brushes (if there is a sufficiently large space between teeth), as well as cleaning behind the last tooth, the third molar, in each quarter.
- Using an antiseptic mouthwash.
- Chlorhexidine gluconate based mouthwash in combination with careful oral hygiene may cure gingivitis, although they cannot reverse any attachment loss due to periodontitis.
- Using a “soft” toothbrush to prevent damage to tooth enamel and sensitive gums.
- Using periodontal trays to maintain dentist-prescribed medications at the source of the disease. The use of trays allows the medication to stay in place long enough to penetrate the biofilms where the bacteria are found.
- Regular dental check-ups and professional teeth cleaning as required. Dental check-ups serve to monitor the person’s oral hygiene methods and levels of attachment around teeth, identify any early signs of periodontitis, and monitor response to treatment.

Typically dental hygienists (or dentists) use special instruments to clean (debride) teeth below the gumline and disrupt any plaque growing below the gumline. This is a standard treatment to prevent any further progress of established periodontitis. Studies show that after such a professional cleaning (periodontal debridement), bacteria and plaque tend to grow back to pre-cleaning levels after about three to four months. Hence, in theory, cleanings every three to four months might be expected to also prevent the initial onset of periodontitis. However, analysis of published research has reported little evidence either to support this or the intervals at which this should occur. Instead, it is advocated that the interval between dental check-ups should be determined specifically for each patient between every three to 24 months.

Nonetheless, the continued stabilization of a patient’s periodontal state depends largely, if not primarily, on the patient’s oral hygiene at home as well as on the go. Without daily oral hygiene, periodontal disease will not be overcome, especially if the patient has a history of extensive periodontal disease.

A contributing cause may be low selenium in the diet: “Results showed that selenium has the strongest association with gum disease, with low levels increasing the risk by 13-fold.”

The cornerstone of successful periodontal treatment starts with establishing excellent oral
hygiene. This includes twice daily brushing with daily flossing. Also the use of an interdental brush (called a proxi-brush) is helpful if space between the teeth allows. Persons with dexterity problems such as arthritis may find oral hygiene to be difficult and may require more frequent professional care and the use of a powered toothbrush. Persons with periodontitis must realize that it is a chronic inflammatory disease and a lifelong regimen of excellent hygiene and professional maintenance care with a dentist/hygienist or periodontist is required to maintain affected teeth.

Initial therapy
Removal of bacterial plaque and calculus is necessary to establish periodontal health. The first step in the treatment of periodontitis involves nonsurgical cleaning below the gumline with a procedure called scaling and debridement. In the past, root planing was used (removal of cemental layer as well as calculus). This procedure involves use of specialized curettes to mechanically remove plaque and calculus from below the gumline, and may require multiple visits and local anesthesia to adequately complete. In addition to initial scaling and root planing, it may also be necessary to adjust the occlusion (bite) to prevent excessive force on teeth with reduced bone support. Also it may be necessary to complete any other dental needs such as replacement of rough, plaque retentive restorations, closure of open contacts between teeth and any other requirements diagnosed at the initial evaluation.

Re-evaluation
Multiple clinical studies have shown that nonsurgical scaling and root planing is usually successful in periodontal pocket depths no greater than 4.5mm. It is necessary for the dentist or hygienist to perform a re-evaluation four to six weeks after the initial scaling and root planing to determine whether the treatment was successful in reducing pocket depths and eliminating inflammation. It has been found that pocket depths which remain after initial therapy of greater than 5-6 mm with bleeding upon probing are indicative of continued active disease and will very likely show further bone loss over time. This is especially true in molar tooth sites where furcations (areas between the roots) have been exposed.

Periodontal surgery
If nonsurgical therapy is found to be unsuccessful in managing signs of disease activity, periodontal surgery may be needed to stop progressive bone loss and regenerate lost bone where possible. There are many surgical approaches used in treatment of advanced periodontitis, including open flap debridement, osseous surgery, guided tissue regeneration and bone grafting. The goal of periodontal surgery is access for definitive calculus removal and surgical management of bony irregularities that have resulted from the disease process to reduce pockets as much as possible. Long-term studies have shown that in moderate to advanced periodontitis, surgically treated cases often have less further breakdown over time and when coupled with a regular post-treatment maintenance regimen are successful in nearly halting tooth loss in nearly 85 percent of patients.

Maintenance
Once successful periodontal treatment has been completed, with or without surgery, an ongoing regimen of “periodontal maintenance” is required. This involves regular checkups and detailed cleanings every three months to prevent repopulation of periodontitis-causing bacteria, and to closely monitor affected teeth so that early treatment can be rendered if disease recurs. Usually periodontal disease exists due to poor plaque control, so if the brushing techniques are not modified, a periodontal recurrence is probable.

Assessment and prognosis
Dentists and dental hygienists “measure” periodontal disease using a device called a periodontal probe. This is a thin “measuring stick” that is gently placed into the space between the gums and the teeth and slipped below the gumline. If the probe can slip more than 3 millimetres length below the gumline, the patient is said to have a “gingival pocket” around that tooth. This is somewhat of a misnomer, as any depth is in essence a pocket, which in turn is defined by its depth, i.e., a 2 mm pocket or a 6 mm pocket. However, it is generally accepted that pockets are self-cleansable (at home, by the patient, with a toothbrush) if they are 3 mm or less in depth. This is important because if there is a pocket which is deeper than 3 mm around the tooth, at-home care will not be sufficient to cleanse the pocket, and professional care should be sought. When the pocket depths reach 6 and 7 mm in depth, the hand instruments and cavitrons used by the dental professionals may not reach deeply enough into the pocket to clean out the bacterial plaque that cause gingival inflammation. In such a situation the bone or the gums around that tooth should be surgically altered or it will always have inflammation, which will likely result in more bone loss around that tooth. An additional way to stop the inflammation would be for the patient to receive subgingival antibiotics (such as minocycline) or undergo some form of gingival surgery to access the depths of the pockets and perhaps even change the pocket depths so that they become 3 mm or less in depth and can once again be properly cleaned by the patient at home with his or her toothbrush.

If a patient has 7 mm or deeper pockets around the teeth, then he or she would likely risk eventual tooth loss over the years. If this periodontal condition is not identified and the patient remains unaware of the progressive nature of the disease then, years later, they may be surprised that some teeth will gradually become loose and may need to be extracted, sometimes due to a severe infection or even pain. According to the Sri Lankan Tea Labourer study, in the absence of any oral hygiene activity, approximately 10 percent will suffer from severe periodontal disease with rapid loss of attachment (more than 2 mm/year). Eighty percent will suffer from moderate loss (1-2 mm/year), and the remaining 10 percent will not suffer any loss.

Alternative treatments
Periodontitis has an inescapable relationship with subgingival calculus (tartar). The first step in any procedure is to eliminate calculus under the gumline, as it houses destructive anaerobic bacteria that consume bone, gum and cementum (connective tissue) for food.

Most alternative “at-home” gum disease treatments involve injecting antimicrobial solutions, such as hydrogen peroxide, into periodontal pockets via slender applicators or oral irrigators. This process disrupts anaerobic bacteria colonies and is effective at reducing infections and inflammation when used daily. There are any number of potions and elixirs that are commercially available which are functionally equivalent to hydrogen peroxide; but at substantially higher cost. These treatments, however, do not address calculus formations and are therefore short-lived, as anaerobic bacteria colonies quickly regenerate in and around calculus.

In a new field of study, calculus formations are addressed on a more fundamental level. At the heart of the formation of subgingival calculus, growing plaque formations starve out the lowest members of the community, which calcify into calcium phosphate salts of the same shape and size of the original, organic bacilli. Calcium phosphate salts (unlike calcium phosphate; the primary component in teeth) are ionic and adhere to tooth surfaces via electrostatic attraction. Smaller, free floating calcium phosphate salt particles are equally attracted to the same areas, as are additional calcified bacteria, growing calculus formations as unorganized, yet strong, “brick and mortar” matrices. The microscopic voids in calculus formations house new anaerobic bacteria, as does the top “diseased layer.”

Because the root cause of subgingival calculus development is ionic attraction, it was hypothesized that the introduction of oppositely charged particles around the formations might chelate calcium phosphate salt components away from the matrix, thus actually reducing the size of subgingival calculus formations.

To accomplish this, a sequestering agent solution comprised partly of sodium tripolyphosphate...
(STPP) and sodium fluoride (charge-1) was tested on a patient with burnished and new subgingival calculus at a depth of 6 mm. The patient delivered the solution using an oral irrigator, once a day for 60 days. The results of this test were the successful elimination of all calculus formations studied. This test was conducted using a subgingival endoscopic camera (perioscope) by an independent periodontist.

This alternative treatment keeps subgingival calculus at bay, in concert with traditional periodontal treatments. In this way, periodontitis may be controlled by the patient, with complete restoration of dental health being a collaborative effort between the patient and the dental professional.

**Gingivitis**

Gingivitis is an inflammation of the gums characterized by a change in color from normal pink to red, with swelling, bleeding and often sensitivity and tenderness. These changes result from an accumulation of biofilm along the gingival margins and the immune system’s inflammatory response to the release of destructive bacterial products. The early changes of gingivitis are reversible with thorough toothbrushing and flossing to reduce plaque. Without adequate oral hygiene, however, these early changes can become more severe, with infiltration of inflammatory cells and establishment of a chronic infection. Biofilm on tooth surfaces opposite the openings of the salivary glands often mineralizes to form calculus or tartar, which is covered by unmineralized biofilm – a combination that can exacerbate local inflammatory responses. A gingival infection may persist for months or years, yet never progress to periodontitis.

Gingival inflammation does not appear until the biofilm changes from one composed largely of gram-positive streptococci (which can live with or without oxygen) to one containing gram-negative anaerobes (which cannot live in the presence of oxygen). Numerous attempts have been made to pinpoint which microorganisms in the supragingival (above the gumline) plaque are the culprits in gingivitis. Frequently mentioned organisms include Fusobacterium nucleatum, Veillonella parvula, and species of Campylobacter and Treponema. But as Ranney (1989) notes, “The complexity of the results defies any attempt to define a discrete group clearly and consistently associated with gingivitis.”

Gingival inflammation may be influenced by steroid hormones, occurring as puberty gingivitis, pregnancy gingivitis, and gingivitis associated with birth control medication or steroid therapy. The presence of steroid hormones in tissues adjacent to biofilm apparently encourages the growth of certain bacteria and triggers an exaggerated response to biofilm accumulation. Again, thorough oral hygiene can control this response.

Certain prescription drugs can also lead to gingival overgrowth and inflammation. These include the antiepileptic drug phenytoin (Dilantin); cyclosporin, used for immunosuppressive therapy in transplant patients; and various calcium channel blockers used in heart disease. Treatment often requires surgical removal of the excess tissue followed by appropriate personal and professional oral health care.

A form of gingivitis common 50 years ago but relatively rare today is acute necrotizing ulcerative gingivitis, also known as Vincent’s infection or trench mouth. This aggressive infection is characterized by destruction of the gingiva between the teeth, spontaneous bleeding, pain and oral odor. People under extreme stress have an increased susceptibility. Spirochetes and other bacteria have been found in the connective tissue of those affected. An association between smoking and this type of gingivitis is well recognized and was demonstrated as early as 1946. This condition has been seen in some HIV-positive patients. Treatment requires a combination of professional periodontal treatment and antibiotic therapy along with professional smoking cessation assistance as appropriate.

**Adult periodontitis**

The most common form of adult periodontitis is described as general and moderately progressing; a second form is described as rapidly progressing and severe, and is often resistant to treatment. The moderately progressive adult form is characterized by a gradual loss of attachment of the periodontal ligament to the gingiva and bone along with loss of the supporting bone. It is most often accompanied by gingivitis. It is not necessarily preceded by gingivitis, but the gingivitis-related biofilm often seeds the subgingival plaque. The destruction of periodontal ligament and bone results in the formation of a pocket between the tooth and adjacent tissues, which harbors subgingival plaque. The calculus formed in the pocket by inflammatory fluids and minerals in adjacent tissues is especially damaging.

The severity of periodontal disease is determined through a series of measurements, including the extent of gingival inflammation and bleeding, the probing depth of the pocket to the point of resistance, the clinical attachment loss of the periodontal ligament measured from a fixed point on the tooth (usually the cemento-ENAMEL junction), and the loss of adjacent alveolar bone as measured by x-ray. Severity is determined by the rate of disease progression over time and the response of the tissues to treatment.

Adult periodontitis often begins in adolescence but is usually not clinically significant until the mid-30s. Prevalence and severity increase but do not accelerate with age. One view proposes that destruction occurs at a specific site during a defined period, after which the disease goes into remission. The current view is that the disease process may not be continuous but rather progresses in random bursts in which short periods of breakdown of periodontal ligament and bone alternate with periods of quiescence. These episodes occur randomly over time and at random sites in the mouth. Part of the difficulty in determining the pattern of progression reflects variation in the sensitivity of the instruments used to measure the loss of soft tissue and bone. The latest generation of probes finds evidence of both continuous and multiple-burst patterns of loss in different patients and at different times.

Most researchers agree that periodontitis results from a mixed infection but that a particular group of gram-negative bacteria are key to the process and markedly increase in the subgingival plaque. The bacteria most frequently cited arePorphyromonas gingivalis,Prevotella intermedia,Bacteroides forsythus,Treponema denticola, and Actinobacillus actinomycetemcomitans. Their role in disease initiation and progression is determined in part by their “virulence factors.” These include the ability to colonize subgingival plaque, generate products that can directly injure tissues, and elicit an inflammatory or immune response. The potentially noxious bacterial products include hydrogen sulfide, polyamines, the fatty acids butyrate and propionate, lipopolysaccharide (also known as endotoxin), and a number of destructive enzymes. The interaction of this arsenal with the host response is at the core of periodontal pathology, (Socransky and Haffajee 1991, 1992). Sequencing of the genomes of several key periodontal pathogens is under way and should provide further insight into these pathogens as well as catalyze new treatment approaches.

This drawing shows a dentist using a probe to check for inflammatory pockets.

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Delicate balances

Neutrophils (a type of white blood cell) and antibodies are the major immune defenses against bacterial attack. Neutrophils move to the site of infection, where they engulf bacteria and elaborate antibacterial agents and enzymes to destroy bacteria. Although stimulation of the immune system to attack the offending bacteria is generally protective, immune hyper-responsiveness and hypersensitivity can be counterproductive, leading to the destruction of healthy tissue. Nevertheless, the neutrophil/antibody axis is critical for full protection against periodontal diseases.

Also important is the release of certain potent molecules called cytokines and prostaglandins, especially prostaglandin E2 (PGE2) which can contribute to tissue destruction. Cytokines are proteins secreted by immune cells that help regulate immune responses and also affect bone,
Epidemiological studies have confirmed that certain systemic diseases heighten susceptibility. However, female hormones may play a protective role (as they do in protecting against osteoporosis). 

Susceptibility and resistance

PGE2 may play a central role in the tissue destruction that occurs in periodontal diseases. Levels of PGE2 in periodontal tissue are low or undetectable in health, increase in gingivitis, and rise significantly in periodontitis. Now there is increasing evidence that the level of PGE2 produced in response to bacterial challenge (especially by endotoxin) can be used as a measure of susceptibility. Presumably, the level of PGE2 production is subject to genetic influence. Studies of identical and fraternal twins, either reared together or apart, provide evidence that genetic factors may indeed influence susceptibility or resistance to the common adult form of periodontitis. Recently, a commercial test for a genetic marker of susceptibility has been introduced. The marker is associated with increased production of a particular form of interleukin 1-beta (IL-1-beta) when stimulated by periodontopathic bacteria. In 1996, it was found that nonsmoking adults who are positive for the marker are 6 to 19 times more likely to develop severe periodontitis. Susceptibility to adult periodontitis has also been explored in relation to a variety of behavioral and demographic variables as well as to the presence of other diseases. One of the strongest behavioral associations is with tobacco use. The risk of alveolar bone loss for heavy smokers is seven times greater than for those who have not smoked. Cigarette smoking also may impair the normal host response to neutralizing infection, resulting in the destruction of healthy periodontal tissues adjacent to the site of infection. Smokers also have decreased levels of salivary and serum immunoglobulins to Prevotella intermedia and Fusobacterium nucleatum and edentulousness have been reported in osteoporotic women. Two studies in 1996 showed that estrogen replacement therapy in postmenopausal women not only gives protection against osteoporosis, but also results in fewer teeth lost to periodontal disease. The less common rapidly progressive form of adult periodontitis typically affects people in their early 20s and 30s. It is characterized by severe gingival inflammation and rapid loss of connective tissue and bone. Many patients have an inherent defect in neutrophil response to infection. Several systemic diseases have been associated with this form of periodontal disease, including type 1 diabetes, Down syndrome, Papillon-Lefèvre syndrome, Chediak-Higashi syndrome and HIV infection. Specific bacteria associated with rapidly progressive disease include Porphyromonas gingivalis, Prevotella intermedia, Eikenella corrodens, and Wolinella recta. Most recently, mutations in the cathepsin C gene have been associated with the Papillon-Lefèvre syndrome and how the defect can result in periodontal disease.

Refractory periodontitis

Refractory periodontitis is not a specific form of disease, but refers to cases in which patients continue to exhibit progressive disease at multiple sites despite aggressive mechanical therapy to remove biofilm and calculus, along with the use of antibiotics. Refractory sites exhibit elevated levels of a number of different bacteria, with the dominant species different in different subjects. It is not known whether variations in pathogenicity of the bacteria, defects in the subject’s defense systems, or combinations of these factors are responsible for the refractory nature of the disease. The adoption of new diagnostic technology to detect predominant bacterial species, followed by selective antibiotic treatment, may help resolve infection and disease in these patients.

Early-onset periodontitis

The forms of periodontitis occurring in adolescents and young adults generally involve defects in neutrophil function. Localized juvenile periodontitis (LJP) mainly affects the first molar and incisor teeth of teenagers and young adults, with rapid destruction of bone but almost no telltale signs of inflammation and very little supragingival plaque or calculus. Actinobacillus actinomycetemcomitans has been isolated at 90 to 100 percent of diseased sites in these patients, but is absent or appears in very low frequency in healthy or minimally diseased sites. It is possible that the bacteria are transmitted among family members through oral contacts such as kissing or sharing utensils, because the same bacterial strain appears in affected family members. However, evidence of a neutrophil defect argues for a genetic component. Another organism frequently associated with LJP is Capnocytophaga ochracea. Neither of these bacteria dominate in the generalized adult form of the disease, where Porphyromonas gingivalis is considered of greatest significance.

Prepubertal periodontitis is rare and can be either general or localized. The generalized form begins with the eruption of the primary teeth and proceeds to involve the permanent teeth. There is severe inflammation, rapid bone loss, tooth mobility and tooth loss. The localized form of the disease is less aggressive, affecting only some primary teeth. The infection involves many of the organisms associated with periodontitis, but the mix may differ somewhat, with Actinobacillus actinomycetemcomitans, Prevotella intermedia, Eikenella corrodens and several species of Capnocytophaga implicated. Defects in neutrophil function noted in both forms of the may explain why patients are highly susceptible to other infections as well.

Selected mucosal infections and conditions

Like the skin, the mucosal lining of the mouth serves to protect the body from injury. This lining is itself subject to a variety of infections and conditions, ranging from benign canker sores to often fatal cancers.

Oral candidiasis

Chronic hyperplastic candidiasis is a red or white lesion that may be flat or slightly elevated and may adhere to soft or hard tissue surfaces, including dental appliances. It is caused by species of Candida, especially Candida albicans, the most common fungal pathogen isolated from the oral cavity. Normally, the fungi are present in relatively low numbers in up to 65 percent of healthy children and adults and cause no harm. Problems arise when there is a change in oral homeostasis – the normal balance of protective mechanisms and resident oral flora that maintain the health of the oral cavity, so that defense mechanisms are compromised. Under these circumstances, the fungal organisms can overgrow to cause disease. A primary disruption in homeostasis occurs with the use of antibiotics and corticosteroids, which can markedly change the composition of the oral flora.

Deficiencies in the immune and endocrine systems are also important. Indeed, the diagnosis of candidiasis in an otherwise seemingly healthy young adult may be the first sign of HIV infection. Other causes of candidiasis include cancer chemotherapy or radiotherapy to the head and neck, xerostomia resulting from radiation to the head and neck, medications, chronic mucosal irritation, certain blood diseases and other...
systemic conditions. Also, tobacco use has been identified as a cofactor.

Candidiasis often causes symptoms of burning and soreness as well as sensitivity to acidic and spicy foods. Patients may complain of a foul taste in the mouth. However, it can also be asymptomatic. Genomic analysis of the Candida albicans genome is helping investigators identify numerous genes that code for virulence factors, including enzymes that can facilitate adhesion to and penetration of mucous membranes. At the same time, researchers are exploring novel gene technologies to increase production of a family of native salivary proteins, the histatins that have known antifungal and other antimicrobial effects.

The most common form of oral candidiasis is denture stomatitis. It occurs when tissues are traumatized by continued wearing of ill-fitting or inadequately cleaned dental appliances and is described as chronic erythematous candidiasis. Another form, candidal angular cheilosis, occurs in the folds at the angles of the mouth and is closely associated with denture sore mouth. Other common forms of Candida infection are pseudomembranous candidiasis (thrush), which may affect any of the mucosal surfaces, and acute erythematous candidiasis, a red and markedly painful variant commonly seen in AIDS patients.

In most cases, Candida infection can be controlled with antifungal medications used locally or systemically. Control is difficult, however, in patients with immune dysfunction, as in AIDS, or other chronic debilitating diseases. Often the organisms become resistant to standard therapy, and aggressive approaches are necessary. The spread of oral candidiasis to the esophagus or lungs can be life-threatening and is one of the criteria used to define frank AIDS.

Herpes simplex virus infections
In any given year, about a half-million Americans will experience their first encounter with the herpes simplex virus type 1 (HSV-1), the cause of cold sores. That first encounter usually occurs in the oral region and may be so mild as to go unnoticed. But in some people, particularly young children and young adults, infection may take the form of primary herpetic stomatitis, with symptoms of malaise, muscle aches, sore throat and enlarged and tender lymph nodes before the appearance of the familiar cold sore blisters. These blisters usually show up on the lips, but any of the mucosal surfaces can be affected. Bright-red ulcerated areas and marked gingivitis may also be seen.

Herpes viruses also cause genital infections, which are transmitted sexually. Both HSV-1 and HSV-2 have been found in oral and genital infections, with HSV-1 predominating in oral areas and HSV-2 in genital areas. Herpes viruses have also been implicated as cofactors in the development of oral cancers. Crowded living conditions can result in greater contact with infected individuals, which aids in transmission of HSV.

Normally, the immune system mounts a successful attack on the viruses, with symptoms abating by the time neutralizing antibodies appear in the bloodstream, in about 10 days. However, herpes viruses are notorious for their ability to avoid immune detection by taking refuge in the nervous system, where they can remain latent for years. In oral herpes, the virus commonly migrates to the nearby trigeminal ganglion, the cluster of nerve cells whose fibers branch out to the face and mouth. In about 20 to 40 percent of people who are virus-positive, the virus may reactivate, with infectious virus particles moving to the oral cavity to cause recurrent disease.

The usual site of a recurrent lesion is on or near the lips. Recurrences are rarely severe, and lesions heal in seven to 10 days without scarring. The recurrences may be provoked by a wide range of stimuli, including sunlight, mechanical trauma and mild fevers such as occur with a cold. Emotional factors may play a role as well.

Oral human papillomavirus infections
There are more than 100 recognized strains of oral human papillomavirus (HPV), a member of the papovavirus family, implicated in a variety of oral lesions. Most common are papillomas (warts) found on or around the lips and in the mouth. HPV is found in 80 percent of these oral squamous papillomas. The virus has also been identified in 30 to 40 percent of oral squamous cell carcinomas and has been implicated in cervical cancer as well. Whether a cancer or nonmalignant wart develops may depend on which virus is present or on which viral genes are activated.

Oral warts are most often found in children, probably as a result of chewing warts on the hands. In adults, sexual transmission from the anogenital region can occur. In general, viral warts spontaneously regress after one or two years. The immune system normally keeps HPV infections under control, as evidenced by the increased prevalence of HPV-associated lesions in HIV-infected patients and others with immunodeficiency.

Recurrent aphthous ulcers or canker sores
Recurrent aphthous ulcers (RAU), also referred to as recurrent aphthous stomatitis, is the technical term for canker sores, the most common and generally mild oral mucosal disease. Between 5 and 25 percent of the general population is affected, with higher numbers in selected groups, such as health professional students. The disease takes three clinical forms: RAU minor, RAU major and herpetiform RAU. The minor form accounts for 70 to 87 percent of cases. The sores are small, discrete, shallow ulcers surrounded by a red halo appearing at the front of the mouth or the tongue.

The ulcers, which usually last up to two weeks, are painful and may make eating or speaking difficult. About half of RAU patients experience recurrences every one to three months; as many as 30 percent report continuous recurrences.

RAU major accounts for 7 to 20 percent of cases and usually appears as one to 10 larger coalescent ulcers at a time, which can persist for weeks or months. Herpetiform RAU has been reported as occurring in 7 to 10 percent of RAU cases.

The ulcers appear in crops of 10 to 100 at a time, concentrating in the back of the mouth and lasting for seven to 14 days.

This picture shows a canker sore in the mouth on the upper lip.

RAU can begin in childhood, but the peak period for onset is the second decade. About 50 percent of close relatives of patients with RAU also have the condition, and a high correlation of RAU has been noted in identical but not fraternal twins. Associations have been found between RAU and specific genetic markers.

RAU has also been associated with hypersensitivities to some foods, food dyes and food preservatives. Nutritional deficiencies – especially in iron, folic acid, various B vitamins or combinations thereof – have also been reported, and improvements noted with suitable dietary supplements.

The two factors that have been found to have the strongest association with RAU are immunologic abnormality, possibly involving autoimmunity and trauma.

Volunteers with and without a history of RAU were studied for their reaction to the trauma of a needle prick to the inner cheek tissue. No ulcers developed in non-RAU subjects, but nearly half of those prone to canker sores had a recurrence.

RAU also can occur in a number of systemic diseases, including HIV infection, ulcerative colitis, Crohn’s disease and Behçet’s disease. In general, people who are immunocompromised are more susceptible to RAU, as are people with a variety of blood diseases.

RAU itself does not give rise to other illnesses but is uncomfortable. Symptomatic treatment includes topical analgesics, antibacterial rinses, topical corticosteroids and a new prescription medication that reduces pain and healing time.

Developmental disorders
The importance of the face as the bearer of identity, character, intelligence and beauty is universal. Craniofacial birth defects, which can include such manifestations as cleft lip or palate, eyes too closely or widely spaced, deformed ears, eyes mismatched in color and facial asymmetries, can be devastating to the parents and child affected. Surgery, dental care, psychological counseling and rehabilitation may help to ameliorate the problems but often at great cost and over many years.

Although each developmental craniofacial disease or syndrome is relatively rare, the
number of children affected worldwide is in the millions. In addition, craniofacial defects form a substantial component of many other developmental birth defects, largely because they occur very early in gestation, when many of the same genes that orchestrate the development of the brain, head, face and mouth are also directing the development of the limbs and many vital internal organs, such as the heart, lungs and liver.

By about the third week after fertilization, the three germ layers of the embryo – the ectoderm, endoderm and mesoderm – have formed, as well as the first of four sets of paired swellings – the branchial arches – that appear at the sides of the head end of the embryo. Some craniofacial defects result from failure of the arches to complete their morphogenetic development. Other craniofacial defects are the result of the abnormal differentiation of cells derived from the ectoderm and endoderm or from ectomesenchyme cells, which originate in a part of the ectoderm (the neural crest), in interaction with future connective tissue (the mesenchyme).

Craniofacial anomalies caused by altered branchial arch morphogenesis

Cleft lip/palate and cleft palate
The most common of all craniofacial anomalies and among the most common of all birth defects, are clefts of the lip with or without cleft palate and cleft palate alone; these occur at a rate of 1 to 2 out of 1,000 births, resulting in more than 8,000 affected newborns every year. Cleft lip/palate and cleft palate are distinct conditions with different patterns of inheritance and embryological origins. The male to female ratio of cleft lip/palate is 2 to 1; the ratio for cleft palate alone is just the reverse, 1 to 2.

These anomalies result from the failure of the first branchial arches to complete fusion processes. Clefting can occur independently or as part of a larger syndrome that may include mental retardation and defects of the heart and other organs. Not all cases of clefting are inherited; a number of teratogens (environmental agents that can cause birth defects) have been implicated, as well as defects in essential nutrients such as folic acid. Smoking by the mother during pregnancy also increases the risk. It is becoming increasingly evident that most diseases and disorders, not just craniofacial anomalies, result from interactions involving multiple genes and environmental factors.

Infants with clefts have difficulty with vital oral functions such as feeding, breathing, speaking and swallowing. They are also susceptible to repeated respiratory infections. As these children grow, they must cope with the social consequences of a facial deformity, delayed and altered speech, frequent illness and repeated surgeries that may persist through late adolescence.

Current molecular epidemiology investigations have examined both syndromic and nonsyndromic (isolated) cleft lip/palate and cleft palate. Linkage studies have identified a number of candidate genes, including MSX1, RAR, an X-linked locus and the genes for TGF-beta-3 and TGF-alpha. The pattern of inheritance in cleft lip/palate and cleft palate suggests that between 2 and 20 genes may be involved, with one gene representing a major component in the development of the cleft. One of the common syndromic forms of cleft lip/palate, the Van der Woude syndrome, is caused by an autosomal dominant form of inheritance at a locus on chromosome 1. Future molecular genetic studies will be needed to provide the information necessary for prenatal diagnosis, calculation of risk, and potential gene therapy.

The Treacher Collins syndrome – mandibulofacial dysostosis
Children with the Treacher Collins syndrome have downward-sloping eyelids; depressed cheekbones; a large fishlike mouth; deformed ears with conductive deafness; a small, receding chin and lower jaw; a highly arched or cleft palate; and severe dental malocclusion. These defects result from defective cranial neural crest cell differentiation, migration and proliferation. Consequently, the first branchial arch structures are deficient, and all derivative craniofacial components are affected.

The underdeveloped facial structures can contribute to airway blockage and repeated upper respiratory infections, either of which can be fatal. The faulty development of the ears leads to a conductive deafness. The severe facial deformities exacerbate the psychological difficulties these youngsters face.

Investigators have identified the gene involved in an autosomal dominant form of the syndrome. The function of the gene is not yet known, but its identity will provide opportunities for prenatal diagnosis, gene therapy and further understanding of craniofacial development.

The Pierre Robin syndrome
Deficient development of the first-branchial-arch-derived mandibular portion results in the lower jaw being set far back in relation to the forehead. As a result, the tongue is set back and may obstruct the posterior airway, compromising respiration and, in severe cases, leading to inadequate aeration and failure to thrive. The infant is also at risk for the development of cor pulmonale, an enlargement of the right ventricle of the heart that occurs secondarily to a chronic lung condition. Cleft palate may be another consequence.

The DiGeorge/Velocardiofacial syndrome
The primary defect in the DiGeorge syndrome results from altered development of the fourth branchial arch and the third and fourth pharyngeal pouches. Deficiencies affecting the thymus, parathyroid glands and the great vessels that derive from these structures result. The facial features are subtle and include a squared-off nasal tip, small mouth and widely spaced eyes. Similar facial features, along with heart defects, are seen in the velocardiofacial syndrome. Both syndromes are associated with deletions on the long arm of chromosome 22 (22q11). Further characterization of this chromosomal deletion region will provide information on the specific gene(s) affected and its function in craniofacial development.

The thymus defects severely compromise cellular immunity, depriving the body of thymus-derived T cells and paving the way for severe infectious disease. Inadequate or missing parathyroid glands cause severe hypocalcemia (low blood calcium levels) and seizures. The great vessel abnormalities alter cardiac output and lead to compromised circulation to heart tissues.

Cranial bone and dental anomalies
Defects in the timing of developmental events can cause premature fusion of cranial bones. Impairments of tooth development can result from interruptions of the developmental sequence at several different stages.

This picture is of a child with a facial deformity caused by a cleft lip/palate.

Craniosynostoses
Some craniofacial anomalies are associated with so-called master genes that orchestrate a program by which the embryo assumes its basic shape. Craniosynostosis, which occurs in approximately 1 out of 3,000 births, is one such anomaly. It results in the premature fusion of the cranial sutures, a dangerous condition that puts pressure on the developing brain. A number of diseases and syndromes, including Crouzon’s, Apert’s Boston-type craniosynostosis, Pfeiffer’s and Saethre-Chotzen, share this anomaly, but differ in other features, which can include structural defects such as webbing of the hands and feet as well as mental retardation. Boston-type craniosynostosis has been linked to MSX2, one of the master genes. Several of the other syndromes involve point mutations at one or another locus in genes that code for fibroblast growth factor receptors (FGFR 1, 2 and 3). Collectively, these genes are associated with cell regulation, either through mediating growth factor effects or by serving as transcription factors.

Hereditary hypodontia or anodontia
Conditions of underdeveloped teeth (hypodontia) or their complete absence (anodontia) have been correlated with specific genes, such as MSX1 and...
The complete absence of teeth alters the bony development of the mandible and maxilla.

Amelogenesis imperfecta and dentinogenesis imperfecta
Amelogenesis imperfecta and dentinogenesis imperfecta are linked to defects in structural genes that code for proteins essential to the development of tooth enamel (amelogenesis imperfecta) or dentin (dentinogenesis imperfecta). The teeth are weak and extremely sensitive to temperature and pressure. The ordinary forces of chewing are painful and can lead to further wear and pain.

The enamel matrix genes include tuftelin, ameloblastin, and amelogenin; researchers have begun to link mutations in these genes with amelogenesis imperfecta. Similarly, genes labeled DSP and DPP have been characterized for dentin matrix and are associated with the inheritance of dentinogenesis imperfecta.

Craniofacial defects secondary to other developmental disorders
A number of genetic diseases occur in which craniofacial defects are secondary to a more generalized structural defect.

This picture shows Treacher Collins Syndrome patients with jaw deformity.

Osteogenesis imperfecta
Inherited mutations of collagen genes lead to a number of “brittle bone” diseases characterized by defects in mineralized tissues that form from a collagen-rich matrix. Osteogenesis imperfecta presents a spectrum of deficiencies that includes fragile bones, clear or blue sclera, deafness, loose ligaments, and painful dentinogenesis imperfecta-like changes in the teeth.

Epidermolysis bullosa – recessive dystrophic type
The gene defect in epidermolysis bullosa – recessive dystrophic type – manifests as blisters or bullae that appear shortly after birth on skin areas following minor trauma. Mutations in keratin genes that contribute to the epithelial cell cytoskeleton have been correlated with this condition.

The oral manifestations include both mucosal bullae and altered teeth. Altered teeth with hypoplastic enamel develop and exhibit an increased susceptibility to caries. Oral bullae develop from even the slightest mucosal trauma. The condition is painful and dangerous because of the constant risk that the bullae will become infected.

Craniofacial manifestations of single-gene defects
In many craniofacial defects, mutations within a single gene manifest as complex syndromes with varied organ and limb defects as well as facial anomalies.

Ectodermal dysplasias
The ectodermal dysplasias (EDs) are a family of hereditary diseases first observed by Charles Darwin over a century ago. They involve defects in two or more tissues derived from the ectoderm – skin, hair, teeth, nails and sweat glands. The ectodermal structures fail to differentiate properly owing to altered epithelial-mesenchymal signaling. A gene, EDA, at an X-linked locus has been linked to the syndrome, and ongoing research is aimed at determining the function of the gene and the molecular mechanism of the syndromes. More recently, investigators have discovered genes linked to autosomal (i.e., non-sex-linked) forms of ED, displaying both dominant and recessive inheritance. Oral manifestations of the ectodermal dysplasias are associated with the teeth. Alterations in tooth development can include hypodontia, anodontia and conically shaped teeth.

The Waardenburg syndrome
The Waardenburg syndrome has been subdivided into several types. All involve a variety of abnormalities in the position and appearance of the nose and eyes, with pigment changes that may cause one eye to differ in color from the other. Other signs include deafness, a mildly protruding jaw, cleft lip and palate, and skeletal deformities. The syndrome is inherited in an autosomal dominant manner with complete penetrance and variable expression. Specific genes associated with this syndrome are members of the homeobox family that regulate the transcription of other genes: Waardenburg type 1 with PAX3; Waardenburg type 2 with MITF, 3q14.1; and Waardenburg type 3 with PAX3, 2q35.

Cleidocranial dysplasia
The inheritance of a regulatory gene defect in cleidocranial dysplasia leads to features that include delayed tooth eruption, supernumerary teeth, altered or missing collarbones, short stature and possible failure of cranial suture closure. The exact mechanism of the associated gene, CBFA1, located on chromosome 6, has not been determined but appears to be essential for bone development.

Injury
The common perception is that injuries are random occurrences that are unpredictable and hence preventable. In actuality, experts in the field make the point that there are no basic scientific distinctions between injury and disease. Injuries have been categorized as “intentional” and “unintentional.” People identified as being at risk for certain injuries, as well as the causes of those injuries, can be targeted for appropriate prevention strategies. Such an approach is broadly applicable to sports, falls and motor vehicle injuries (unintentional) as well as to injuries caused by abusive and violent behaviors (intentional).

Injuries are a major public health problem, outranking cancer and heart disease as a leading cause of death in some age groups of the population. Cranial injuries in particular are a leading cause of mortality. Oral-facial injuries can bring disfigurement and dysfunction, greatly diminishing the quality of life and contributing to social and economic burdens.

The leading causes of oral and craniofacial injuries are sports, violence, falls and motor vehicle collisions. Oral cavity injuries may also be caused by foreign objects in food.

Sports
Craniofacial sports injuries occur not only in contact sports, but also in individual activities such as bicycling, skating, and gymnastics, especially on trampolines. Each sport predisposes its participants to a specific array of extrinsic risk factors. These include physical contact, projectiles such as balls and pucks, and the quality of the playing field and equipment.

In contact sports the absence of protective equipment such as headguards and mouthguards is a major risk factor. In a recent survey of school-aged children in organized sports, football was the only sport in which the majority of participants used mouthguards and headgear.

There are intrinsic risk factors as well, relating to characteristics of the individual participant. These include age, sex, injury history, body size, aerobic fitness and muscle strength, central motor control, and general mental ability.

Falls
Falls are a major cause of trauma to teeth, primarily to incisors. Unlike bone fractures, fractures of the crowns of the teeth do not heal or repair, and affected teeth often have an uncertain prognosis. Problems may develop later due to damage to the pulp.

Motor vehicle collisions
The effects of motor vehicle collisions may range from minor and reversible effects to long-term medical, surgical and rehabilitative consequences. Post-traumatic headaches and chronic oral-facial pain can occur. Neuromuscular and glandular damage may cause short- or long-term problems with chewing, swallowing and tearing or result in facial tics or paralysis.

Violence
The family is the single most frequent locus of violence in Western society. Domestic violence includes child abuse, spousal and elder abuse, and abuse of the disabled. Child abuse is of particular concern to the oral health community because 65 percent of cases involve head and
oral-facial trauma and dentists are required to report suspected cases of child abuse. In the young child, head injury is the most common cause of death.

Psychological trauma from abuse can result in sleep disturbances, eating disorders, developmental growth failure in young children and nervous habits such as lip and fingernail biting and thumb sucking. Effects may also include chronic underachievement in school and poor peer relationships. In abusive families, physical neglect is commonplace, with inadequate provision of basic needs, including medical and oral health care.

Selected chronic pain conditions
Oval, dental or craniofacial signs and symptoms play a critical role in autoimmune disorders such as Sjögren’s syndrome and in a number of chronic and disabling pain conditions.

Sjögren’s syndrome
Sjögren’s syndrome is one of several autoimmune disorders in which the body’s own cells and tissues are mistakenly targeted for destruction by the immune system. Like other autoimmune conditions, Sjögren’s syndrome is more prevalent among women. The ratio of females to males affected is 9 to 1, with symptoms usually developing in middle age. There are an estimated 1 million to 2 million individuals in the United States with Sjögren’s syndrome.

The disease occurs in two forms. Primary Sjögren’s involves the salivary and lacrimal (tear) glands. In secondary Sjögren’s the glandular involvement is accompanied by the development of a connective tissue or collagen disease, most often rheumatoid arthritis, lupus erythematosus, scleroderma or biliary cirrhosis.

The glandular involvement causes a marked reduction in fluid secretion, resulting in xerostomia and xerophthalmia (dry eyes). The constant oral dryness causes difficulty in speaking, chewing and swallowing; the dry eyes often itch and feel gritty. There is no cure for Sjögren’s, and patients often carry eyedrops and artificial tears, some medications, such as pilocarpine and cevimeline, are prescribed to increase salivary flow from the residual healthy gland tissue, again providing symptomatic relief only. The problems that develop in the other organ systems are also treated symptomatically.

At advanced stages, steroids are employed intermittently to alleviate problems.

Acute and chronic oral-facial pain
Since the 19th century when two dentists, Horace Wells and Frederick Morton, demonstrated the analgesic powers of nitrous oxide and ether, oral health investigators have been recognized leaders in the field of pain management worldwide. Their analyses of the cells, pathways and molecules involved in the transmission and modulation of pain have given rise to a growing variety of medications, often combined with other approaches, that can control acute and chronic pain. Pain researchers today stress that chronic pain can become a disease in itself, causing long-term detrimental changes in the nervous system. These changes may affect resistance to other diseases as well as effectively destroy quality of life. Most people have experienced acute pain involving teeth and the oral tissues at one time or another.

Atypical facial pain
Atypical facial pain is characterized by a continuous dull ache on one or both sides, most frequently in the region of the maxilla (the upper jaw). The pain tends to be episodic and is aggravated by fatigue, worry or emotional upset. It is often accompanied by pain elsewhere in the body and depression. Once a dental cause can be ruled out, pain resolution depends on the successful use of antidepressants, psychotherapy or both.

Tic douloureux
The oral-facial region is also subject to pain that can be paroxysmal or continuous along a distinct nerve distribution. The most frequently encountered of these oral facial neuralgias is tic douloureux, or trigeminal neuralgia, a disease of unknown etiology affecting one, two or all three branches of the trigeminal nerve. The pain is highly intense and of a stabbing nature, and lasts for a few seconds. This transient attack may be repeated every few minutes or several hours. There may be no precipitating factor, or it may occur in response to a gentle touch or a breeze wafting across the face—a condition experts call allodynia, the feeling of pain in response to a normally nonpainful stimulus. On other occasions, there may be a specific trigger zone. Although spontaneous remission for weeks or months may occur, it is rarely permanent.

Given the unknown, unpredictable nature of tic douloureux, it is not surprising that fear of pain comes to dominate these patients’ lives, as they avoid doing anything that might trigger an attack.

Trigeminal neuralgia generally occurs in later life, but also occurs in younger individuals affected by multiple sclerosis, where it is assumed to be associated with lesions (multiple sclerosis “plaques”) in the brain stem. Medical treatment depends largely on the use of a drug that has become a virtual specific, the antiepileptic drug carbamazepine. For those patients with no consequential adverse effects, it can control the disease. An alternative for chronic sufferers is the surgical removal of a small vein or artery that may be exerting pressure on the nerve root or the selective destruction of the nerve fibers themselves using chemical or electrical methods. In many cases, these procedures can produce complete relief from pain.

Temporomandibular disorders
Various etiological factors, including trauma, can give rise to pain and dysfunction in the temporomandibular joint and surrounding muscles, conditions collectively called temporomandibular disorders (TMDs). The pain may be localized or radiate to the teeth, head, ears, neck, and shoulders. Abnormal grating, clicking or cracking sounds, known as crepitus, in the joint often accompany localized pain. Pain is also found in response to clinical palpation of the affected structures. TMDs are common, occurring in as many as 10 million Americans. Although surveys indicate that both sexes are affected, the majority of individuals seeking treatment are women of childbearing age, a phenomenon suggesting that hormonal influences should be investigated.

Several factors can contribute to the onset or exacerbation of TMD symptoms. These factors include:

- Certain developmental anomalies.
- Injury to the jaw from accidents or abuse.
- Oral habits that greatly stress the joint and musculature, such as tooth grinding (bruxism).
- Jaw manifestations of systemic diseases such as fibromyalgia and arthritic diseases; and some irreversible treatments for initial signs and symptoms.

The multiplicity of factors that may cause or contribute to TMDs has unfortunately led to...
a multiplicity of treatments. Most of these treatments have not been tested in randomized controlled clinical trials. During the 1970s and 1980s, many individuals underwent surgery that proved unsuccessful in many cases.

Leading investigators have proposed standardized research diagnostic criteria to clarify the kinds of pathology that can give rise to TMDs and to classify the most common forms of TMDs. Such criteria could be used in designing clinical trials and could ultimately lead to better diagnostics, treatments and prevention.

The criteria use two dimensions or axes:
- Axis I delineates various forms of joint or muscle pathology.
- Axis II explores pain-related disability and psychological status.

The approach requires detailed clinical examinations and patient histories.

A mirror, a model and a better understanding of diseases and disorders

Studying the diseases and disorders that affect craniofacial tissues can provide scientists with models of systemic pathology. Because some craniofacial tissues, such as bones, mucosa, muscles, joints and nerve endings, have counterparts in other parts of the body and these tissues are often more accessible to research analysis than deeper-lying tissues, researchers studying craniofacial tissues can gain valuable insights into how cancer develops, the role of inflammation in infection and pain, the effects of diet and smoking, the consequences of depressed immunity and the changes that can arise from a mutated gene.

Other craniofacial tissues, teeth, gingiva, tongue, salivary glands and the organs of taste and smell, are unique to the craniofacial complex. Study of the diseases affecting these tissues has revealed a wealth of information about their special nature as well as the molecules and mechanisms that normally operate for the protection, maintenance and repair of all the oral, dental and craniofacial tissues. When factors perturb these nurturing elements, the oral health scale can tip toward disease. When those factors stem from systemic diseases or disorders, the mouth can sometimes mirror the body’s ill health.

Similarly underscoring the connection between oral and general health are studies suggesting that poor dental health, mainly due to chronic dental infections, may heighten the risk for both cardiovascular disease and stroke independently of factors such as social class and established cardiovascular risk factors.

Infection and disorder highlights

- Microbial infections, including those caused by bacteria, viruses and fungi, are the primary cause of the most prevalent oral diseases. Examples include dental caries, periodontal diseases, herpes labialis and candidiasis.
- The etiology and pathogenesis of diseases and disorders affecting the craniofacial structures are multifactorial and complex, involving an interplay among genetic, environmental and behavioral factors.
- Many inherited and congenital conditions affect the craniofacial complex, often resulting in disfigurement and impairments that may involve many body organs and systems and affect millions of children worldwide.
- Tobacco use, excessive alcohol use and inappropriate dietary practices contribute to many diseases and disorders. In particular, tobacco use is a risk factor for oral cavity and pharyngeal cancers, periodontal diseases, candidiasis and dental caries, among other diseases.
- Some chronic diseases, such as Sjögren’s syndrome, present with primary oral symptoms.
- Oral-facial pain conditions are common and often have complex etiologies.

The range of oral, dental and craniofacial diseases and conditions that take a toll on the U.S. population is extensive. This course provides highlights of conditions and diseases affecting Americans using available national and state data to describe the burden of disease in the United States. There is no single measure of oral health or the burden of oral diseases and conditions, just as there is no single measure of overall health or overall disease. The relationship of oral health to the use of dental services is described. However, the effects of health care visits and of specific services rendered need further study.

Physical signs and symptoms of disease and risk factors

A number of signs and symptoms of disease, lifestyle behaviors and exposure to toxins can be detected in or around the craniofacial complex. Pathogens entering the mouth may proliferate locally with oral and pharyngeal signs and symptoms; other pathogens may enter the bloodstream directly or through lymphatic channels and cause generalized disease. Oral signs suspected to be indications of systemic illness may be confirmed by the presence of rash, fever, headache, malaise, enlarged lymph nodes or lesions elsewhere on the body.

Swollen parotid glands are a cardinal sign of infection with the mumps virus and can also be seen in individuals with Sjögren’s syndrome and HIV. The salivary glands are also frequently involved in tuberculosis and histoplasmosis infections. Oral signs of infectious mononucleosis, caused by Epstein-Barr virus, include sore throat, gingival bleeding and multiple pinpoint-sized hemorrhagic spots (petechiae) on the oral mucosa. There can be a large overlap in the clinical appearance of oral manifestations of various diseases with different etiologies, and the clinical diagnosis often involves ancillary procedures, which may include laboratory tests, diagnostic imaging and biopsy.

Oral tissues may also reflect immune deficiency. For example, nearly all HIV-infected individuals develop oral lesions at some time during their illness. Other immunosuppressed individuals may have the same lesions. However, the presentation and the extent, severity and management of some of these lesions may reflect nuances due to variation in the underlying systemic condition. For example, the linear gingival erythema and necrotizing ulcerative periodontitis sometimes seen in HIV infection have been difficult to resolve with routine dental curettage and prophylaxis.

The appearance of soft or hard tissue pigmentation is associated with a number of diseases and treatments. Malignant melanoma can appear in the mouth as brown or black flat or raised spots. Kaposi’s sarcoma can appear as a flat or raised pigmented lesion. Addison’s disease causes blotsches or spots of bluish-black or dark brown pigmentation to occur early in the disease. Congential discrete brown or black patches (nev) can appear in any part of the mouth. Pigmentation of the tooth crowns may be seen in children with cystic fibrosis and porphyria and those exposed to tetracycline during tooth development.

The oral tissues can also reflect nutritional status and exposure to risk factors such as tobacco. The tongue appears smooth in pernicious anemia. Group B vitamin deficiency is associated with oral mucositis and ulcers, glossitis, and burning sensations of the tongue. Scurvy, caused by severe vitamin C deficiency, is associated with gingival swelling, bleeding, ulceration and tooth loosening. Lack of vitamin D in utero or infancy impairs tooth development. Enamel hypoplasia may result from high levels of fluoride or from disturbances in calcium and phosphate metabolism, which can occur in hypoparathyroidism, gastroenteritis, and celiac disease. The mouth also can reflect the effects of tobacco use, perhaps providing the only visible evidence of its adverse effects.

Oral manifestations of HIV infection and of osteoporosis

The mouth can serve as an early warning system, diagnostic of systemic infectious disease and predictive of its progression, such as with HIV infection. In the case where oral cells and tissues have counterparts in other parts of the body, oral changes may indicate a common pathological process. During routine oral examinations and perhaps in future screening tests, radiographic or magnetic resonance imaging of oral bone may be diagnostic of early osteoporotic changes in the skeleton. The following sections provide details.

HIV infection

The progressive destruction of the body’s immune system by HIV leads to a number of oral lesions, such as oral candidiasis and oral hairy leukoplakia, that have been used not only in diagnosis but also in determining specific stages of HIV infection. Oral candidiasis is rarely seen in previously healthy young adults who have not received prior medical therapy such as cancer chemotherapy or treatment with other immunosuppressive drugs. It was associated with AIDS as early as 1981 in the first report of the syndrome and was frequently noted among otherwise asymptomatic HIV-positive populations. Oral candidiasis may be the first sign of HIV infection and often occurs
as part of the initial phase of infection, the acute HIV syndrome. It tends to increase in prevalence with progression of HIV infection when CD4 lymphocyte counts fall. It also appears to be the most common oral manifestation in pediatric HIV infection and has been demonstrated to precede to esophageal candidiasis, a sign of overt AIDS. Both the pseudomembranous and the erythematous forms of candidiasis appear to be important predictors of progression of HIV infection.

Like oral candidiasis, oral hairy leukoplakia in HIV-positive persons heralds more rapid progression to AIDS. Oral hairy leukoplakia is an oral lesion first reported in the early days of the AIDS epidemic. Since its discovery, hairy leukoplakia has been found in HIV-negative persons with other forms of immunosuppression, such as organ or bone marrow recipients and those on long-term steroid therapy and less frequently among immunocompetent persons.

Osteoporosis, a degenerative disease characterized by the loss of bone mineral and associated structural changes, has long been suspected as a risk factor for oral bone loss. In addition, measures of oral bone loss have been proposed as potential screening tests for osteoporosis. Osteoporosis affects over 20 million people in the United States, most of whom are women, and results in nearly 2 million fractures per year. The disease is more prevalent in white and Asian American women than in black women. Oral bone loss has been reported to be more prevalent in women than in men.

**Oral-fluid-based diagnostics: The example of saliva**

The diagnostic value of salivary secretions to detect systemic diseases has long been recognized, and oral fluids and tissues (buccal cells) are increasingly being used to diagnose a wide range of conditions. Saliva- and oral-based diagnostics use readily available samples and do not require invasive procedures. Researchers have detected antibodies in saliva that are directed against viral pathogens such as human immunodeficiency virus and hepatitis A virus or B virus. Saliva is being used to detect antibodies, drugs, hormones, and environmental toxins. The simplest tests are those that detect the presence or absence of a substance in the saliva, such as various drugs. Greater technical challenges are presented for tests that will be used for therapeutic monitoring since accurate levels of a substance and/or its metabolites are needed. In these instances the saliva/plasma concentration ratio must be determined experimentally. Most recently, oral fluids have been used as a source of microbial or host DNA.

Saliva has the potential of replacing blood, the current standard for testing many diseases and conditions (e.g., diabetes, infectious disease, Parkinson’s disease, alcoholic cirrhosis, Sjögren’s syndrome and cystic fibrosis sarcoidosis). Important goals for the future are the development of new diagnostic tests for early disease detection, defining individual patient risk of adverse response to drugs, monitoring therapeutic progress and determining outcomes of treatment. Key issues in the development of a new generation of saliva diagnostics include their selectivity, sensitivity, response time, dynamic range (values of interest), representative sampling and, perhaps most important, their reliability or stability as well as their ability to assess multiple substances simultaneously.

For the clinician, the mouth and face provide ready access to physical signs and symptoms of local and generalized disease and risk factor exposure. These signs and symptoms augment other clinical features of underlying conditions. Comprehensive care of the patient requires knowledge of these signs and symptoms, their role in the clinical spectrum of general diseases and conditions, and their appropriate management. Oral biomarkers and surrogate measures are also being explored as means of early diagnosis. With further development and refinement, oral-based diagnostics such as salivary tests can become widely used and acceptable tools for individuals, health care professionals, researchers and community programs. The continued refinement of imaging techniques also has the potential of using oral imaging to identify early signs of skeletal bone degeneration.

**The mouth as a portal of entry for infection**

More than 500 bacterial strains have been identified in dental biofilm, and more than 150 bacterial strains have been isolated from dental pulp infections. More recently, 37 unique and previously unknown strains of bacteria were identified in dental plaque (biofilm). Most oral lesions are opportunistic infections – that is, they are caused by microorganisms commonly found in the mouth, but normally kept in check by the body’s defense mechanisms. These microorganisms can induce extensive localized infections that compromise general well-being in and of themselves.

However, they also may spread to other parts of the body if normal barriers are breached. The oral mucosa is one such barrier that provides critical defense against pathogens and other challenges. Salivary secretions are a second major line of defense. Damage to the oral mucosa from mechanical trauma, infection or salivary dysfunction with resulting derangements in lubricatory and antimicrobial functions of saliva as a result of chemotherapy, radiation and medications causing hyposalivation, allows a portal of entry for invading pathogens.

**Oral infections and bacteremia**

Oral microorganisms and cytotoxic byproducts associated with local infections can enter the bloodstream or lymphatic system and cause damage or potentiate an inappropriate immune response elsewhere in the body. Dissemination of oral bacteria into the bloodstream (bacteremia) can occur after most invasive dental procedures, including tooth extractions, endodontic therapy, periodontal surgery and scaling and root planing. Even routine oral hygiene procedures such as daily toothbrushing, subgingival irrigation and flossing may cause bacteremia. However, these distant infections have been seen more often in high-risk patients such as those who are immunocompromised.

Oral bacteria have several mechanisms by which they invade mucosal tissues, perhaps contributing to their ability to cause bacteremias. For example, oral bacteria and their products may invade the peridontal tissues directly. Actinobacillus actinomycetemcomitans has been found in gingival connective tissue in patients with localized juvenile periodontitis. Invasion of tissue by Porphyromonas gingivalis has also been described in vivo and in vitro. Although oral bacteria can enter the blood through injured or ulcerated tissue, bacterial invasion of periodontal tissues represents another possible mechanism.

In the immunocompetent individual, bacteremia originating from the oral cavity is usually transient and harmless. However, if the individual’s immune system is compromised, the normally harmless oral bacteria may pose a significant risk. The morbidity and mortality associated with oral foci of infections are hard to assess. This is due to the formidable task of tracking the source of an infection unless the responsible pathogen is indigenous to a specific anatomic location.

Viridans group streptococci (VGS) have a low degree of virulence but can be associated with morbidity and mortality under certain circumstances. Increased pathogenicity of Streptococcus viridans is most prominent in individuals with neutropenia (low blood counts of circulating white blood cells called neutrophils) and has been associated with a toxic-shock-like syndrome (TSLS) or viridans streptococcal shock syndrome (VSSS), as well as with adult respiratory distress syndrome (ARDS). Although a high degree of morbidity is associated with viridans streptococcal bacteremia, a low incidence of mortality has been reported. Several studies have shown that under adverse circumstances, oral flora and oral infections are associated with increased incidence of morbidity. Reduction of oral foci of infection decreases systemic complications, specifically in severely neutropenic patients undergoing chemotherapy. In addition, hospital stays for patients with oral mucositis undergoing autologous bone marrow transplants were longer than for those without oral mucositis.

Other cohorts identified at increased risk for systemic complications due to oral bacteria include hospitalized patients unable to perform adequate oral hygiene, those receiving salivareducing medications and those taking antibiotics that alter the oral flora. A positive dental plaque culture for aerobic pathogens was significantly associated with the development of hospital-acquired pneumonia and bacteremia in a study of individuals in an intensive care unit.

In addition, several case reports have been published implicating indigenous oral flora in the development of brain abscesses. This serious condition is associated with a mortality rate of
 almost 20 percent, and full recovery in only slightly more than 50 percent of all patients. These data are based on single case reports and most probably represent rare events. However, they provide additional examples that point to the potential pathogenicity of the normal oral flora during special adverse circumstances.

Oral infections as a result of therapy

Chemotherapy

Oral mucositis can be a major dose-limiting problem during chemotherapy with some anticancer drugs, such as 5-fluorouracil, methotrexate, and doxorubicin. It is estimated that approximately 400,000 patients undergoing cancer therapy each year will develop oral complications (NIH 1990). Infection of ulcerated mucous membranes often occurs after chemotherapy, especially since patients are usually immunocompromised. Bacterial, fungal, and viral causes of mucositis have been identified.

The mechanism by which cancer-chemotherapy-induced mucositis occurs is likely associated with the rapid rate of turnover of oral epithelial cells. In addition, other components likely include upregulation of pro-inflammatory cytokines and metabolic by-products of colonizing oral microflora. Chemotherapy alters the integrity of the mucosa and contributes to acute and chronic changes in oral tissue and physiologic processes. The ulcerated mucosa is susceptible to infection by microbial flora that normally inhabit the oral cavity, as well as by exogenous organisms, and exacerbates the existing mucositis. Further, these microflora can disseminate systemically. Compromised salivary function can further elevate risk for systemic infection of oral origin.

Both indigenous oral flora and hospital-acquired pathogens have been associated with bacteremias and systemic infection. Changes in infection profiles in myelosuppressed (immunosuppressed) cancer patients tend to occur in cyclic fashion over many years. This evolving epidemiology is caused by multiple factors including use of antibiotics. Gram-positive organisms including viridans streptococci and enterococci are currently associated with systemic infection of oral origin in myelosuppressed cancer patients. In addition, gram-negative pathogens including P. aeruginosa, Neisseria spp., and Escherichia coli remain of concern.

Cancer patients undergoing bone marrow radiation who have chronic periodontal disease may also develop acute periodontal infections with systemic complications. The extensive ulceration of gingival sublethal epithelium associated with periodontal disease is not directly observable clinically, yet may represent a source for disseminated infection by an extensive array of organisms. Inflammatory signs may be masked due to the underlying bone marrow suppression.

Viruses are also associated with clinically important oral disease in patients receiving chemotherapy. Infections caused by herpes simplex virus, varicella-zoster virus and Epstein-Barr virus typically result from reactivation of a latent virus, whereas cytomegalovirus infections can result via reactivation of a latent virus or a newly acquired virus. The severity of the infection, including fatal outcome, depends on the degree of immunocompromise.

Radiation therapy

Radiation therapy disrupts cell division in healthy tissue as well as in tumors and also affects the normal structure and function of craniofacial tissues, including the oral mucosa, salivary glands and bone. Oral-facial complications are common after radiation therapy to the head and neck. The most frequent, and often the most distressing, complication is mucositis, but adverse reactions can affect all oral-facial tissues.

Radiation can cause irreversible damage to the salivary glands, resulting in dramatic increases in dental caries. Oral mucosal alterations may become portals for invasion by pathogens, which may be life-threatening to immunosuppressed or bone-marrow-suppressed patients. A less common but very serious adverse consequence is destruction of bone cells and bone death, called osteoradionecrosis (ORN). ORN can result in infection of the bone and soft tissue and can require surgery to excise the dead tissue, which can in turn leave the face badly disfigured as well as functionally impaired. The likelihood of ORN is increased with trauma to the bone, including that caused by tooth extraction. The risk is especially marked when the trauma occurs near the time of radiation. Management includes elimination of acute or potential dental and periodontal foci of disease, increased patient participation in oral hygiene, use of oral topical fluorides for caries prevention and use of antiviral, antifungal or antimicrobial therapy for management of infections associated with mucositis.

Combined cancer therapies

Rapid developments have occurred in the use of blood cell growth factors for treatment of various conditions, including the anemia of end-stage renal disease, the neutropenia occurring with cancer care and the bone marrow toxicity and mucositis that can follow aggressive chemotherapy or radiation therapy. Researchers have found that topical application of transforming growth factor beta (TGF-B) in the hamster model of oral mucositis significantly reduced basal cell proliferation and reduced the severity of mucositis associated with 5-fluorouracil treatment.

Other growth factors considered for use in reducing mucositis include granulocyte-monocyte colony-stimulating factor and granulocyte colony-stimulating factor. Bone morphogenetic proteins are also in development for alleviating the toxicity and mucositis that follow chemotherapy and radiation therapy. Other approaches to reducing mucositis and adverse oral effects of chemotherapy and radiation therapy include fractionating the dose of radiation and combining chemotherapy with growth factors or with less toxic oncostatic agents.

Although the oral mucositis occurring in chemotherapy and in head and neck radiation patients shares many characteristics, distinct differences also exist. For example, in contrast to chemotherapy-associated lesions, radiation damage is anatomically site-specific; toxicity is localized to irradiated tissue volumes. The degree of damage depends on treatment-regimen-related factors, including the type of radiation used, the total dose administered, the fractionation, and field size. Thus, research involving both cohorts of cancer patients remains essential to enhancing patient management.

Development of new technologies to prevent cancer-therapy-induced oral mucositis could substantially reduce the risk for oral and systemic infections, oral pain and the number of hospital days. Improvement in quality of life and reduction in health costs are also likely and desirable outcomes.

The new technologies could also provide a setting in which novel classes of chemotherapeutic drugs, used at increased doses, could be implemented. These advances in turn could lead to enhanced cancer patient survival and lengthen the duration of disease remission.

Pharmaceuticals

A number of medications used to treat systemic diseases can cause oral complications, ranging from xerostomic effects to alterations in the surface structure of the enamel or mucosa. More than 400 over-the-counter and prescription drugs have xerostomic side effects. These include tricyclic antidepressants, antihistamines and diuretics. The dimensions and impact of these side effects vary depending on the response of the individual patient and the duration of medication use.

Staining of the teeth or mucosa is associated with a variety of drugs, including tranquilizers, oral contraceptives and antimalarials. The antibiotic tetracycline can cause enamel hypoplasia when taken by the mother during pregnancy and by children during tooth development. The antimicrobial mouthrinse agent chlorhexidine also can stain the teeth, but this staining is external and can be removed by dental prophylaxis.

Other drugs have been associated with gingival overgrowth, including cyclosporin, which has been used as an immunosuppressant in the United States since 1984 to prevent rejection of transplanted organs and bone marrow. This drug has also been used in other countries for treatment of type 2 diabetes, rheumatoid arthritis, psoriasis, multiple sclerosis, malaria, sarcoidosis and several other diseases with an immunological basis. Other drugs that cause gingival overgrowth include calcium ion channel blocking agents used in the treatment of angina pectoris and postmyocardial syndrome, such as nifedipine and verapamil, and phenytoin (sodium 5,5-phenylhydantoin), used in the treatment of epilepsy and also for management of other neurological disorders. Treatment often consists of using an alternate drug, although this is not always possible. Conservative periodontal therapy can reduce the inflammatory component of enlargement, but surgery is often required. Drugs that cause systemic bone marrow suppression, oral mucosal injury or salivary compromise collectively promote the risk for clinical infection.
In addition, antibiotics and concurrent steroid therapy often alter oral flora, thereby creating an environment for fungal overgrowth. In high-risk cancer patients, fungal infection can cause severe morbidity and even death.

**Infective endocarditis**
The purported connection between oral infection and a specific heart disease, infective endocarditis, has a long history. Endocarditis is caused by bacteria that adhere to damaged or otherwise receptive surfaces of the tissue that lines heart valves (the endocardium). Dental and other surgical procedures may predispose susceptible patients to infective endocarditis by inducing bacteremias. However, bacteremias from oral infections that occur frequently during normal daily activities, coincidental even with chewing food, toothbrushing and flossing, contribute more substantially to the risk of infective endocarditis. Oral organisms are common etiologic agents of infective endocarditis. For example, strains of S. sanguis, as well as gram-negative oral bacteria including Haemophilus aphrophilus, A. actinomycetemcomitans, E. corrodens, Capnocytophaga spp., and Fusobacterium nucleatum, have been associated with bacterial endocarditis.

Infective endocarditis occurs with different incubation periods, which differ in causative bacteria and signs and symptoms. For example, Staphylococcus aureus endocarditis may have a rapid onset and fatal course if it affects the left side of the heart. With a more indolent course, patients may often be unaware of infection and may experience fever, night chills, myalgia and arthralgia for a considerable period of time before diagnosis. The infection is often curable if diagnosed and treated early.

The classic risk factors for endocarditis include cardiac valve disorders (valvulopathies) that include rheumatic and congenital heart disease, complex cyanotic heart disease in children, and mitral valve prolapse with regurgitation. Recent studies indicate that the use of certain diet drugs (fenfluramine and dexfenfluramine) has induced cardiac valvulopathy, which may in some cases be transient. Among at-risk persons, bacteremias are more likely to occur in those with periodontal disease. However, the oral pathogens causing periodontitis have only rarely been shown to cause endocarditis.

Prevention of infective endocarditis from oral bacteria depends on limiting the entry and dissemination of bacteria through the bloodstream and lymphatic circulation. Antibiotic prophylaxis for dental procedures that are likely to provoke bacteremia has historically been recommended. A recent study, however, suggests that receiving dental treatment does not significantly increase the risk of infective endocarditis, even in patients with valvular abnormalities. Further research is necessary to determine whether some heart or valvular conditions or certain dental procedures, such as surgery or scaling, would require coverage with pre-procedural antibiotics and others would be precluded.

**Oral infections and respiratory disease**
Pathogens in the oral cavity can also gain access to the airway, sometimes with serious consequences. In adults, bacterial pneumonias are strongly associated with aspiration of bacteria into the lower respiratory tract, which is normally sterile. Common respiratory pathogens such as Streptococcus pneumoniae, Streptococcus pyogenes, Mycoplasma pneumoniae and Haemophilus influenzae can colonize the oropharynx and the lower airway. In addition, oral bacteria including A. actinomycetemcomitans, Actinomyces israelii, Capnocytophaga spp., Eikenella corrodens, Prevotella intermedia and Streptococcus constellatus can be aspirated into the lower airways.

Chronic obstructive pulmonary disease, characterized by obstruction of airflow due to chronic bronchitis or emphysema and by recurrent episodes of respiratory infection, has been associated with poor oral health status. Although oral bacteria, including periodontal pathogens, have the potential for causing respiratory infections, the frequency and nature of such infections are not known and merit further study.

**Oral transmission of infections**
Besides being a portal of entry for infections, the mouth is an important source of potentially pathogenic organisms and is often the vehicle by which infection is delivered to the bodies of others. Microorganisms were not discovered in the mouth until the 17th century, when van Leewenhoek examined dental plaque using a microscope he had constructed. In 1884, Koch demonstrated that tuberculosis could be transmitted by airborne droplets from the mouth and respiratory tract. Since that time, we have learned that many common respiratory infections, such as influenza, the common cold, pneumonia and tuberculosis, can be transmitted from oral secretions. Before the development of effective vaccines, orally transmitted diseases such as chickenpox, measles, mumps, polo and diphtheria were a major source of morbidity and mortality in childhood. Viral diseases such as hepatitis B, herpes labialis, acute herpetic gingivostomatitis, cytomegalovirus and infectious mononucleosis may also originate from oral contact.

Disease-causing microorganisms can be spread by direct contact (with saliva or blood from the mouth) or indirect contact (with saliva- or blood-contaminated surfaces, including hands or lips), droplet infection (from coughing, sneezing or even normal speech), or by aerosolized organisms. These organisms can be inhaled, ingested or taken in through mucous membranes in the eyes, nose or mouth or through breaks in the skin. A number of diseases can be spread via oral sexual contact, including gonorrhea, syphilis, trichomoniasis, chlamydia and mononucleosis.

As mentioned earlier, the oral mucosa and saliva provide significant defense against disease transmission. Epidemiological and animal studies are providing evidence, however, that the oral cavity may be the site for transmission of serious systemic infections despite the protective factors in saliva. Infection with HIV provides a case in point.

Early in the 1980s, when AIDS was first identified in the United States, concern was expressed about casual (i.e., nonsexual) transmission of HIV. Detailed household studies did not demonstrate transmission of HIV, even when family members shared eating utensils and toothbrushes with an HIV-affected member. Similarly, surveillance data collected over time showed no evidence of casual transmission.

Only one nonoccasional episode of HIV transmission has been attributed to blood-contaminated saliva; this incident involved intimate kissing between sexual partners. There have been a few cases of HIV transmission from performing oral sex on a person infected with HIV, and it is also possible to become infected with HIV by receiving oral sex. In the San Francisco Options Study of men who have sex with men identified within 12 months of HIV seroconversion, oral transmission represented 7.8 percent of primary HIV infections. Rothenberg et al. (1998) reviewed epidemiologic studies and reports of 38 cases of oral transmission of HIV in the literature. They concluded that although oral-genital contact may be less efficient than needle-sharing or anal intercourse for the transmission of HIV, its increased use by men who have sex with men and in crack cocaine smokers may increase its contribution to HIV transmission over time. Several studies provide evidence that when the oral environment is compromised, the mouth can be a potential site of transmission of infectious microbes. Data suggest that there is a positive association between the presence of oral lesions resulting from crack cocaine use, receptive oral intercourse and HIV transmission. A case report has documented the passage of HIV from a partner who is HIV-positive to one who is HIV-negative in the presence of periodontal disease but in the absence of other risk factors. Because the type, duration and frequency of oral contact in past studies may not have been specified, the risk could be somewhat higher for oral transmission of HIV than previously reported. The risk might also vary depending on factors such as viral load, infectious dose, area of exposure and presence or absence of oral lesions. Additional studies are needed to evaluate the risk of oral-genital transmission of HIV; some are under way.

Other sexually transmitted diseases (STDs) can occur through oral contact. For example, pharyngeal infection with Chlamydia trachomatis has been found in 3 to 6 percent of men and women attending STD clinics. Most infections are asymptomatic. Another common sexually transmitted infection, herpes simplex virus, commonly infects the pharynx and is seen in 20 percent of patients with primary genital herpes. The painless chancre of primary syphilis can be found in the oral cavity; however, there are no data on the prevalence of this site of infection for Treponema pallidum. Among persons with gonorrhea, pharyngeal infection occurs in 3 to 7 percent of heterosexual men, 10 to 20 percent of heterosexual women and 10 to 25 percent of men who have sex with men. Gonococcal infection
can cause acute pharyngitis, but is usually asymptomatic. The transmission of pharyngeal gonorrhea to sex partners had been thought to be rare. However, in one study, 17 of 66 men who had sex with men who had urethral gonorrhea reported insertive oral sex as their only risk factor in the past two months.

The role of the mouth as a portal of entry for infection presents ever-new challenges for study. Although oral tissues and fluids normally provide significant barriers and protection against microbial infections, at times these infections can not only cause local disease but also, under certain circumstances, can disseminate to cause infections in other parts of the body. The control of existing oral infections is clearly of intrinsic importance and a necessary precaution to prevent systemic complications.

Associations among oral infections and diabetes, heart disease/stroke and adverse pregnancy outcomes
Recent studies have reported associations between oral infections, primarily periodontal infections, and diabetes, heart disease and stroke, and adverse pregnancy outcomes, but sufficient evidence does not yet exist to conclude that one leads to the other. This section characterizes the nature of these associations by describing the quality of the evidence supporting the reports. Both observational and experimental studies were accepted as admissible evidence. Where there are operative mechanisms proposed that support an association between oral infectious agents and the systemic conditions in question, they are introduced at the outset.

The periodontal disease-diabetes connection
There is growing acceptance that diabetes is associated with increased occurrence and progression of periodontitis, so much so that periodontitis has been called the “sixth complication of diabetes.” The risk is independent of whether the diabetes is type 1 or type 2. Type 1 diabetes is the condition in which the pancreas produces little or no insulin. It usually begins in childhood or adolescence. In type 2 diabetes, secretion and utilization of insulin are impaired; onset is typically after age 30. Together, these two types of diabetes affect an estimated 15.7 million people in the United States and represent the seventh leading cause of death. The goal of diabetic care is to lower blood glucose levels to recommended levels. Some investigators have reported a two-way connection between diabetes and periodontal disease, proposing that not only are diabetic patients more susceptible to periodontal disease, but also the presence of periodontal disease affects glycemic control.

Effects of diabetes on periodontitis prevalence and severity
Several reviews have described candidate mechanisms to explain why individuals with diabetes may be more susceptible to periodontitis. These include vascular changes, alterations in gingival crevicular fluid, alterations in connective tissue metabolism, altered host immunological and inflammatory response, altered subgingival microflora and hereditary patterns. Studies were classified by type of diabetes and age of study population.

Type 1 diabetes
Ten reports focused principally on children and adolescents with type 1 diabetes, comparing them with groups of similar ages without diabetes. All but one of the studies reported greater prevalence, extent or severity of at least one measure or index of periodontal disease (e.g., gingival inflammation, probing pocket depth, loss of periodontal attachment or radiographic evidence of alveolar bone loss) among subjects with diabetes, even though these investigations were conducted in a variety of countries across several continents.

Another set of studies on the relationship between type 1 diabetes and periodontal disease included subjects with and without diabetes between the ages of 15 and 35. All six studies reported greater prevalence, extent or severity of at least one measure or index of periodontal disease. A third set of studies conducted in Scandinavia looked at the relationship between periodontal disease and type 1 diabetes (or diabetes reported as requiring insulin therapy without specification of diabetes type) in adults between 20 and 70 years old. Three of the four studies were cross-sectional, and one was a treatment follow-up study. All four studies reported greater prevalence, extent or severity of at least one measure of periodontal disease.

Type 2 diabetes
There are fewer reports on the relationship between type 2 diabetes and periodontal disease, particularly where type 2 diabetes is explicitly identified or discernible from the ages of subjects. Seven studies limited to subjects with type 2 diabetes included a comparison group without diabetes. Two of these studies included only adult subjects; the remaining five were large population-based studies of diabetes and periodontal disease in Pima Indians, a group with the highest known prevalence of type 2 diabetes in the world. The Pima Indian studies included subjects aged 5 years and older or 15 and older. All seven studies reported greater prevalence, extent or severity of periodontal disease among subjects with diabetes for at least one measure of periodontal disease. Three of these studies were longitudinal and showed that the progression of periodontal disease was greater in diabetes patients than in individuals without diabetes.

In addition to finding significant differences in various measures of periodontal status between subjects with and without type 2 diabetes, a number of these reports also provide estimates of association and risk. Using periodontal attachment loss as the measure estimated that people with type 2 diabetes were 2.8 times more likely to have destructive periodontal disease. When they used radiographic bone loss as the measure and controlled for other important covariates, the estimate rose to 3.4.

Diabetes type not specified
The final set of reports on the association between diabetes and periodontal diseases consists of seven cross-sectional studies in which the type of diabetes was not specified and was not easily determined from other information provided. Four of the seven studies included only adults. In the other three studies, subjects ranged in age from childhood to older adulthood. All seven studies found subjects with diabetes to have increased prevalence, extent, and severity of periodontal disease.

Diabetes is a risk factor for the occurrence and prevalence of periodontal diseases. Although there is insufficient evidence of a causal association, the findings of greater prevalence, severity, or extent of at least one manifestation of periodontal disease in individuals with diabetes is remarkably consistent in the overwhelming majority of studies. Furthermore, there are no studies with superior design features in the literature to refute this assessment. The studies were conducted in distinctly different settings, with subjects from different ethnic populations and of different ages, and with a variety of measures of periodontal status. This inevitable variation in methodology and study populations limits the possibility that the same biases apply in all the studies. There is a need for further research using stronger designs that also control for confounding variables such as socioeconomic status.

Glycemic control
Several lines of evidence support the plausibility that periodontal infections contribute to problems with glycemic control, thus compromising the health of diabetic patients. It has been reported that the chronic release of tumor necrosis factor alpha (TNF-alpha) and other cytokines such as those associated with periodontitis interferes with the action of insulin and leads to metabolic alterations. Other studies have noted relationships between insulin resistance and active inflammatory connective tissue diseases, other clinical diseases, acute infections and periodontal disease.

The body of literature concerning the relationship between periodontal infection and impaired glycemic control is varied in the strength, quantity, breadth and consistency of evidence presented. The preliminary evidence, while encouraging, does not support a clear-cut conclusion that treating periodontal infection can contribute to management of glycemic control in type 1 or type 2 diabetes. Only studies using systemic antibiotic treatment affected glycemic control favorably. The results suggest that infections other than periodontitis may be implicated or that intensive treatment of periodontal infections with systemic antibiotics is necessary to affect glycemic control favorably. Further rigorous controlled studies in diverse populations are warranted.

The oral infection, heart disease and stroke connection
During the past decade, infectious agents have become recognized as causes of systemic
diseases, without fever or other traditional signs of infection. Helicobacter pylori is associated with peptic ulcers and, along with Chlamydia pneumoniae and cytomegalovirus, is now thought to be associated with increased risk for cardiovascular disease as well as malignancies (Wu et al. 2000). Studies investigating the relationship between oral and dental infections and the risk for cardiovascular disease suggest that there is potential for oral microorganisms, such as periodontopathic bacteria, and their effects to be linked with heart disease.

Mechanisms of action
Infectious agents are thought to affect the risk of heart disease through several possible mechanisms. Bacteria or viruses originating in tissues such as the lungs or oral mucosa may directly infect blood vessel walls. Such infection may be largely asymptomatic, but may cause local vascular inflammation and injury, which would contribute to the development of lipid-rich plaques and atherosclerosis. Bacteria or viruses may also interact with white blood cells or platelets, both of which integrate into the developing atherosclerotic plaque. Cells of the blood vessel wall and white blood cells and platelets can release prostaglandins (especially PGE2), interleukins (especially IL-1), thromboxane B2 (TXB2) and tumor necrosis factor alpha (TNF-alpha). Bacterial products in the blood may also stimulate liver production of other pro-inflammatory or pro-coagulant molecules such as C-reactive protein and fibrinogen.

Microbes may also stimulate expression of tissue factor, which would activate coagulation. During the process of coagulation, platelets would become trapped in the growing clot or thrombus. Microthrombus formation is one of the key factors in the development of atherosclerotic plaques. As atherosclerotic plaques enlarge, the lumen of the coronary blood vessels narrows and the blood supply to the heart muscle becomes reduced. A frank heart attack or myocardial infarction results when a larger part of the coronary artery lumen becomes occluded. Failing to receive enough blood, the heart muscle dies, resulting in an infarct.

- The oral cavity is a portal of entry as well as the site of disease for microbial infections that affect general health status.
- The oral cavity and its functions can be adversely affected by many pharmaceuticals and other therapies commonly used in treating systemic conditions.
- The oral complications of these therapies can compromise patient compliance with treatment. Individuals such as immunocompromised and hospitalized patients are at greater risk for general morbidity due to oral infections.
- Individuals with diabetes are at greater risk for periodontal diseases.
- Animal and population-based studies have demonstrated an association between periodontal diseases and diabetes, cardiovascular disease, stroke and adverse pregnancy outcomes. Further research is needed to determine the extent to which these associations are causal or coincidental.

Prevention and control of dental caries
Although many caries prevention strategies, notably community water fluoridation and use of a fluoride-containing dentifrice, benefit adults and children alike, most of our understanding of the effectiveness of these strategies comes from the study of children, during a life stage when caries incidence is high. Caries prevention programs have been designed and evaluated for children and have used a variety of fluoride and dental sealant strategies applied separately and together. Because these strategies are complementary, their use in combination has the potential of virtually eliminating dental caries in all children.

However, dental caries is a problem for all ages. Although direct evidence of caries preventive strategies in adults is limited, the evidence that does exist is consistent with expected effects based on experience with children. The Centers for Disease Control and Prevention (CDC) recently convened an expert work group to develop recommendations for modalities to prevent and control dental caries based on a review of publications selected by the work group and other experts.

Fluoride
Fluoride reduces the incidence of dental caries and slows or reverses the progression of existing lesions (i.e., helps prevent cavities). Today, all Americans are exposed to fluoride to some degree, and there is little doubt that widespread use of fluoride has been a major factor in the overall decline in recent decades in the prevalence and severity of dental caries in the United States and other economically developed countries. Fluoride is the ionic form of the element fluorine, the 13th most abundant element in the crust of the Earth. Because of its high affinity for calcium, fluoride is mainly associated with calcified tissues (i.e., bones and teeth). The ability of fluoride to inhibit, and even reverse, the initiation and progression of dental caries is well known. Fluoride’s mechanisms of action include incorporation of fluoride into enamel pre-eruptively, inhibition of demineralization, enhancement of remineralization and inhibition of bacterial activity in dental plaque.

A variety of theories regarding fluoride’s mechanisms of action account for the range of fluoride products available. The initial theory of action was based on the belief that incorporation of fluoride into the hydroxyapatite of developing tooth enamel in the pre-eruptive phase reduced the mineral’s solubility, thereby increasing enamel resistance. Because of the length of time the tooth is at risk of caries during the post-eruptive phase, however, the topical effects of fluoride are considered to predominate. These effects are based on fluoride’s role in the aqueous phase around the tooth, both in saliva and in dental biofilm (plaque). Fluoride in plaque contributes to the remineralization of demineralized enamel when bound fluoride is released during an acid challenge, resulting in a more acid-resistant enamel surface structure. Fluoride also has been shown to inhibit the process of glycolysis by which fermentable carbohydrates are metabolized by cariogenic bacteria to produce acid. All these effects occur after the tooth erupts, while it is functioning in the mouth, enabling fluoride to prevent caries over a lifetime in both children and adults.

The first use of fluoride for caries prevention was in 1945 in the United States and Canada, when the fluoride concentration was adjusted in the drinking water supplying four communities. This public health approach followed a long period of epidemiologic studies of the effects of naturally occurring fluoride in drinking water.

The success of the community water fluoridation trials in reducing dental caries led to the development of other important fluoride-containing products, such as dietary supplements and, most notably, fluoride-containing dentifrices, in the early 1960s. Fluoride-containing gels, solutions, pastes and varnishes were also developed for topical use, either applied by professionals or self-applied at home or in other settings. All of these products were tested for safety and effectiveness in reducing caries. Products designed for professional use generally have higher concentrations and are used at less frequent intervals than those designed for self-application.

Controlled clinical trials from the 1940s through the 1970s documented the benefits of professionally applied fluoride in reducing dental caries, and several excellent reviews are available. Professional application of fluoride is inherently more expensive than self-applied methods, so the use of such an approach for groups and individuals at low risk of dental caries is unlikely to be cost-effective. For patients at high risk of dental caries, however, professionally applied fluoride is still considered cost-effective. It is not clear whether fluoride varnishes and gels would be most efficiently used in clinical programs targeting groups at high risk of dental caries or whether they should be reserved for individual high-risk patients.

The U.S. Preventive Services Task Force and the Canadian Task Force on Periodic Health Examination affirm that there is strong evidence to support the major methods for providing fluoride to prevent dental caries.

The safety of fluoride is well documented and has been reviewed comprehensively by several scientific and public health organizations (Institute of Medicine (IOM) 1997, National Research Council (NRC) 1993, Newbrun 1996, U.S. Department of Health and Human Services (USDHHS) 1991, World Health Organization (WHO) 1984). When used appropriately, fluoride has been demonstrated to be both safe and effective in preventing and controlling dental caries. The IOM (1997) classified fluoride as a micronutrient, citing it, along with calcium, phosphorus, magnesium and vitamin D, as an important constituent in maintaining health.
Appropriate use of fluoride products can minimize the potential for enamel fluorosis, a broad term applied to certain visually detectable changes in the opacity of tooth enamel associated with areas of fluoride-related developmental hypomineralization. There are also many developmental changes in enamel that are not fluoride-related. Most enamel fluorosis seen today is of the mildest form, which affects neither aesthetics nor dental function. Cosmetically objectionable enamel fluorosis can occur when young children ingest higher than optimal amounts of fluoride from any source while tooth enamel is forming (up to age 6). Its occurrence appears to be most strongly associated with the total cumulative fluoride intake during the period of enamel development, but the condition’s severity depends on the dose, duration and timing of fluoride intake. Specific recommendations have been made to control fluoride intake by children during the years of tooth development.

**Fluoridation of drinking water**

For more than half a century, community water fluoridation has been the cornerstone of caries prevention in the United States; indeed, CDC has recognized water fluoridation as one of the great public health achievements of the 20th century. All water contains at least trace amounts of fluoride. Water fluoridation is the controlled addition of a fluoride compound to a public water supply to achieve a concentration optimal for dental caries prevention. In the 1940s, it was concluded that 1 ppm (part per million) fluoride was the optimal concentration for climates similar to that of the Chicago area; this concentration would significantly reduce the prevalence of dental caries with an acceptably low prevalence of enamel fluorosis.

Current U.S. Public Health Service (USPHS) recommendations for fluoride use include an optimally adjusted concentration of fluoride in drinking water ranging from 0.7 to 1.2 ppm, depending on the mean maximum daily air temperature of the area. A lower fluoride concentration is recommended for communities in warmer climates than cooler climates because it is assumed that persons living in warmer climates drink more tap water.

**Effectiveness**

Numerous studies in naturally fluoridated areas preceded the field trials. There are no randomized, double-blind, controlled trials of water fluoridation because its community-wide nature does not permit randomization of people to study and control groups. Similar results have been derived from numerous well-conducted field studies by various investigators on thousands of subjects in different parts of the world. Conducting a study in which individuals are randomized to receive or not receive fluoridated water is unnecessary and is not feasible.

In 1945, Grand Rapids, Mich., became the first city in the United States to fluoridate its water supply; the oral health of its schoolchildren was periodically compared with that of schoolchildren in the control city, Muskegon, Mich. Dramatic declines in dental caries among children in Grand Rapids and three other cities conducting studies shortly thereafter led to fluoridation in many other cities. In an extensive review of 95 studies conducted between 1945 and 1978 reported the modal caries reduction following water fluoridation to be between 40 and 50 percent for primary teeth and 50 and 60 percent for permanent teeth. Fluoridation also benefits middle-aged and older adults. Benefits to adults include reductions in both coronal and root caries.

These benefits are important because older people typically experience gingival recession, which results in exposed root surfaces, which are susceptible to caries. In addition, tooth retention in older U.S. cohorts has increased in recent decades, so that the number of teeth at risk for caries in older age groups is also increasing. Finally, many medications used to treat chronic diseases common in aging have the side effect of diminished salivary flow, depriving teeth of the many protective factors in saliva. Other evidence of the benefits of fluoridation comes from studies of populations where fluoridation has ceased. Examples in the United States, Germany and Scotland have shown that when fluoridation is withdrawn and there are few other fluoride exposures, the prevalence of caries increases. In Wick, Scotland, which began water fluoridation in 1969 but stopped it in 1979, the caries prevalence in 5- to 6-year-olds with limited exposure to other sources of fluoride increased by 27 percent between 1979 and 1984. This was despite a national decline in caries and increased availability of fluoride-containing dentifrices.

**Fluoride mouth rinses**

Several different formulations of fluoride mouthrinses are available, differing in the amount of fluoride and suggested frequency of use. Rinses with low fluoride concentrations (0.05 percent neutral sodium fluoride or 0.1 percent stannous fluoride) are designed for daily use and are available over-the-counter. Higher-concentration rinses (0.2 percent sodium fluoride) are designed for weekly use and are available only by prescription or in public programs.

**Dental sealants**

The pits and fissures that characterize the biting surfaces of posterior teeth provide a haven for food debris and decay-causing bacteria. Not surprisingly, these sites are often the first and most frequent to be affected by decay in children and adolescents. The width of most pits and fissures is narrower than a single toothbrush bristle, making cleaning of their deepest recesses almost impossible. According to national estimates, as much as 90 percent of all dental caries in schoolchildren occurs in pits and fissures. The teeth at highest risk by far are permanent first and second molars.

Enamel bonding, a technology introduced in the mid-1950s, led to the development of sealants. These are clear or opaque plastic resinous materials designed for professional application to the pit-and-fissure surfaces of teeth. The material hardens within 60 seconds or so into a thin, hard, protective coating. Sealants were introduced in the late 1960s and received the American Dental Association Seal of Approval in 1976. Most of the dozen products approved by the ADA do not contain a therapeutic agent, but work by providing a physical barrier that prevents microorganisms and food particles from collecting in the pits and fissures. First-generation sealants used ultraviolet light to harden or “cure” the material; improved second- and third-generation sealants cure by chemical or visible light activation, respectively.

Sealant placement requires meticulous attention to technique, but they can be successfully provided in “field” settings using portable dental equipment. To be most effective, sealants should be placed on teeth soon after they erupt, but they can be applied across a wide age range. Not only does the risk for caries continue across the life span, but an individual’s risk also can increase for any number of reasons. Sealants are particularly helpful for persons with medical conditions associated with higher caries rates, children who have experienced extensive caries in their primary teeth, and children who already have incipient caries in a permanent molar tooth.

**Efficacy**

Initial clinical trials using a random half-mouth design and first- or second-generation sealant materials established their efficacy. Several comprehensive reviews and a meta-analysis of the amount of caries prevented testify to the utility of these materials a systematic process to select and review studies of one-time sealant placement on permanent teeth in subjects unexposed to other preventive measures. Pooled results from 17 studies meeting their selection criteria found that second-generation sealants reduced caries over 70 percent.

These early trials firmly established retention as essential to preventing caries; a sealant is virtually 100 percent effective if it is fully retained on the tooth.

**Effectiveness**

Administrators of school-linked sealant programs and of school-based programs with either fixed clinics or portable equipment reported on their experiences with these programs. These studies, using second-generation sealants, have shown effectiveness results comparable to those of clinical trials, regardless of the physical delivery site or personnel used for sealant application. Complete retention after approximately one year varied from 83 to 94 percent.

A Consensus Development Conference sponsored by the National Institutes of Health concluded that “an extensive body of knowledge has firmly established the scientific basis for the use of sealants.” The panel urged the development of educational materials to enhance public and professional acceptance as well as third-party reimbursement. Consensus on the value of sealants is reflected by the inclusion of sealant...
objectives in Healthy People 2000 and Healthy People 2010. In addition, sealant placement is supported in federally funded programs for women and children, and sealants are covered services in all state Medicaid programs. A workshop on guidelines for sealant use has made recommendations for sealant use in both community and individual care programs.

Community dental sealant programs
Several community-based public health initiatives have arisen to promote sealant use among private practitioners and through community-based programs. These activities include reaching dentists through continuing education courses; directing large-scale promotional activities to consumers, community leaders and third-party payers; and providing sealants directly to children in school programs.

Prevention and control of periodontal diseases
Periodontal diseases, caused by specific bacteria in dental plaque, affect most adults at some point in their lives. The mildest and most common form of periodontal disease is gingivitis. Over time, periodontitis, the more severe form of periodontal disease, can lead to the destruction of the soft tissue and bone that anchor the teeth into the jaw. Lacking support, teeth can loosen and be lost.

Periodontal diseases can be prevented and controlled through an array of mechanical and chemical means. Conscientious oral hygiene and professional oral cleanings to reduce plaque can reverse gingivitis. Methods for personal oral hygiene include toothbrushing and flossing, which may be augmented by over-the-counter and prescription mouthrinses with antimicrobial action.

Community programs to prevent gingivitis
With the confirmation of specific bacteria in dental plaque as the cause of gingivitis, public health officials began to seek ways to educate the public about plaque control in community settings, primarily in schools. These efforts have had equivocal results. Although knowledge and attitudes were enhanced in demonstration programs, improvements in plaque levels and gingivitis were short-lived in clinical trials.

Prevention of periodontitis
Toothbrush use is a major risk factor for the development and progression of periodontal diseases, and proven strategies aimed at reducing tobacco use should aid in the prevention of periodontitis. The following section on oral and pharyngeal cancers includes a discussion of such intervention strategies. Until recently, most interest in controlling tobacco use reflected concerns about oral cancers. As appreciation of the role of tobacco in the progression of periodontal diseases and tooth loss increases, attention to these oral health effects may increase attention to tobacco cessation in primary oral health care. Periodontitis can also be a complication of poorly controlled diabetes.

Some efforts have been directed at alerting dental practitioners to the need to educate patients about diseases affecting the periodontal tissues. These efforts have met with some success, but they tend to reach only those people who already use dental services. Currently, there are no broad community-based intervention programs that address periodontal diseases.

Gingivitis can be controlled with available methods, and its control is the principal way to prevent periodontitis. However, the currently available methods are individually or professionally based and require conscientious oral hygiene practices and regular dental visits. Although some schools instruct children in proper methods of oral hygiene, no community methods, other than programs designed to discourage tobacco use, are available for preventing gingivitis or periodontitis in the general population.

Prevention and control of craniofacial birth defects
The causes of craniofacial birth defects are often complex and multifactorial, the result of gene-environment interactions occurring from the time of conception to birth. Even when a mutation in a single gene has been discovered as the cause of a particular syndrome, there can be considerable variation in susceptibility, with some infants showing little or no sign of a problem and others experiencing multiple organ defects.

The work to complete the mapping and sequencing of the human genome will undoubtedly shed light on the hundreds of genes involved in craniofacial development and provide details on when and how they function in development. This knowledge may in turn lead to gene therapies that restore or “rescue” the function of a defective gene and thus prevent the anomaly.

Craniofacial defects also may occur because the susceptible embryo or fetus was exposed to an environmental teratogen, a diminished oxygen supply or a deficit in an essential nutrient. An association may exist between low-birth-weight, premature babies who may show other subtle craniofacial anomalies and mothers with chronic oral infectious disease.

In addition, diets poor in folic acid increase the risk of spina bifida and possibly clefting syndromes. Clinical trials using vitamin supplementation with varying levels of folic acid are under development to determine whether they can lower the risk of clefts in high-risk pregnancies. Outcomes of clinical trials of nutrient supplementation in pregnancy may lead to new nutritional guidelines and the development of enriched food products, which can form the basis for community-wide health promotion and disease prevention programs.

Given the array of variables affecting prenatal growth and development, the key to public health programs aimed at preventing birth defects lies primarily in health promotion and education campaigns. Individuals need to be made aware of known risk and protective factors in pregnancy. Such programs should emphasize the importance of good nutrition, avoidance of tobacco and alcohol use, and prenatal care. Education includes knowledge about the teratogenic effects of prescription drugs, such as the antiepileptic drug phenytoin and the retinoic acid drugs used to treat cystic acne.

As information from developmental biology, genetics and epidemiologic and clinical studies accrues, dental care providers are better positioned to provide counseling. The public is best served by health promotion and disease prevention campaigns that communicate findings about risk and protective factors in pregnancy.

Prevention and control of intentional and unintentional injury
Intentional and unintentional injuries are related to behaviors and are thus amenable to prevention. As studies of motor vehicle and sports injuries have demonstrated, injuries are frequently due to a sequence of predictable events, and a public health approach can be successful in injury prevention and control.

The interventions that have proved to be most effective in controlling injuries have been passive; that is, they do not require the individual to participate. Examples include the use of environmental controls such as vehicle and roadway design, speed limits, passenger restraints and airbags to prevent injuries from motor vehicle collisions. Passive measures such as these are more easily implemented at the state or federal level. However, many preventive measures for oral-facial injuries have been directed at the individual and professional health service levels, rather than at the population at large.

Craniofacial injuries
The principal causes of craniofacial injuries are motor vehicle collisions, falls, assaults and sporting activities. Except in relation to sports, injuries to the craniofacial region have received little attention. These injuries are hardly insignificant, however, and efforts to prevent them are gaining acceptance. For example, to increase public awareness of the importance of facial protection, the inaugural National Facial Protection Month was celebrated in April 2000. This national campaign, providing information to the media and the public, was sponsored by the American Association of Oral and Maxillofacial Surgeons (AAOMS 2000).

Motor vehicle collisions are the leading cause of death during the first three decades of life in the United States and the leading cause of death from injury over most of the life span. Data from multiple sources indicate that craniofacial injuries account for a substantial subset of these injuries annually. Even though it is likely that passive measures enacted to reduce fatalities have reduced nonfatal craniofacial injuries, no supporting data exist.

Various sources report the number of motorcycle- and pedal-cycle-related craniofacial injuries. Data from the National Electronic Injury Surveillance System indicate that head injuries account for 50 percent of all pedal-cycle-related injuries; of those, bicycle-related events accounted
for 19 percent of all facial injuries within the study period. In similar studies, tricycle-related incidents were found to be responsible for up to 61 percent of injuries to the head, face or mouth. Motorcycle injuries are a major source of fatal and nonfatal head trauma in the United States.

Helmet use reduces head and facial injuries among bicyclists and motorcyclists by up to 50 percent. Health promotion efforts have increased acceptance at the community level for helmet use by bicyclists; however, helmet use regulations vary by state and with the public whim. Over a dozen states currently have bicycle helmet laws, and half of the states have motorcycle helmet laws.

Many authors have described craniofacial injuries related to sports. Information is usually obtained from community or regional surveys of injuries or mouthguard use and effectiveness. Craniofacial injuries sustained during sporting activities are a major source of nonfatal injury and disability, possibly accounting for up to one third of all sports injuries. The increasing participation of women in competitive sports means that young women should be alerted to the risks and advised of the need for additional protective gear as appropriate.

Health professionals are in an ideal position to provide up-to-date health information and care to their patients. They also have an opportunity to enhance their knowledge and practices as well as increase their communication to patients about the procedures they provide and the reasons for these procedures.

Daily hygiene and dental caries prevention
The use of a fluoride-containing dentifrice is critical for dental caries prevention. Even more beneficial than the physical removal of plaque in toothbrushing is the delivery of a small amount of fluoride to the tooth surfaces. Investigators have conducted numerous clinical trials on fluoride dentifrices using rigorous designs and including randomized groups, double-blind designs and placebo controls. All together, these studies provide strong evidence that using a fluoride dentifrice is effective. Fluoride dentifrices account for more than 90 percent of the market in the United States, Canada and other developed countries.

A fluoride dentifrice is an effective means of reducing the prevalence of dental caries for all persons. Although children’s teeth should be cleaned daily from the time they erupt, parents and caregivers should consult a dentist or other health care provider about the use of a fluoride dentifrice for children under the age of 2. For children under 6, fluoride dentifrices should be used in small amounts to minimize swallowing of the product. Mild enamel fluorosis can result from excessive dentifrice use, because children under 6 do not have adequate control of the swallowing reflex or may intentionally swallow a flavored dentifrice. Experts recommend that for children under 6, the parent or caregiver should supervise toothbrushing, apply a pea-sized amount (0.25 gram) of dentifrice to the toothbrush, and encourage the child to spit out the excess.

Because the topical benefits of fluoride have been shown to be highly effective and daily exposure to small amounts of fluoride can reduce the risk of dental caries in all age groups, experts recommend that all persons drink water with an optimal fluoride concentration in addition to brushing daily with a fluoride dentifrice. This combination provides a cost-effective and easy way to prevent dental caries and is an excellent example of the individual-community partnership. For persons at low risk of dental caries, these two exposures to fluoride may be the only ones necessary. For persons at moderate or high risk of dental caries, additional fluoride may be helpful and can come from daily use of another fluoride product. These can include mouth rinses, prescribed supplements and professionally applied topical fluoride products.

Dental caries
Dental caries is caused by a transmissible microbial infection that affects tooth mineral. A number of factors play a role in the initiation and progression of the disease, including bacterial biofilm, specifically the presence of...
mutans streptococci and species of lactobacilli; the frequency of simple sugars in the diet; the flow and composition of saliva; the availability of fluoride; the structure of tooth mineral in a given individual; and oral hygiene behaviors. Sound caries management takes all these factors into account. Today there is the prospect that clinicians will be able to balance protective and pathologic factors and work with the patient to control disease.

Risk assessment
Reviews of caries risk prediction models conclude that clinical variables, especially past caries experience, are the best predictors of new caries experience. At the time of initial tooth eruption, the presence of mutans streptococci appears to be the primary predictor of future caries. With continued tooth eruption, this variable disappears as a primary predictor and is replaced by the status of the most recently exposed or erupted tooth surface. For example, the presence of carious lesions in the primary incisors has been found to be the best predictor of caries in the later-erupting primary molars.

Despite recent declines, dental caries is a prevalent disease, with some age and population groups particularly vulnerable. A guide for the identification of vulnerable patients and the treatment of caries as an infectious disease developed by the American Dental Association proposes questions to be considered at an initial examination. These questions, together with information gathered at recall examinations, allow classification of child and adult patients into high-, moderate- and low-risk disease categories. This approach has been incorporated in a variety of caries risk assessment forms adopted by some dental schools and managed care programs. Studies are needed to determine the validity and reliability of such approaches for different patient populations and practice settings.

The use of tests to assess caries risk to determine the activity status of preclinical disease is becoming more widespread. A range of diagnostic aids for caries activity testing are available. Microbial tests can detect the presence and quantify the levels of lactobacilli and mutans streptococci. The development and use of these tests are based on studies that have associated these microbes individually and together with different types of carious lesion development. Measurements of plaque and salivary pH have been used to evaluate the oral environment overall and to note the changes in pH that occur after eating various foods. Salivary flow and composition analyses add another dimension. Decreased flow has been related to caries susceptibility, as have increases in viscosity. These factors warrant further study to determine their sensitivity and specificity.

Diagnosis
Clinical signs, patient-derived history and radiographic images remain the primary means of dental caries diagnosis. Tooth surface pitting and caviation, white and/or brown spots, areas soft to tactile probing and radiolucencies are used to detect the effects of this disease. The most common diagnostic approaches include visual inspection, the use of an explorer (a probelike instrument) to determine the integrity of the tooth surface, the use of a light source to detect difference in reflectance across tooth structure (transillumination) and radiographs.

The most basic diagnostic methods – visual alone and visual examination with an explorer – have limited sensitivity but excellent specificity. The visual examination may be combined with a radiographic series for the initial assessment. Bite-wing radiographs are frequently used to diagnose interproximal caries (between teeth) and for these surfaces provide excellent sensitivity and specificity. Radiographic examination allows examination of otherwise inaccessible areas. Specifically, the depth of a lesion and its relationship to the pulp chamber can be evaluated for interproximal lesions. However, radiographs are of little value in detecting caries on the occlusal surfaces of the teeth. For these surfaces, a negative radiographic diagnosis does not imply lack of a carious lesion in enamel.

Precavitated carious lesions and caries in restored teeth pose an additional diagnostic challenge. A review of the literature on the clinical diagnosis of precavitated carious lesions concluded that visual detection of these lesions has low sensitivity and moderate specificity. It is difficult with these lesions to determine whether there are no caries or whether only the enamel or outer layer of dentin is involved. Carious lesions forming around restorations are seen more frequently at the approximal and cervical margins of these restorations. Distinctive color changes around a restoration alone are not diagnostic of active caries. Currently, the progression of carious lesions is the most definitive diagnostic parameter for disease activity. Progression can be determined over specific time intervals only by professional assessment.

Prevention
The primary prevention of dental caries starts with adequate prenatal and perinatal nutrition to ensure normal development of the teeth and supporting structures. It continues with interventions aimed at preventing transmission of cariogenic microbes from caregivers to infants, and proceeds with specific strategies employed across the life span. These approaches include the provision of sufficient fluoride, the use of dental sealants, the adoption of healthy behaviors, including avoiding unhealthy dietary practices and practicing appropriate oral hygiene, and the timely use of care services. Although many factors are brought to bear on the primary prevention of dental caries, the combination of fluoride in its multiple forms and dental sealants is the foundation.

Fluoride is available in a variety of products that can be used by health professionals, individuals and public programs. Topical solutions and gels, mouth rinses and dentifrices are available for daily, weekly or as-prescribed frequency. In addition, fluoride-containing prophylactic pastes are available for professional application. Clinical judgment of risk factors determines the type and frequency of interventions needed. Although there is general agreement on the overall value of topical fluorides in reducing dental caries, comparative clinical trials are needed to determine which of the existing fluoride formulations (acidulated phosphate fluoride, stannous fluoride, amino-fluoride or sodium fluoride) and which delivery system (gel, varnish, dentifrice or solution) are most efficacious.

A second line of defense is through control of the etiologic agent. Chemotherapeutic agents (including the antimicrobial mouthrinse agent chlorhexidine and fluoride) can be used to reduce plaque. Dietary measures aimed at reducing the frequency and quantity of sugars and the substitution of sugars by sugar-free sweeteners may effectively starve the bacteria.

The process of tooth demineralization and remineralization has received significant attention over the past four decades. Investigators are studying the effectiveness of therapeutic agents for arresting carious lesions and remineralizing enamel in populations at high risk for dental caries. For example, a combined chlorhexidine-fluoride solution can enhance remineralization of incipient lesions and arrest caries in patients who suffer from radiation-induced caries. The use of a twice-daily rinse with 0.05 percent sodium fluoride to prevent demineralization and induce remineralization in subjects with radiation-induced hyposalivation has also been found to be effective.

This study also addressed the effects of chlorhexidine use alone, which has been associated with tooth staining, alterations in taste and potential hypersensitivity reactions. It showed that the application of 40 percent by weight chlorhexidine varnish every three months enhanced remineralization of root caries more than fluoride varnish, although both treatments were associated with fewer filled root surfaces than the control group after one year. A chlorhexidine varnish has not yet been approved in the United States, and large-scale, double-blind, placebo-controlled clinical trials are not yet available to test the effects of specific regimens in relation to caries risk.

Studies also are evaluating interventions to prevent mutans streptococci transmission. Findings from cross-sectional studies indicate that infants are initially infected by their parents, specifically mothers, around the time the teeth erupt. A longitudinal study using DNA fingerprinting demonstrated that mothers were the source of the bacteria in their infants and the degree of matching to maternal strains was higher for female infants than for males.

Based on a study of child-mother pairs (with the child initially at 1 year of age), the application of a 1.0 percent chlorhexidine rinse alternated with a 0.2 percent sodium fluoride gel to the mother’s teeth (three times per day on two consecutive days, twice per year for three years) delayed, and in some cases prevented, the colonization of their
children’s teeth by mutans streptococci. Timing of colonization has been shown to be correlated with caries prevalence. In a longitudinal study that followed children in four-month intervals from 15 months to 4 years of age, children who were infected earlier had a higher caries prevalence than those in whom the infection was detected at later ages. Studies also have been aimed at reducing the levels of cariogenic bacteria in the infants themselves.

Work continues on the development of a caries vaccine. One approach focuses on the production and release of antibodies against cariogenic bacteria antigens. Specific antigens have been purified and synthesized. Another approach involves biological replacement therapy, where nonpathogenic bacteria instilled in the mouth prevent pathogenic bacteria from colonizing. Yet another approach employs passive immunization in which antibodies, produced outside the body (in cultures, animals, eggs, or plants), are applied to the teeth and oral tissues to protect against disease. A recent study indicated that “plantbodies” painted on the teeth could prevent mutans streptococci colonization for 120 days, the period of the experiment.

**Treatment**

Prompt treatment of early carious lesions permits the preservation of tooth structure through conservative approaches. A 10-year study reported that caries did not progress under a dental sealant placed over cavitated lesions where the lesions were no more than halfway through the dentin.

Materials that can bond to enamel and to dentin continue to be refined and improved. Glass ionomer cements have contributed to materials that can bond to enamel and dentin, release fluoride and increase remineralization in adjacent teeth. These cements, together with polymeric resin composites and hybrids of these two materials, are now available for tooth restoration with other materials. Based on the available materials and emerging techniques, such as air abrasion and laser ablation, restoration procedures are more conservative than ever before.

A proposed categorization of carious lesions for the purpose of conservative management places lesions into three categories: lesions where no treatment is advised, lesions where preventive care is advised and lesions where restorative treatment is advised. This approach, using caries as an infectious disease paradigm, resulted in a marked reduction of operative procedures in Danish schoolchildren and has been proposed as a means to preserve tooth structure and maximize appropriate care in the United States.

New imaging and laser technologies are emerging as tools for early diagnosis and prompt treatment of dental caries. For example, quantitative light-induced fluorescence is showing promise for dental caries diagnosis. Two different methods, the quantitative infrared laser fluorescence method and electrical conductance measurements, are currently commercially available. At present, these methods are being used to augment conventional diagnostic tools but are not yet part of routine practice. However, they could potentially be used for close monitoring of the lesions and for patient motivation. Laser treatments for soft tissue surgery have been used in dentistry in recent years. Currently, in vitro studies are under way for the application of lasers for hard tissues, specifically to prevent dental caries by altering tooth mineral and inhibiting progression of artificial caries-like lesions.

Despite the best efforts of the individual and health care provider, caries may progress. Advances in materials science over the last two decades have fortunately led to major improvements in dental restorative materials, resulting in a wide range of aesthetically pleasing, longer-lasting restorations that can be placed with less trauma. Traditional materials such as amalgam fillings and gold crowns are now augmented by aesthetic materials, including bonded composite resins, porcelain fused to metal crowns and facings.

When teeth have been lost, the options for rehabilitation include a range of prosthetic devices. Removable full and partial dentures and fixed bridges provide aesthetic and serviceable restorations for many patients. Still another option is the use of dental implants. These are used not only in patients who have lost teeth due to caries and periodontal diseases, but also to restore form and function in patients treated for trauma, craniofacial cancers, hereditary tooth defects and other abnormalities.

The evidence base for the survival of the endosseous dental implants, an implant that is placed directly into a tooth socket, is extensive and has been recently reviewed. The predictability of endosseous dental implants in fully and partially edentulous patients has been clearly demonstrated in longitudinal studies. Many implant designs and surfaces have shown high success rates (often exceeding 95 percent in good-quality bone and 85 percent in poorer-quality bone, such as the posterior maxilla).

Rehabilitation of lost tooth structure or even the whole tooth itself may be revolutionized in the next century, based on discoveries of the natural repair and regeneration mechanisms the body uses. The new sciences of biomimetics and tissue engineering combine engineering principles and materials science with rapidly growing knowledge of the progenitor cells and molecules that give rise to specific tissues such as skin, bone, teeth and cartilage. Already it is possible to generate new cartilage and bone of a prescribed shape to replace tissue lost from injury or disease. Eventually, it may be possible to use a patient’s own oral cells and cell products to generate new tooth enamel, dentin and cementum for the natural repair of carious lesions.

**Periodontal diseases**

Periodontal diseases are caused by microbial infections, and are plaque-related complex diseases like dental caries, presenting as several clinical variants. The mildest form is gingivitis, characterized by inflammation of the gingiva with a marked loss of gingival collagenous material. In a more advanced disease, periodontitis, there is involvement of the soft tissue and bone that support the teeth. If untreated, periodontitis may progress and result in abscesses, mobile teeth and tooth loss. Periodontitis also may be associated with certain systemic diseases and conditions.

Gram-negative anaerobic bacteria in plaque are implicated as causative agents in periodontitis. However, host immune system factors, specifically, a chronic inflammatory response, are now considered to be the primary determinants of disease progression and outcome. The disease process is very similar across the different types of periodontal disease and involves interactions between infectious agents and their virulence factors and host defense mechanisms, operating within a context of environmental, acquired and genetic risk factors specific to a given individual. Sufficient knowledge of demographic and systemic risk factors and indicators has been acquired to guide clinical decisions in the management of periodontal diseases. The presence of pathogenic bacteria, poor oral hygiene, tobacco smoking, diabetes mellitus and pre-existing periodontal disease are some of the factors that contribute to the likelihood of disease presence, progression and treatment outcomes.

A systematic identification of risk factors, indicators and predictors has been proposed as the first step in diagnosing and managing periodontal diseases. Clinicians can weigh the known risks for individual patients and devise treatment plans appropriate to their risk category. These same factors and the outcomes of treatment can also be used to assess progression upon completion of therapy. Studies are under way to determine the feasibility and validity of assessing a complex of risk factors to predict states of periodontal health and disease.

Most recently, putative genetic markers for susceptibility for oral disease have been studied. In particular, a specific genotype of the polymorphic IL-1 gene cluster has been shown to be associated with severe periodontitis in nonsmokers. IL-1-beta is of interest because the proinflammatory cytokines are key regulators of the host immune response to microbial infection and extracellular matrix catabolism and bone resorption. Functionally, this polymorphism is associated with high levels of IL-1 production, and high levels of IL-1 have been associated with progressive periodontal breakdown.

A consensus has been reached by a specialty organization that all patients in general and specialty care should be screened for periodontal disease. The recommended approach is to apply the periodontal screening and recording examination (PSR). Related screening tests include the community periodontal index of treatment needs (CPITN) and the basic periodontal examination.

**Diagnosis**

Most diagnostic tests for periodontal diseases rely on a physical examination to note any swelling, redness, gingival bleeding or tooth mobility. Periodontal probing, radiographs and microbiologic and histological examinations of biopsied tissue provide important additional information. These
tests indicate the presence, extent and severity of gingival and periodontal tissue destruction; they do not indicate the cause of disease or whether it is quiescent or actively progressing.

Gingival inflammation may be assessed using a variety of methods, including bleeding on probing and the use of indices such as the gingival index to grade redness and bleeding. In adult periodontitis, the absence of inflammation is associated with a lack of disease progression, but the presence of inflammation does not indicate inevitable progression to destruction. Longitudinal studies have also been conducted in patients who participate in maintenance programs.

The absence of gingival bleeding, especially at recall visits, has been shown to be a valid indicator of gingival health in these patients. Measurement of probing depths (also termed pocket depths) is an integral part of the periodontal examination. Longitudinal studies have shown that shallow probing depths and minimal loss of attachment are associated with lack of disease progression. The mere presence of a pocket does not herald progressive periodontitis at that site.

Although teeth with moderate to deep probing depths are at higher risk for additional destruction, a single examination cannot determine the fate of the tooth with certainty. Radiographs are used to obtain a visual image of the bony support around a tooth or dental implant. They are an essential tool in planning complex prosthetic reconstructions, as well as a necessary diagnostic aid in assessing periodontal progression.

**Prevention**

Because periodontal diseases are plaque-associated infections, prevention and management of the early signs of these diseases depend on effective plaque control. This can be accomplished using both mechanical and chemotherapeutic approaches. The prophylaxis performed in the dental office on periodontally healthy patients reduces plaque and removes stains and calculus. How often patients should be recalled for such preventive procedures is based on an assessment of risk factors, such as the patient’s age, oral hygiene, personal habits (e.g., smoking and diet) and a medical history indicating a heightened risk of infection (such as noted with diabetes or HIV infection).

Chemical plaque control has become an important part of the clinician’s armamentarium and may be prescribed for patient care at home. Significant reductions in gingival inflammation have been demonstrated for chlorhexidine, triclosan co-polymer when used in conjunction with a fixed combination of essential oils and stannous fluoride. The magnitude of gingival inflammation reduction was greatest for chlorhexidine. The evidence supporting these effects includes multiple randomized, double-blind controlled clinical trials.

**Treatment**

Once periodontal disease is established, the resultant bone and connective tissue loss may be quiescent or actively progressing. The goal of treatment is to determine whether the disease is active in order to prevent further tissue loss. This entails professional plaque removal and careful instruction of the patient on scrupulous self-care.

The concept of management of a patient’s risk factors as part of treatment is reasonably well documented for individuals who smoke and those who are diabetic and may be important for other risk factors such as stress and low dietary calcium. Several studies have shown that treatment of periodontal disease in smokers is not as successful as in nonsmokers. Thus, the management of smoking as a risk factor will contribute to the success of periodontal therapy. Furthermore, it appears that treatment of diabetic patients with periodontal disease may require more intense therapy since several studies have shown that antibiotic therapy is successful not only in reducing periodontal disease, but also in reducing glycerated hemoglobin.

Professional plaque removal typically employs scaling and root planing, in which hardened deposits of plaque and other debris are removed from the periodontal pocket and the tooth root surface is smoothed over. The effectiveness of scaling and root planing has been demonstrated repeatedly in longitudinal, cohort and randomized clinical trials and was reviewed by Cobb. Demonstrated benefits include decreased gingival inflammation, decreased probing depth and facilitation of maintenance of clinical attachment level. The evidence indicates that similar results may be obtained with ultrasonic and sonic instruments as with manual instruments. Regardless of the methods used, meticulous attention to detail is required to achieve optimal results.

Topical administration of antimicrobial agents contributes to the control of gingival inflammation. Supragingival irrigation (e.g., applying a jet of water under pressure) may be used as an adjunct to toothbrushing and has been shown to aid in the reduction of gingival inflammation. However, no clear substantial long-term benefits for the treatment of periodontitis have been shown if irrigation is applied subgingivally. Surgical therapy is employed to provide access to root surfaces and bony defects for debridement and root planing. Surgery can facilitate regeneration, augment the gingiva and promote root coverage. It is also necessary in placing dental implants.

Palcanis reviewed the evidence regarding surgical therapy. The overall goal is to make plaque control easier for the patient, thereby reducing disease progression. Many surgical techniques are available. Extensive randomized clinical trials and longitudinal studies form the basis of the evidence for the efficacy of these procedures. All procedures decrease pocket depth, and, with the exception of gingivectomy, all increase clinical attachment level. A caveat to be noted, however, is that procedures designed to reduce probing depth may increase gum recession, exposing the root and possibly compromising aesthetics. Thus, selection of a particular surgical procedure must always be based on the individual needs of the patient. Regardless of the approach selected, maintenance is important to long-term success.

Systemic administration of antibiotics, including the tetracyclines, 3 metronidazole, spironomycin and clindamycin, has been extensively studied and reviewed. The risk of generating antibiotic resistance in bacteria precludes the use of systemic agents in treating simple gingivitis. Similarly, systemic antibiotics should not be used for the routine first-line treatment of common forms of adult periodontitis. The preponderance of evidence from well-controlled, randomized, blinded clinical trials indicates that the agents do not offer sufficient benefit to overcome risks of either drug sensitivity or the emergence of antibiotic-resistant pathogens.

The situation is different in cases of aggressive forms of periodontitis, such as early-onset, rapidly progressive or refractory periodontitis, which affect less than 10 percent of periodontitis patients. Randomized, double-blind clinical trials as well as longitudinal assessments indicate that the use of systemic antibiotics can slow disease progression in these patients.

To circumvent the problems of systemic therapy, investigators have applied antimicrobial agents directly into the pocket. Antimicrobials incorporated into either resorbable and nonresorbable interpoocket delivery systems have been studied in randomized, double-blind, controlled clinical trials and are now FDA-approved and on the market. When used as an adjunct to scaling and root planing, gains in clinical attachment level and decreases in probing depth and gingival bleeding were demonstrated. Because these delivery systems are relatively new, there is a paucity of evidence addressing their long-term effectiveness.

For patients who have lost significant bone and/ or connective tissue, there are a number of regeneration procedures to facilitate the growth of new periodontal ligament, cementum and alveolar bone over previously diseased root surfaces. The evidence base for bone-grafting techniques using either natural or synthetic bone materials has been reviewed by Garrett (1996). Natural bone grafts may use autografts, in which bone is transferred from one site to another in the same patient; allografts, which use bone grafts from a human donor; and xenografts, which use tissues from other species. Limited case-report evidence shows that extraoral autogenous bone, such as hip grafts, has high potential for bone growth. Extraoral sites require a second surgical site, and in some cases fresh grafts may be associated with root resorption. Case report evidence indicates bone fill exceeding 50 percent of the osseous defect may be achieved. Controlled studies comparing grafted to nongrafted sites report significant improvements in clinical attachment levels and bone gain, but the magnitude of gain is less than that indicated in case reports.

Freeze-dried demineralized bone represents one of the most frequently used and well- studied bone graft materials in periodontics. Freeze-dried demineralized bone is an allograft
material, harvested, prepared and demineralized prior to grafting. The demineralization step is important because it retains the activity of bone morphogenetic proteins, compounds in the graft material found to be essential for new bone formation. Case reports and controlled clinical trials have demonstrated the bone-forming potential of such material, with some variability in the amount of bone fill achieved. Because allografts are derived from donor tissues, proper collection, handling and storage are essential to ensure viability and prevent contamination with viruses or other pathogens. Alloplasts represent a class of synthetic resorbable or nonresorbable graft materials. When evaluated in controlled clinical trials, they demonstrated improvements in probing depth and attachment level. Histology, however, indicates that, in general, synthetic grafts act primarily as space fillers, with little, if any, regeneration. Beginning in the 1980s, a number of investigators explored a procedure called guided tissue regeneration. The idea was to employ either a resorbable or nonresorbable membrane at the diseased site that would selectively allow passage of cells able to regenerate periodontal attachment apparatus and bone while prohibiting migration of nonregenerative cells such as fibroblasts. The evidence for the efficacy of guided tissue regeneration ranged from randomized controlled clinical trials to case reports. Although less evidence is available for resorbable membranes than for nonresorbable membranes, significant improvements in clinical attachment levels have been shown compared to debridement alone. Most favorable results are reported for bone loss between the roots of mandibular tooth defects (Class II furcation). Less favorable results were reported in maxillary molar and Class III (through and through) furcation defects (Garrett 1996).

**Birth defects**

There are hundreds of genetic diseases and syndromes as well as congenital anomalies that affect the craniofacial, oral and dental tissues. However, some craniofacial anomalies may be spontaneous and manifest only at the time of birth. Rapidly advancing knowledge of the genetics of development and of mutations associated with specific birth defects is aiding in the development of screening tests for genetic disorders and identifying high-risk individuals and families. A complete diagnosis of the craniofacial disorder may involve a multidisciplinary team of experts in imaging, genetics and other areas. Similarly, long-term management of the disorder, often extending to adulthood, generally calls for a team of specialists, including physicians and dentists, surgeons, nurses, rehabilitation experts, speech pathologists, psychologists and social workers. Quality of life considerations, including social and psychological effects of birth defects such as cleft lip and palate, are taken into account.

**Prevention**

Primary prevention involves minimizing exposure to known teratogens, and genetic counseling as appropriate. The importance of educating parents or potential parents on behavioral risk factors, especially tobacco and alcohol use, the teratogenic potential of certain prescription drugs and the need for adequate nutrition in the perinatal period is emphasized. A study performed in 1995, supplementation of the diet by multivitamins and folic acid during the periconceptional period (i.e., before, during and after conception) markedly diminished the occurrence of cleft lip and palate in a high-risk group. Unfortunately, only about 29 percent of women of childbearing age consume recommended amounts of these essential nutrients.

The evidence associating moderate to severe periodontal disease in pregnant women with low-weight preterm births warrants attention to the importance of maintaining optimal oral health in pregnancy. The oral care clinician can contribute to birth defect prevention not only by treating oral disease, but also by providing educational messages to patients to promote the birth of healthy, full-term babies.

**Treatment**

A number of birth defects may not be apparent at birth because they are not manifested until later in development. One example is the ectodermal dysplasias (ED), disorders characterized by abnormalities of skin, hair, sweat glands and teeth. Dentists are essential in the management of care for children with these disorders, who must be repeatedly fitted with dentures throughout childhood. More recently, clinical studies have demonstrated that fitting ectodermal dysplasia patients as young as 12 years old with dental implants not only is effective, but also provides greater functional utility and satisfaction. As with other complex craniofacial anomalies, management by a multidisciplinary team is the best approach, with experts able to advise on the various oral, skin and sweat gland complications. Mutations have recently been identified for several forms of ED, including the anhidrotic form (absence of sweat glands). Ultimately, the development of genetic diagnostic tests can confirm the diagnosis in the child and permit counseling of parents.

**Chronic craniofacial pain and sensorimotor conditions**

A variety of problems involving pain and other sensorimotor abnormalities affect the craniofacial complex. These conditions can include burning mouth syndrome, trigeminal neuralgia, various facial palsy, postherpetic neuralgia affecting branches of the trigeminal nerve, temporomandibular disorders, fibromyalgia and disorders of taste or olfaction. Some of these are infectious in origin (e.g., postherpetic neuralgia and some taste disorders); some are traumatic (e.g., some cases of temporomandibular disorder); and for others, the cause or causes are unknown. Patients with facial palsies and trigeminal neuralgia are generally referred to neurologists for treatment. Disorders of taste and smell also require neurological consultation as well as brain imaging because they can be symptomatic of brain tumors. Pain relief may also improve function and can be combined with adjunctive measures such as the use of hot or cold compresses and behavioral treatments such as relaxation and imaging therapy to reduce muscle tension. The variety of pain medications has greatly increased in recent years. They include aspirin and other nonsteroidal anti-inflammatory drugs, tricyclic antidepressants, new antiepileptic drugs, the selective serotonin re-uptake inhibitors and the more potent opiate family of drugs.

If the pain problem has recently developed, providers can take steps to prevent the pain from becoming chronic. This will entail a general health assessment to determine whether there are co-morbidities, including other pain problems, as well as patient questionnaires to provide information on how the pain problem is affecting overall health and well-being. The data collected will record the extent to which the problem interferes with work, social interaction and sleep, whether the patient is experiencing mood changes and symptoms of depression, and what coping skills are manifest. Such patient profiles allow for more selective treatment tailored to the needs of the individual patient.

Patients in whom pain has become chronic and intractable may be referred to an established pain clinic for multidisciplinary treatment and may also be alerted to patient organizations where individuals with similar pain problems can find information and support.

**Temporomandibular disorders**

Among the common types of craniofacial pain likely to be seen by oral care providers are temporomandibular disorders, characterized by symptoms of pain and dysfunction in and around the temporomandibular joints or the masticatory muscles. Temporomandibular disorders may occur as a result of injury, arthritis or fibromyalgia or for unknown reasons. Approaches used to obtain a differential diagnosis of these conditions can range from a physical examination that may include palpation and measuring the mouth opening, to the use of complex imaging and instrumentation, including procedures such as arthroscopy.

Diagnosis of temporomandibular disorders is based on the physical examination and a complete medical and dental history, including information about hearing, speech and swallowing problems, as well as pain and dysfunction. This information can be complemented by data from imaging and other diagnostic tests. Evaluation encompasses examination of oral-facial tissues, musculature and neurological function. Particular attention is paid to measures of the range of motion, mouth opening, existence of any parafunctional conditions (e.g., clenching, grinding), and the presence of joint or muscle tenderness and cutaneous hyperalgesia. Features of the reliability studies on the examination methods have been reviewed. Psychosocial assessments using validated instruments can determine the extent to which pain and
dysfunction diminish the patient’s quality of life and can suggest appropriate treatments. The evidence base for the efficacy of treatment modalities is severely limited and has resulted in a wide range of diagnostics and therapies. Treatments range from conservative and reversible approaches to joint surgical procedures. At present the evidence is insufficient to warrant prophylactic intervention for management of these disorders.

Currently available epidemiological evidence suggests that temporomandibular disorders can frequently resolve over time and that conservative, reversible approaches are the treatments of choice. Ideally, the practitioner and the patient should work together to develop a treatment plan that is evidence-based and patient-centered, taking into consideration all etiologic factors, the level of pain and dysfunction the patient is experiencing, and their impact on the patient’s quality of life.

Factors affecting future dental health care practices

The last decades of the 20th century were witness to major improvements in the prevention, diagnosis and treatment of oral diseases, a trend that will continue to accelerate the paradigm shift in the management of oral diseases from repair of damaged tissues to the control of infections. In addition, modification of risk factor exposures will result in improvements in health and in the management of disease.

A closer look into factors that will affect the future of oral health care requires an overview of the current state of guidelines for oral care and the status of evidence-based practice. The approaches used to determine the evidence for practice and the development of guidelines for care are an emerging field of activity. Education in the health professions is already emphasizing the importance of relying on randomized, controlled clinical trials, the gold standard for judging the merits of proposed interventions, wherever possible.

Evidence-based practice

During the 1990s, “evidence-based medicine” emerged as both popular phraseology and practice philosophy. The origins of evidence-based medicine go back to mid-19th century Paris and earlier, yet the approach is still a relatively young discipline that is now rapidly evolving. Evidence-based medicine has been defined as the integration of “individual clinical expertise with the best available external clinical evidence from systematic research” and with patients’ choices. The skills required include defining a clinical problem, critically appraising the relevant literature, and deciding whether and how to integrate this information into practice (Evidence-Based Medicine Working Group 1992).

Evidence-based medicine is neither a “cookbook” nor an ivory tower approach. The philosophy is being adopted across a range of disciplines, leading to the terms “evidence-based dentistry,” and “evidence-based nursing,” among others. The practice of evidence-based dentistry “incorporates the judicious use of the best evidence available from systematic reviews, when possible, with knowledge of patients’ preferences and clinicians’ experiences to make recommendations for the provision of the right care, for the right patient, and at the right time.”

The reliance on evidence using systematic reviews of the literature has led to initiatives in the United States, Canada and Europe to enhance the conduct and use of systematic reviews. The Agency for Healthcare Research and Quality (AHRQ) created 12 evidence-based practice centers in 1997 to conduct systematic reviews and develop evidence reports. The Cochrane Collaboration and the Centre for Reviews and Dissemination at the University of York are examples of prominent activities in the United Kingdom to support systematic reviews. The Cochrane Oral Health Review Group, one of 50 specialty review groups within the Cochrane Collaboration, has a number of systematic reviews completed or under way of interest to oral health practitioners. In Canada, considerable contributions to the field have been made by McMaster University and the Canadian Coordinating Office for Health Technology Assessment.

In the United States, the National Institute of Dental and Craniofacial Research joined efforts with AHRQ in 1999 to designate one of AHRQ’s Evidence-based Practice Centers to conduct reviews on oral, dental, and craniofacial diseases and disorders. The work of this center should significantly strengthen the scientific base of knowledge related to the diagnosis and management of oral, dental, and craniofacial conditions. Examples of topics that will be reviewed include the management of dental caries and dental care of medically compromised patients, including patients with HIV disease.

Conclusion

During the past several decades, there have been major improvements in the prevention, diagnosis and treatment of oral diseases. Enhanced disease prevention and health promotion will require the participation of all health professions, especially in addressing common risk factors such as tobacco, alcohol and inappropriate dietary practices. The field of diagnostic tests for oral diseases should continue to expand, enabling clinicians to analyze the risk of disease and disease progression for individual patients. Full assessment of the strengths and weaknesses of new diagnostic tests and evaluation of when they are best used will be key to proper interpretation of the results, permitting tailored referrals and treatments.

Treatment options for individual patients are increasing, including the recent efforts to understand and define early childhood caries and other periodontal infections. The increased knowledge of risk factors, the importance of monitoring disease progression and treatment effects, and the ability to diagnose conditions and intervene earlier will necessitate increased involvement of all health professionals in oral health care and may reflect changes in care provision and referral patterns.

Management of conditions such as oral and pharyngeal cancers, cleft lip/palate, and chronic pain requires multidisciplinary teams. The promotion of oral health and the prevention of oral disease are at a turning point. A systematic approach to integrate the scientific findings into evidence-based assessments will provide clearer guidance to all health care professions and the public. To capitalize on the rapidly emerging science base, the active participation of a full range of dentists and additional health care providers with individuals in the community is needed.

References

American Surgeon General World Health Organization

ORAL DISEASES, INFECTIONS AND CRANIOFACIAL DISORDERS

Final Examination Questions

Select True or False for questions 1-5 and mark your answers on the Final Examination sheet found on page 81 or complete your test online at www.elitecme.com.

1. The periodontal ligament, which anchors the teeth in the jaws, is a tactilely sensitive tissue providing important feedback with regard to mastication and dental occlusion.

   True  False

2. The effects of xerostomia may be particularly severe in cancer patients receiving radiation to the head or neck because the rays can destroy salivary gland tissue rather than simply inhibiting salivary secretion.

   True  False

3. Patients with diabetes mellitus are less susceptible to periodontal diseases.

   True  False

4. Current U.S. Public Health Service (USPHS) recommendations for fluoride use include an optimally adjusted concentration of fluoride in drinking water ranging from 1.5 to 2.0 ppm, depending on the mean maximum daily air temperature of the area.

   True  False

5. Temporomandibular disorders may occur as a result of injury, arthritis or fibromyalgia or for unknown reasons.

   True  False

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