CHAPTER

8

Oral Health in Elderly People

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As the first segment of the gastrointestinal system, the oral cavity provides the point of entry for nutrients. The condition of the oral cavity, therefore, can facilitate or undermine nutritional status. If dietary habits are unfavorably influenced by poor oral health, nutritional status can be compromised. However, nutritional status can also contribute to or exacerbate oral disease. General well-being is related to health and disease states of the oral cavity as well as the rest of the body. An awareness of this interrelationship is essential when the clinician is working with the older patient because the incidence of major dental problems and the frequency of chronic illness and pharmacotherapy increase dramatically in older people.

REVIEW OF ANATOMY AND FUNCTIONS OF THE ORAL CAVITY

ANATOMY OF THE ORAL CAVITY

The major parts of the oral cavity (**Figure 8-1**) are lips; vestibules; teeth; maxilla and mandible (upper and lower jaws, respectively); alveolar bone (termed *residual bone* if there are no teeth); gingivae (gums); hard and soft palates (roof of the mouth); tongue and mucous membranes (floor of the mouth); temporomandibular joint (TMJ); buccal mucosa (lining of the cheeks); salivary glands; and muscles of mastication and facial expression (orofacial musculature). Throughout the mouth there are blood vessels, lymphatics, and nerves to ensure rapid communication between the oral cavity and other major organ systems.

At the lips, the skin of the face is continuous with the mucous membranes of the oral cavity. The bulk of the lips is formed by skeletal muscles and a variety of sensory receptors that judge the taste and temperature of foods. Their reddish color is to the result of an abundance of blood vessels near their surface.

The vestibule is the cleft that separates the lips and cheeks from the teeth and gingivae. When the mouth is closed, the vestibule communicates with the rest of the mouth through the space between the last molar teeth and the rami of the mandible.

Thirty-two teeth normally are present in the adult mouth: two incisors, one canine, two premolars, and three molars in each half of the upper and lower jaws. The teeth in the upper jaw are termed *maxillary*, and the teeth in the lower jaw are termed *mandibular.* The mandible is the movable member of the two jaws, whereas the maxilla is stationary. The components of an individual tooth provide a framework within which to appreciate changes that occur with age (**Figure 8-2**). Teeth are highly calcified structures composed of four parts: (1) enamel, the hard, brittle substance covering the outer surface of the crown of the tooth; (2) dentin, a bonelike substance forming the main body of the tooth, surrounding the pulp cavity, and covered by enamel on the crown and cementum on the root; (3) cementum, a bonelike substance that covers the tooth root; and (4) pulp chamber and canal(s), the soft central parts of the tooth that contain the blood vessels, nerves, and lymphatics. Each tooth in the mouth is surrounded and supported by alveolar bone. The visible portion of the tooth is termed the crown. The portion

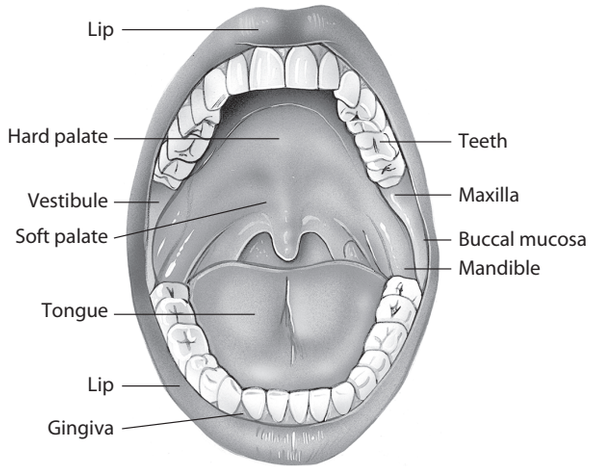

Figure 8-1 Major parts of the oral cavity

submerged below the gum line is the root. The region where these portions meet is called the *neck* of the tooth. The gingiva surrounds the necks of the teeth and covers the alveolar bone. It is composed of dense, fibrous tissue covered by a smooth vascular mucosa. The tooth roots are joined to the alveolar bone by periodontal ligaments.

The hard palate forms the roof of the mouth in the chewing area, and the soft palate lies just posterior to it. The floor of the mouth is formed by the tongue, which nearly fills the oral cavity when the mouth is closed, and mucous membranes. The tongue is a mobile mass of mostly skeletal muscle covered by a mucous membrane with numerous papillae on the surface.

The TMJ, located just anterior to the earlobe, is the only joint needed for chewing. A hingelike movement occurs bilaterally in the TMJ during mouth opening and closing. During chewing, the mandible also exhibits protrusive and lateral movements.

The buccal mucosa forms the side walls of the oral cavity and contains numerous mucous glands. The secretions from these glands mix with food in the mouth to aid in both chewing and swallowing.

There are three major (bilateral) salivary glands, which secrete saliva into the mouth: parotid, submaxillary, and sublingual. The parotid glands are the largest, and their ducts open into the vestibules opposite the upper second molar teeth. The submaxillary (or submandibular) gland ducts open into the floor of the mouth under the tongue from their location in the angles of the mandible. The sublingual glands, the smallest of the major salivary glands, are embedded in the mucous membranes of the floor of the mouth. Their ducts open under the tongue as well. The major salivary glands contribute about 95% of the total daily volume of saliva; the remaining 5% comes from numerous minor salivary glands in the mucous membranes of the lips, tongue, palates, and cheeks.[1] The primary role of saliva is to protect and maintain oral health.[2] Human saliva contains lubricatory factors (mucins) to keep oral tissues hydrated, pliable, and insulated; contains many antibacterial proteins that regulate colonization of oral bacteria; buffers the acid produced by oral bacteria to maintain tooth integrity; aids in carbohydrate digestion; mediates taste acuity; and is necessary for mastication and preparation of food for swallowing.[3–5]

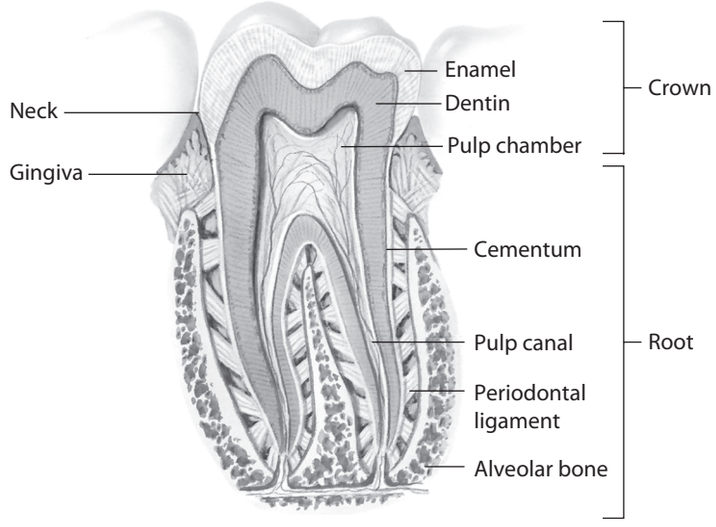

Figure 8-2 Components of a tooth

The orofacial musculature consists of almost 20 muscles of facial expression and four muscles of mastication: the masseter, the lateral pterygoid, the medial pterygoid, and the temporalis muscles. One of the facial muscles, the buccinator, acts as an accessory muscle of mastication by eliminating the space of the vestibule between the cheek and the jaws during chewing.

FUNCTIONS OF THE ORAL CAVITY

The oral cavity serves in the masticating, tasting, and swallowing of food; as a phonetic box for speech; and as a secondary pathway for breathing. The major and minor salivary glands provide moisture to soften foods as well as supply carbohydrate-digesting enzymes.

Mastication (Chewing)

The teeth are designed for chewing; the anterior teeth provide a strong cutting action, and the posterior teeth provide a grinding action. The names of the teeth demarcate their four basic functions. The incisors cut or slice food, the canines tear food, the premolars shred food, and the molars grind food in preparation for swallowing.

Proper chewing is important in the digestion of all foods, especially most fruits and raw vegetables, which contain indigestible cellulose membranes that must be broken down before the food can be used by the body. Also, because digestive enzymes act only on the surfaces of food particles, the rate of digestion is highly dependent on the total surface area of the chewed food that is exposed to intestinal secretions.

The act of chewing has more significance than the mere preparation of food for swallowing. The food is moved around the mouth so that the taste buds are stimulated, and odors are released that stimulate the olfactory receptors. Much of the satisfaction and pleasure of eating depends on these stimuli.

Digestion in the Mouth

Saliva contains the digestive enzyme ptyalin (amylase), which functions to hydrolyze starches into two disaccharides, maltose and isomaltose. This is the first step in the digestion of carbohydrates. However, because food stays in the mouth for such a limited amount of time, only 3% to 5% of the starches that are eaten are hydrolyzed by the time the food is swallowed.[2] Most naturally occurring starches are digested poorly by ptyalin because they are protected by a thin cellulose cover. Cooking destroys these cellulose membranes and facilitates digestion in the mouth.

In general, swallowing can be divided into three stages: (1) the voluntary stage, which initiates the swallowing process; (2) the pharyngeal stage, which is involuntary and involves the muscular contractions for the passage of food through the pharynx to the esophagus; and (3) the esophageal stage, another involuntary phase that promotes the passage of food from the pharynx to the stomach. The oral cavity is involved with only the first (voluntary) stage of swallowing, which takes about 1 second.[6] When the food is ready to be swallowed, pressure from the tongue upward and backward against the palate forces the bolus of food posteriorly into the pharynx. From here on, the process of swallowing becomes automatic and usually cannot be stopped.

Speech

Speech is a complex behavior that integrates the processes of respiration, phonation, oral sensation, resonation, and articulation.[6] The mouth is one of the resonators for speech and other vocalizations. The three major organs of articulation are the lips, tongue, and soft palate. Speech, therefore, relies heavily on the anatomic structures of the oral cavity.

ORAL HEALTH STATUS AND NEEDS IN OLDER ADULTS

ORAL HEALTH STATUS

Oral health implies a state that is stable, relatively disease free, and comfortable and that permits adequate functioning for mastication, swallowing, and speech. Poor oral health can be viewed as a state of inadequate functioning resulting from decayed teeth; periodontal disease; ill-fitting dentures or lack of dentures; neglect of oral hygiene; and the presence of pain, inflammation, or infection in the oral cavity. Although few of these conditions pose mortality risks, they can lead to physical dysfunction, pain, and psychological anguish in the older patient.

A major criterion of successful aging is how well the individual maintains oral health, the ability to chew, the ability to talk, and personal satisfaction with appearance.[7] Unfortunately, the mouth often becomes one of the first areas of the body to be neglected by people who have chronic diseases and infirmities in old age.

There have been five national surveys indicating the oral status of elderly adults, and the older aged were excluded in two of the studies: the National Health Examination Survey of 1960–1962 excluded participants older than 79 years,[8] and the National

Health and Nutrition Examination Survey (HANES I) of 1971–1974 included no subjects older than 74 years.[9]

In 1978, the National Institutes of Health (NIH) introduced an oral physiology and aging component to the Baltimore Longitudinal Study of Aging.[10] In Iowa, a longitudinal survey was initiated in 1981 to determine the prevalence and incidence of oral conditions in noninstitutionalized rural elderly Iowans. In 1987, the National Institute of Dental Research (NIDR) published national and regional data on the prevalence of oral conditions in 15,000 working adults and 5600 elderly people who attended multipurpose senior centers.[11] Useful data were obtained from this survey of adult oral health, even though the older participants were not entirely representative of the nation's elderly population. The findings disclosed that Americans are keeping their teeth longer, are going to the dentist more often for preventive checkups, are reducing the number of cavities in their mouths, and have practically eliminated edentulousness in middle age. Serious dental problems, however, still exist among elderly individuals (**Table 8-1**).

ORAL HEALTH NEEDS

In general, studies reveal that many unmet treatment needs affect a large portion of the noninstitutionalized elderly population. This is true whether the study has been conducted by direct examination of patients or by survey. Data from HANES I indicated that 60% of elderly subjects had at least one dental treatment need.[9] The survey of rural elderly Iowans also found a high level of treatment needed: 40% required at least one restoration, 16% at least one extraction, and 27% some prosthodontic treatment; more than 60% of the dentate subjects needed some periodontal treatment.[12–14]

Many local studies and one statewide survey have documented the large need for dental care among institutionalized residents.[15,16] The results have ranged from 2.3 dental services needed per person, with 3.2 services required for those having remaining natural dentition, to 82.5% of 3247 patients screened in Vermont nursing homes in 1982.[15,16] In the latter study, examiners found that 37.2% of the residents required immediate attention to eliminate pain, infection, concern of malignancy, or a combination of these symptoms. More studies are needed to explore the association between oral health status and nutrition in institutionalized older adults.[17]

CHANGES IN ORAL AND CIRCUMORAL STRUCTURES WITH AGING

OVERVIEW

Differentiation of normal aging changes from disease processes in old age is of paramount importance. Not knowing the changes that occur with age might lead to excessive or unnecessary treatment. Erroneously evaluating a disease process as normal aging might have equally serious consequences. Unfortunately, lack of research on the aging oral cavity has resulted in a number of stereotypes and generalizations.[18] Standard graphs, tables, and information in many geriatric medical and dental textbooks show inevitable decrements with age. However, these studies included subjects who, although superficially healthy, in fact had some disease or were taking medication

Table 8-1 Comparison of Oral Health Status of Employed Adults and Older Adults

Oral Status/Treatment Need	% of Employed Adults	% of Older Adults
Calculus deposits	83.9	88.9
Edentulousness	4.2	41.1
Gingival bleeding (after probing)	43.6	46.9
Gingival recession (1+ mm)	51.1	88.3
Periodontal attachment loss (1+ sites)	76.7	95.1
Retention of all teeth	36.7	2.1
Root caries	21.1	56.9
Visited dentist in past 2 years	79.6	56.4
Perceived need for dental care	50.4	36.0

Reprinted from: National Survey of Adult Dental Health, *Oral Health of United States Adults: National Findings, 1987*, National Institute of Dental Research.

that affected oral function.[19] Most of this early information therefore probably reflects oral changes resulting from disease or its treatment rather than dysfunctions related directly to increased age.

HARD TISSUES

Bone

In the developmental years, bone resorption and deposition occur synchronously in the process of growth and remodeling. Alveolar bone, however, has a remodeling rate greater than that of the other bones of the body.[20] With maturity, bone is notably less active, although there is still some degree of continuing resorption and deposition. After age 35 to 40 years, approximately 1% of bone mass is lost per year in both men and women.[20]

As physical activity diminishes in the later years, so too does the demand for new bone formation. Resorption exceeds deposition, resulting in a net loss of bone. By the time old age is reached, atrophy has resulted from slow resorption with very little remodeling. Not only is there a generalized decline in bone volume, but the composition of bone gradually alters also, resulting in reduced resilience and increased brittleness and fragility.[21,22]

Alveolar bone is one of the first bones to be affected by loss of mass. The periosteal and periodontal surfaces of alveolar bone become less resistant to harmful local oral trauma, inflammation, or disease.[23] This is a major factor contributing to periodontal disease, loss of teeth, and, in the edentulous patient, inability to obtain adequate support and stability for dentures.[18,24] In both the maxilla and the mandible, the amount, extent, and uniformity of the bone loss differ with varying etiologies and health status.[25] It is now recognized that alveolar bone or residual ridge resorption is confounded by such factors as age, sex, race, and health status of the patient when the teeth are extracted; the tooth extraction technique; the diet of the patient; the presence of local factors; and the frequency of denture use.[25]

Teeth

It is frequently reported that the teeth themselves undergo changes with age (**Table 8-2**). Teeth differ from most other parts of the body in that the reparative or regenerative capacity of their constituent tissues is extremely limited. Also, the blood vessels and nerves become less active with age; as a result, the vitality of the average human tooth pulp lasts approximately 70 years.[22]

Table 8-2 Anatomic Changes in the Teeth with Age

Enamel
Regeneration/repair—incapable
Permeability—decreased
Dentin
Permeability—decreased
Sensitivity—decreased
Calcification—increased
Pain conduction—decreased
Repair—capable with vital tooth pulp
Pulp chamber and canals
Cellularity—decreased
Innervation—decreased
Tooth drainage—decreased
Vascularity—decreased
Volume—decreased (due to deposition of reparative dentin)
Apical foramen
Size—decreased (may cause decreased pulp vascularity and innervation)
Cementum
Deposition—continuous (major cause of decreased apical foramen size)
Repair—capable with vital tooth pulp
Resorption—increased susceptibility
Entire tooth
Brittleness—increased (predisposing tooth to cracks, fractures, shearing)
Darkening—increased
Pain sensitivity—decreased
Thermal sensitivity—decreased
Translucency—decreased

Attrition

The remaining natural teeth are likely to exhibit some flattening of the chewing surfaces induced by repeated contact with opposing teeth during masticatory movements. To compensate for the natural wear of these surfaces, human teeth erupt with their supporting structures throughout adult life.[26]

The patterns of tooth wear vary with each patient and are cumulative because the enamel is incapable of repair or regeneration. The wear can range from minimal faceting to extreme loss of tooth substance, sometimes extending to the gingiva. However, there is no agreement on the point at which physiologic attrition becomes pathologic or contributes to pathologic conditions.[27] In areas of excessive wear, reparative (or secondary) dentin is deposited on the walls of the pulp chamber and canals for protection. This, as well as reduced innervation, helps to explain the reduced tooth pain sensitivity and higher pain threshold in elderly people.

Temporomandibular Joint

The temporomandibular joint (TMJ) is a complex, diarthrodial joint capable of both swinging (hinge-like) and sliding motions on many axes. It undergoes functional remodeling throughout life, usually in response to changes in articulation of the teeth or alterations in the space between the maxilla and mandible. The functional changes in the TMJ are by no means confined to elderly individuals.

Signs of TMJ change include joint clicking, limitation of jaw opening, and deviation of the mandible during function, with the major symptom being pain. Researchers with the Baltimore Longitudinal Study of Aging assessed all of these for arteriosclerosis or obliteration of the capillaries.[28] More research into the aging TMJ is needed because these age-related changes might explain some of the masticatory problems in this age group.

SOFT TISSUES

Mucous Membranes

The stereotypic effect of aging on the oral mucosa is that atrophic changes occur. Clinically, these changes involve the surface epithelium becoming thinner, drier, less elastic, less vascular, less firmly attached to the underlying connective tissue and bone, and more susceptible to injury from mild stresses.[22,29] Other changes occur as well with reduction in connective tissue and subcutaneous fat and increased linkage of collagen molecules. Some symptoms have been associated with these alterations, including xerostomia (mouth dryness) and sensations of pain or burning on the tongue, palate, or oral mucosa.[18] These changes, however, must be interpreted with caution. In critical reviews of the literature, Baum and Hill suggested that no conclusions could be drawn about whether atrophy of the oral mucosa is associated with aging.[18,30] Other researchers have concluded that specific alterations of the oral tissues might instead be induced by a host of environmental factors, such as tobacco smoking or chronic systemic disease.[31]

Periodontium

Gingiva

The gum tissue of the elderly individual gradually recedes from the tooth, with subsequent exposure of more of the tooth surface and root. The degree that gingival recession progresses is related to age, tooth movement, inflammatory changes resulting from disease, oral care habits, and heredity.

Periodontal Ligament

The periodontal ligament is not one ligament but a series of short, dense ligaments connecting the cementum of the tooth root to alveolar bone. Because the ligament is made up of connective tissues, aging affects it in the same way it does other connective tissues in the body.[32] The result is a progressive loss of soft tissue attachment, leading to exposure of the root and loosening of the teeth within their bony sockets.

Tongue

The status of the tongue in aging, independent of diseased states and taste acuity, has not been studied in detail. Tongue vascularity changes very little compared with that of other organs because there is little tendency in this tissue for atherosclerosis or obliteration of the capillaries.[31] There is much controversy about whether aging is associated with atrophy of the papillae, increased formation of fissures, and decreasing sensitivity to gustatory stimuli in the tongue.[31,33–35]

CIRCUMORAL TISSUES

Oral Musculature

Changes in aging oral musculature are consistent with those in aging muscle tissue in the body as a whole.[36] In general, there are reductions in muscle tone, muscle performance, number and activity of muscle cells, and number and size of the muscle fibers.[6,37] Replacement of the muscle mass by fat or fibrous connective tissue results in generalized atrophy of the musculature attached to the bones in the oral cavity.[38]

Mastication

The muscles of mastication atrophy with age; this decreases the biting force and slows chewing performance.[39] The atrophy is probably caused in part by disuse because less muscular effort is required for chewing as a result of failing dentition or a progressively softer diet or both. In either case, the generalized loss of muscle mass decreases the biting force and can make chewing difficult.

Deglutition

Aging does not significantly affect the transit of the prepared food bolus through the mouth and pharynx.[40] However, the impact of decreased muscle mass and tone can make swallowing difficult and can alter the ability to form and prepare a bolus in

the oral stage of swallowing.[6] Several studies have demonstrated that as people age they take longer and expend more effort to prepare a food bolus before swallowing.[39,40] In the Oral Health: San Antonio Longitudinal Study of Aging (OH:SALSA), intraoral tactile perception was found to be preserved during aging.[41]

OTHER CHANGES

Salivary Glands

In early reports, decreased salivary flow was generally considered to be concomitant with increased age.[18] More recent evidence indicates that the diminished salivary flow often noted in studies of elderly subjects is caused by pathologic conditions or pharmacologic effects of medications, rather than aging.[2,5–7,42] Because diminished salivary flow does not occur in healthy, nonmedicated individuals, these findings emphasize that the elderly person might be susceptible to situations and therapies that result in a reduction of saliva availability.[43] However, OH:SALSA data revealed significant age-related alterations in salivary function that were associated with caloric intake in nonmedicated elders.[44] These latter results indicate that more studies are needed.

Sense of Taste

The sense of taste is a function of the taste buds in the mouth. Its importance in nutrition lies in the fact that it enables a person to select food in accord with personal desires and needs of the tissues for specific nutritive substances.

Taste Buds

The taste buds are found predominantly on three of the four different types of the tongue papillae (circumvallate, fungiform, and foliate), although they are also located in the epithelium of the palate, tonsillar pillars, and other points around the nasopharynx.[45] In early anatomic studies, marked decreases in the numbers and atrophy of the taste buds with aging were reported.[46] However, more recent investigations indicate there is no significant loss of taste buds in old age.[47]

Taste Sensitivity

Elderly people often complain of altered taste sensations (dysgeusia), decreased ability to perceive taste (hypogeusia), or complete loss of taste (ageusia). Few studies, however, have been able to determine the reasons for these decrements in taste sensitivity.[45,48] One explanation is decreased salivary flow because taste buds react only to dissolved compounds.

Although there is some agreement that taste sensitivity begins to decline after age 55 years, differences in research methods have produced different results.[49] Some studies have indicated that older adults need higher concentrations of the four primary sensations of taste (salty, sour, sweet, bitter) for identification than do children and younger adults.[50] Others have found that the taste buds detecting saltiness and sweetness are the first to deteriorate and that sensitivity to sour and bitter tastes declines later.[51,52] The use of psychophysical procedures found only minimal changes in taste sensitivity, even though a steady decline in taste sensitivity with increasing age is still being found.[34,35]

Sense of Smell

Smell is the least researched and understood sense. This is a result in part of the location of the olfactory sensory receptors in specialized epithelial tissue of the nasal cavity and in part of the fact that the sense of smell is a subjective phenomenon that is not easily measured. In fact, much of what we call taste is actually smell, which largely determines the flavor and palatability of foods and beverages.[53]

Evidence of a decline in olfactory sensitivity with age is still very limited, and the cause of any decrement remains speculative.[54] Some investigators have found reduced smell sensitivity, with the greatest problems in recognition and identification of odors among persons older than 80 years.[55] Others have reported age-related losses with significant individual differences.[56] It has also been suggested that poor health and smoking can cause an even greater decline in smell sensitivity than does age alone.[56]

CHANGES IN ORAL AND CIRCUMORAL STRUCTURES WITH DISEASE

OVERVIEW

There are three major characteristics of dental diseases: universality, irreversibility, and cumulativeness (**Table 8-3**). Although these diseases are rarely debilitating or life threatening, there are indications that they have a significant impact on social, economic, and psychological areas of life, including the quality of life.[57] Older adults, however, tend to place minimal importance on their oral health and accept functionally inadequate dentition as an unavoidable consequence of the aging process.

Table 8-3 The Major Characteristics of Dental Diseases

1. *Universality:* Diseases of the oral cavity are the most prevalent of all diseases; dental caries and periodontal disease usually affect most people throughout life.
2. *Irreversibility:* The damage derived from the common oral diseases, such as dental decay or bone loss, is irreversible, although treatment can usually intercept its spread.
3. *Cumulativeness:* The structural losses induced in the teeth and their supporting alveolar bone by oral diseases are cumulative.

Source: Vergo TJ Jr, Papas A. *Physiological aspects of geriatric dentistry*. J Dent. 1988;37(4):165–168. Reprinted courtesy of the Journal of the Massachusetts Dental Society.

Many of the oral diseases that afflict elderly individuals are diseases of all age groups. Therefore, preventive dentistry remains an important aspect of their oral health care and should involve all three levels of prevention: (1) preventing initiation of disease, (2) preventing the progression and recurrence of disease, and (3) preventing the loss of function and loss of life.[58]

The prevention of dental disease requires that all individuals see a dentist at least yearly, whether they have natural teeth, no teeth, complete dentures, or teeth and dentures. First, this allows primary prevention procedures to be evaluated and reinforced regularly. Detrimental habits, environmental factors, and nutritional status all affect the likelihood that oral pathology will occur. As an example, poor oral hygiene significantly contributes to the diseases caused or aggravated by bacterial plaque infection, and it is well known that the level of oral hygiene deteriorates with age.[59,60] Second, regular dental care can lead to early diagnosis and treatment of the pathologic conditions described in the following sections.

HARD TISSUES

Bone

Resorption

Bone resorption is associated with the loss of mineral content, increase in porosity, and generalized atrophy. Clinical observation of alveolar bone resorption has so far failed to provide a clear understanding of the mechanisms responsible. A portion of the population shows bone loss without the other findings usually associated with active periodontal disease. This loss is also seen in the absence of dentition.[61]

Resorption of alveolar bone occurs in two dimensions: The mandible resorbs primarily in a vertical direction, resulting in loss of bone height. The maxilla resorbs primarily in a horizontal direction away from the covering lips and cheeks. This means that the chin will appear to protrude because the maxilla has receded horizontally. This leads to the characteristic "toothless look": a shortening of the distance between the chin and nose and a pulling inward of the lips.

Resorption is greater in the mandible than in the maxilla and, when severe, constitutes a major problem in the wearing of dentures. The residual bony ridge can become thin and knifelike and unable to withstand the downward compressive forces of a conventional denture.

Osteopenia and Osteoporosis

Osteopenia refers to metabolic bone diseases that are characterized by x-ray findings of a subnormal amount of mineralized bone mass. The most common osteopenia is osteoporosis, generally defined as a decrease in the quantity of bone, with an increased incidence of fractures from minimal trauma.[62] Osteoporosis is observable within the oral cavity as dental osteopenia.

One of the first signs of osteoporosis is alveolar bone loss, followed by loss in the vertebrae and long bones.[63] Indeed, there is a significant correlation between skeletal osteopenia and density of alveolar bone and residual ridges.[63] Radiographically, it appears as diminished bone mass in the mandibular angular cortex, decreased trabeculae, and a diminished alveolar crest.[64] Subsequently, it leads to an inadequate amount of bone mass in the mandible, loss or mobility of teeth, edentulousness, and inability to wear dentures.[65] The loss of teeth and the use of ill-fitting dentures also cause extensive alveolar bone or residual ridge atrophy.[65,66]

Although calcium deficiencies and calcium–phosphorus imbalances are contributing factors in the pathogenesis of osteoporosis, prevention and management include not only increased calcium intake but estrogen therapy, bone-building or antiresorptive agents, dietary vitamin D, and exercise.[63,67–69] Although fluoride therapy is used extensively in preventive dentistry, its widespread use in the prevention of osteoporosis is still being investigated.[70]

Recent reports raise concerns that patients undergoing long-term antiresorptive bisphosphonate therapies might be at risk for developing osteonecrosis of the jaw (bisphosphonate-related osteonecrosis

of the jaw, or BRONJ).[71] Thus far, the vast majority of incidents of BRONJ have occurred in cancer patients given intravenous bisphosphonate therapy for control of bone metastasis and pain and were associated with oral infections and/or trauma and/or surgery. The oral bisphosphonate treatment associated with BRONJ accounts for only a small percentage of cases. However, millions of patients worldwide who have osteoporosis are on an oral bisphosphonate regimen; the long-term adverse effects have not been well evaluated. Prospective and controlled studies are required to understand the epidemiology, pathophysiology, risk factors, and treatment for BRONJ.[72,73] At this time, there is no contraindication for routine dental care in those patients without oral trauma who are on bisphosphonate therapy solely (i.e., not for the treatment of metastatic bone cancer or pain) for the treatment or prevention of osteoporosis.

Teeth

Dental Caries (Tooth Decay): Coronal and Root Surfaces

Dental caries has been considered a disease of young people that stabilizes in the mid-20s and remains dormant until periodontal disease or gingival recession exposes the roots of the teeth and caries of the root surfaces occurs.[58,74,75] However, more recent research indicates that a significant increase in caries, including recurrent decay around restorations, cervical caries at the gingival margin, and root caries, is associated with aging.[11,76,77] Most of the recurrent caries are in the proximal regions of the teeth.[78] Root caries occurs following exposure of the tooth root.[77,79–81]

The diagnosis of dental caries is based on x-ray examination and clinical observation because there is no absolute correlation between the presence or extent of dental decay and symptoms. It is generally recognized that four things are necessary to produce a carious lesion: cariogenic bacteria, a substrate of dietary carbohydrates, a susceptible host (tooth), and time. However, factors that contribute to high caries risk include poor oral hygiene, gingival recession, reduced salivary flow (which increases plaque accumulation), bacterial virulence, and diet.[78]

Conditions that predispose to xerostomia (dry mouth), such as medications, Sjögren's syndrome, and head and neck irradiation, can promote rampant dental caries in all age groups.[82] In fact, the incidence of root surface caries in elderly people is significantly correlated with a low rate of salivary secretion.[83]

Historically, the most effective strategy for preventing dental caries has been increasing tooth resistance to pathogenic plaque through the use of fluorides by systemic introduction (water, diet) and topical application (professional or self applied).[84,85] Even fluoridation of other vehicles, such as salt, milk, and sugar, has been considered in areas where no reticulated water supplies exist.[86] In patients with xerostomia, the development of caries has been avoided largely by daily topical application of either 0.5% sodium fluoride or 0.5% stannous fluoride solutions.[87]

Unfortunately, there are few clinical data supporting the use of topical fluoride for prevention of dental caries in geriatric patients with adequate salivary flow.[87–90] Newbrun, however, believes that the elderly population would benefit from the use of fluoride dentifrices, mouth rinses, and gels applied by brush, finger, or plastic tray.[91] The method to be used is dictated by the anticipated susceptibility to decay and the ability of the patient to manage the regimen.

Because both coronal and root caries are plaque-related diseases, measures that limit or inhibit plaque formation should be effective in prevention. Mechanical oral hygiene techniques and chemical antimicrobial agents such as chlorhexidine reduce bacterial flora and substrate.[92] Even dietary modification decreases the amount of substrate, acid production, and decalcification of teeth if individuals eliminate or reduce the intake of foods that are soft, sticky, retentive, or high in sugar and if they chew firm foods.[9]

Abrasion and Erosion

An aging population with longer retention of teeth is at increased risk for both abrasion (wear of tooth structure by nonmasticatory mechanical forces) and erosion (wear of tooth structure by chemical dissolution). The incidence of both of these conditions increases with age simply because any damage to the teeth is cumulative. In patients with xerostomia, the diminution in the mucin level of the oral cavity provides less lubrication and protection, posing an even greater risk for abrasion and erosion.

A major etiologic factor of abrasion is overzealous and improper toothbrushing with a hard-bristle brush or an abrasive dentifrice.[93] The damage appears as transverse scoring of the tooth surface and tends to be asymmetric in its severity, depending on whether the patient is right-handed or left-handed.[93] Prevention is largely a matter of proper toothbrushing and the use of a soft-bristle brush and toothpaste with minimal abrasivity. Other forms of abrasion result from holding objects with the teeth, chewing tobacco, and using dental floss and toothpicks improperly.

Erosion is a chemical process that occurs when the concentration of acid in the mouth is too high for

the saliva to neutralize. The most common causes are chronic ingestion of fruits, fruit juices, and carbonated beverages; sucking candies containing phosphorus or citric acid; and gastroesophageal reflux.[94–96] Erosion can also result from working in an industry that uses or produces acidic materials.[97,98] To prevent dietary erosion, the use of straws with fruit juices and carbonated beverages and the substitution of sugar-free candies or gums are indicated.

The lesions of abrasion and erosion look different: The former are characteristically narrow in relation to depth, and areas of erosion are usually saucer shaped.[22,99] Generally, these lesions do not require restorative treatment unless they are extensive or symptomatic.[99]

Hypersensitive Dentin and Cementum

Exposure of dentin or cementum from abrasion, erosion, acute or chronic trauma, or various restorative treatment procedures can lead to hypersensitive dentin and cementum. The teeth are exquisitely sensitive to exposure to any chemical, thermal, tactile, or osmotic stimulus. Although there is great individual variation in pain sensation, hypersensitivities to sour, sweet, cold, hot, and mechanical irritations are most common.[100]

Because hypersensitivity can deter a person from establishing or maintaining adequate oral hygiene procedures, decreasing sensitivity is the first step in treatment. One method is "sealing" the exposed tooth surfaces by applying agents or dentifrices such as fluoride gels and rinses.[101] In some cases, dental restoration and even endodontic therapy can be necessary to arrest the progress of the lesion, restore the function and shape of the tooth, and relieve pain.[101]

Tooth Loss and Edentulousness

Tooth loss is an irreversible, cumulative process that is no longer considered a natural consequence of aging. Instead, it is known to be the ultimate sequelae of the two most common dental diseases, dental caries and periodontal disease. Nevertheless, tooth loss increases in frequency with age. By age 65 years, approximately 40% of Americans have lost all their teeth; another 20% have lost more than half their teeth.

In all age groups, the total loss of teeth is historically related to increased sugar consumption, combined with ignorance of prevention and insufficient dental manpower resources at the time.[26] Tooth loss in adults older than 35 years has been consistently attributed to periodontal diseases.[102] However, recent studies of tooth loss in adult populations indicate that caries is most often the cause for tooth extractions.[103,104]

The rate of edentulousness, or total lack of teeth, is declining in the elderly population of the United States. In 1957, 67.3% of persons older than 65 years were edentulous, whereas only 45.5% were so in 1971.[105] This was largely to the result of the introduction of preventive and restorative dental procedures. Today, less than 40% of elderly people are edentulous, and this number is rapidly decreasing.

In the recent national survey of employed and older adults, in the group aged 55 to 64 years, less than 15% were edentulous.[11] However, the prevalence of lost teeth was still extensive enough to compromise the employed adults' dentition and to impair function in most of the older adults.[11,106] This means that the fewer missing teeth predicted to occur in the future will lead not only to an increase in tooth-related diseases, but also to the continuing need for regular dental care.

Temporomandibular Joint (TMJ)

Dysfunctions of the TMJ have their primary base in the joint mechanism, even though the actual dysfunction might involve the ligaments, the muscles, or the bone itself. One-half of the edentulous and one-third of the dentulous older population have signs and symptoms of TMJ disorders, including soreness of the jaw; dull, aching facial pain; severe pain in the joint area; tenderness or pain of the masticatory and facial muscles; dizziness; headaches; impaired hearing or earache; eye pain; chronic fatigue; and popping, clicking, or cracking noises near the ear while opening and closing the mouth.[107,108] These manifestations are dynamic, characterized by periods of quiescence and exacerbation, and have a wide range of expression among patients.[109] However, elderly individuals appear to have more symptoms than do younger persons.[107]

The causes of TMJ dysfunction can be external or internal or both. External causes include degenerative joint disease; alveolar bone resorption; and injuries to the head, neck, or mandible. Internally, attrition, malocclusion, and bruxism, in either natural or artificial teeth, can cause the facial muscles and TMJ to quit working together correctly.[110]

Diagnostic and treatment decisions are based on symptom reports and clinical examination findings. Management therapies advocated for TMJ dysfunction include applying moist heat to the face; using prescribed muscle relaxants or other medications; massaging the muscles; eating soft and nonchewy foods; undergoing counseling; training in biofeedback or relaxation procedures; correcting the bite of the teeth; and, in severe cases, undergoing surgery.

SOFT TISSUES

Mucous Membranes/Epithelium

The oral mucosa is composed of both keratinized and nonkeratinized epithelium. In addition, the mouth has a dark, moist environment that is replete with microorganisms. The oral mucosa also can be subjected to several environmental influences, such as smoking; chewing of the lips and cheeks; eating a variety of foods; and sources of trauma, allergy, and carcinogenesis.[111]

Aphthous Ulcer (Canker Sore, Aphthous Stomatitis, Ulcerative Stomatitis)

Aphthous ulcers appear as shallow white macules or papules with flat, fairly even borders surrounded by an intense erythematous halo. Each ulcer often is covered with a pseudomembrane. One or more ulcers can be present. They tend to recur and are usually very painful during their acute phase. The pain can interfere with eating, swallowing, and moving the tongue. Aphthous ulcers occur more frequently in women than in men.

Aphthae are found on oral mucosal surfaces that are not bound to underlying bone, especially the buccal and labial mucosa, dorsum of the tongue, floor of the mouth, soft palate, gingivae, lips, and oropharynx. The diagnosis depends mainly on exclusion of similar but more readily identifiable diseases, a history of recurrence, and inspection of the ulcer.

The etiology is still unclear because it has never been adequately demonstrated that this lesion is caused by a virus or any other specific chemical, physical, psychological, or hormonal cause.[112] Nuts, coffee, chocolate, and citrus fruits often cause flare-ups, but abstinence will not prevent recurrence. Trauma, nutritional deficiencies, stresses of various types, food components, and allergies have been shown to be contributory to the disease.[112,113]

Healing, which usually occurs in 1–3 weeks without scarring, can be accelerated slightly by treatment. A film-forming medication, hydroxypropyl cellulose (Zilactin), brings impressive pain relief and is able to protect the areas of ulceration from irritants, thus allowing patients to eat and drink more normally.[114] Bland antibiotic or anesthetic mouth rinses, topical steroid-antibiotic therapy, and surface protectants can also reduce pain. Sedatives, analgesics, and vitamins can help indirectly. Good oral hygiene and the minimization of mucosal trauma are helpful. Systemic antibiotics are contraindicated.

Ulcerative stomatitis is a general term for multiple ulcerations on an inflamed oral mucosa. It can be secondary to blood dyscrasias, erythema multiforme, bullous lichen planus, acute herpes simplex infection, pemphigoid, pemphigus, and drug reactions. If the lesions cannot be classified, they are referred to as *aphthae*.

Candidiasis (Moniliasis, Thrush)

Candidiasis is the most common opportunistic infection of the mouth, caused by overgrowth of a species of the fungus *Candida*. The species most frequently implicated in oral infections *is C. albicans*. The yeast phase of the fungus is a component of the normal oral flora of most people.[115] It exists in a symbiotic relationship with many of the other oral microorganisms. Because it has such low virulence in the yeast phase, some change must take place in the local environment to produce conditions favorable for its overgrowth and tissue invasion. The change commonly occurs when there is a reduction in host resistance caused by bacterial and viral infection, systemic disease, or medications.

Oral candidiasis generally presents in one of three distinct clinical forms: acute pseudomembranous candidiasis (thrush), acute atrophic candidiasis (antibiotic sore mouth), or chronic atrophic candidiasis (denture sore mouth). Rare forms include chronic hyperplastic candidiasis and chronic mucocutaneous candidiasis.

The lesions of acute pseudomembranous candidiasis consist of either multifocal or diffuse, white, superficial curdlike plaques occurring anywhere in the oral cavity. The infection is called pseudomembranous because the plaques can be scraped off easily, leaving an erythematous or bleeding base. Most other white mucosal lesions cannot be rubbed off.

Acute atrophic candidiasis often follows prolonged antibiotic or steroid therapy and results, clinically, in a painful erythematous mucosa, particularly involving the tongue. The problem usually resolves with cessation of the medications, but antifungal therapy will hasten recovery.

Chronic atrophic candidiasis presents as a slightly granular or irregularly eroded erythematous mucosa under dentures.

Any of these types of candidiasis can be accompanied by angular cheilitis. The diagnosis is based on the varied clinical picture of the surface white patches or erythematous changes and can be confirmed by laboratory culture. Treatment includes elimination of the causative or predisposing factor, if practical, and administration of antifungal agents.[116]

Leukoplakia (Benign Keratosis, White Patch)

The term *leukoplakia* is used to describe a thickened, white plaque that will not rub or strip off and is not

identifiable clinically or pathologically as any other disease. The lesions might be found on all oral mucous membrane surfaces, varying from a small circumscribed area to an extensive lesion involving a large area of mucosa. They are usually asymptomatic and are discovered on routine dental examination or by patients who feel the thickened plaques in their mouths. Leukoplakia occurs more often in men, and the highest incidence is in the fifth to seventh age decade.[117]

The most common cause of leukoplakia is epithelial hyperplasia, hyperkeratosis, hyperorthokeratosis, dyskeratosis, or acanthosis. These terms refer specifically to reactive conditions of the oral mucosal epithelium, usually in response to an irritant or chronic irritation. The specific etiology is often unknown, but there are risk factors: tobacco, alcohol, deficiency of vitamin A or B complexes, and chronic irritating conditions or habits.[118,119]

Treatment consists of removing all irritants. Failure of a keratotic lesion to regress within 2 weeks after elimination of the apparent cause should arouse suspicion, and the lesion should be biopsied or surgically excised. About 5% of patients with leukoplakia eventually develop squamous cell carcinoma in the area of the white lesion.

Mucositis (Stomatitis)

Mucositis and *stomatitis* are clinical terms describing inflammation, breakdown, and ulceration of the oral mucosal tissues. The disease can vary in its clinical presentation from focal or patchy erythema or ulceration to complete sloughing of the oral mucosa. Secondary hemorrhage is relatively common.

There is a wide variety of causative factors, including chronic mouth breathing, medications, systemic diseases, radiotherapy of the head and neck, and nutritional deficiencies. Patients complain of intense pain, burning, and dysphagia, which lead to an inability to eat or even drink.

Treatment relies heavily on palliation of symptoms, which can be provided by local anesthetic and antacid preparations used singly or in combination. Relief is short-lived, however, because the effect of these agents lasts less than 20 minutes. Benzydamine hydrochloride, a local anesthetic and anti-inflammatory drug, is effective for 1 to 2 hours. The use of any toothpastes or mouthwashes accentuates the problem because of their irritating and desiccating properties.

Radiation-induced mucositis initially appears as reddened and swollen mucosa, but the tissue becomes denuded and ulcerated as therapy continues. The patient experiences pain, burning, and discomfort that are greatly intensified by contact with coarse or highly seasoned foods. Involvement of the pharyngeal mucosa produces difficulties in swallowing and speaking. When therapy ends, spontaneous remission occurs in most patients within several weeks. In the meantime, the use of liquid topical anesthetics in the mouth before mealtimes frequently facilitates eating without discomfort.

Oral Cancer

Oral cancer is clearly a disease of older people: More than 98% of cases occur in persons older than 40 years;[120] the average age at the time of diagnosis is about 60 years.[121] The male-to-female ratio is approximately 2:1. It was estimated that in 1997 more than 31,000 new cases of cancer of the lips, tongue, floor of the mouth, palate, gingiva, buccal mucosa, and oropharynx would be diagnosed in the United States.[122] These oral cancers account for about 3.1% of all malignancies.

The most common type of oral cancer is squamous cell carcinoma, accounting for more than 90% of all oral malignancies.[123] The remaining 10% are predominantly malignant tumors of minor salivary gland tissue and (rarely) lymphomas, sarcomas, and melanomas.[120]

Clinically, an early cancer might appear as a small white patch (leukoplakia); a red velvety patch (erythroplakia); an aphthous-like, crusting, or traumatic ulcer; an erythematous plaque; a slightly raised lesion with central ulceration and a raised border; a verruciform growth; or a small swelling.[123] The most common signs and symptoms of oral cancer are listed in **Table 8-4**.

Frequently, it is impossible to differentiate between squamous cell carcinomas and the benign

Table 8-4 Oral Cancer Warning Signals

- Swelling
- Lumps, growths, exophytic masses
- White, scaly patches
- Red patches
- Oral ulcers (bleed easily, nonhealing)
- Atypical facial pain
- Persistent numbness or pain
- Persistent bleeding
- Difficulty in chewing
- Restricted jaw movement
- Trismus
- Restricted tongue movement
- Difficulty in swallowing
- Sore throat that does not heal
- Hoarseness
- Change in denture fit
- Loose teeth

non-neoplastic lesions seen in aphthous ulceration, herpes simplex infection, or traumatic ulceration. However, the non-neoplastic lesions are usually painful, and most early oral cancers are painless, becoming symptomatic only after they are large enough to impinge on the sensory nerves.[121] The cause of oral cancer is not known. A genetic factor is not apparent, but there is a definite increased risk with the use of tobacco and alcohol.[121,123,124] Independently, each agent is believed to be associated with an increased incidence of the disease, and the two together might, in fact, act synergistically.[121,123,124] Oral leukoplakia (benign keratosis) is an important precancerous lesion, turning into oral cancer in about 3% to 5% of cases.[125] Malignant transformations arising from the oral mucosa are mostly observed between ages 40 and 69 years. Although the peak prevalence is between ages 50 and 59 years, decline is gradual thereafter.[126] Other risk factors for oral cancer include exposure to sunlight, chronic trauma, diet, poor dentition, and history of syphilis infection.[121,123,127]

Oral cancer is a devastating disease with significant morbidity and mortality. Curative treatment consists of surgery, radiation, and chemotherapy, alone or in combination. Unfortunately, despite advances in these therapeutic approaches, only about 50% of patients with oral cancer survive the disease.[128] Of course, survival rates vary substantially depending on the site, ranging from 32% for cancer of the oropharynx to 91% for lip cancer, and on how early in the disease treatment is instituted. The poor overall prognosis for oral cancer results from the fact that the disease is often detected at advanced stages, after the visual detection of tissue changes or the development of symptoms.

Until the process of carcinogenesis is completely understood and true prevention is possible, early detection and treatment remain the best weapons against malignant disease. However, because there are no reliable methods for early diagnosis of squamous cell carcinoma, biopsy is the only definitive means of diagnosis. A biopsy should be done on any oral lesion that has not responded to therapy or resolved in a 2–week period.

Many of the predisposing factors for oral cancer are potentially avoidable. Preventive education should include delineation of the hazards of tobacco and alcohol and the need for regular dental care to reduce irritation and mechanical injury and for early detection.

Traumatic Ulcers

Acute trauma (mechanical, thermal, or chemical) is probably the most common cause of oral ulceration and is a frequent problem in the geriatric patient.[129] Ulcers can occur on any mucous membrane surface and are variable in size and shape. The ulcers are raised and have yellow-gray centers surrounded by an erythematous halo. Secondary infections with bacteria or *C. albicans* can occur.

The diagnosis is made primarily by history because most patients can identify the cause of the trauma. The patient typically complains of an isolated intraoral "sore" with pain or tenderness in the area of the lesion. Symptoms rarely exceed 3 or 4 days, and the lesion heals within 10 to 14 days.[130]

Chronic irritation from decayed or broken teeth and inadequate dentures can lead to chronic ulcers that persist indefinitely. Cheek and tongue biting produce a thin, rough, keratotic film in the area traumatized. Fragments of epithelium are often seen in these cases, as a result of the continuous chewing on the same area.

Treatment is instituted by avoiding the cause of the trauma and contact with any irritants. Dental care is usually necessary to relieve sources of irritation. Surgical repair of any extensive laceration can be necessary.

Denture-Related Oral Pathology

Dental prosthetic appliances are intended to restore the health and well-being of patients, but they are responsible for many of the most commonly occurring oral lesions among older adults. Removable appliances (complete or partial dentures) are implicated with greater frequency than are fixed appliances (bridges) because they can become distorted or broken with use and are frequently abused by the patient.[110] Older patients often do not or cannot comply with instructions for proper removal, placement, maintenance, and cleanliness of their appliances.

Because dentures fit next to teeth and soft tissue, they must be kept clean to maintain oral health. Plaque, food debris, and calculi collect on dentures just as they do on natural teeth. If left uncleaned, the dentures can be a source of irritation, inflammation, infection, or halitosis.

Partial dentures contribute to increased plaque formation around abutment teeth, which increases gingival inflammation over time.[131] Because these appliances can also increase tooth mobility and accelerate bone loss, the reduction of plaque becomes even more important.[131]

Frequently, patients perform their own repairs, relines, or adjustments, which can harm the dentures or oral mucosa. Additionally, all denture wearers should be advised that some servicing or readjustment of dentures is necessary occasionally because of normal changes in the supporting tissues and bone.

Common pathologic changes associated with denture wearing include the problems described in the following subsections.

Candidiasis

Denture-related candidiasis is by far the most common type of oral candidal infection.[132] The characteristic appearance is that of a slightly granular or irregularly eroded erythematous mucosa that corresponds exactly to the area covered by the upper denture. In some cases, the denture fits poorly and serves as a nutrient reservoir to foster fungal growth.[132] In patients with well-fitting dentures, the stability and peripheral seal of the upper denture allow the fungus to flourish in the absence of normal salivary flow. The condition seldom causes any discomfort.

These fungi are capable of growing on denture surfaces, from which they can infect and reinfect the soft tissues. Management therefore requires that both the denture and the mucosal surfaces be treated. Dentures should be kept scrupulously clean and soaked frequently in antifungal agents, germicides, or chlorhexidine.[132,133] The infected tissue is treated with topical antifungal agents.[116]

Denture Stomatitis (Denture Sore Mouth, Stomatitis Prosthetica)

The true etiology of denture stomatitis, a generalized inflammation associated with denture wearing, is unknown. It has been ascribed to contact hypersensitivity to dental materials, bacterial and candidal infections, tissue reaction to ill-fitting or unclean dentures, residual denture cleanser, medication use, and systemic diseases.[120,134] It is known to worsen in patients who do not remove their dentures at night or are negligent in denture hygiene.[120,132–134]

The disease is characterized by a very discrete erythematous reaction that closely follows the outline of the denture. Resolution of the inflammation can be obtained by thoroughly cleaning the denture if it fits well or by constructing a new one if it is ill-fitting. If candidal organisms are a contributing factor to the stomatitis, the denture can be covered with an antifungal ointment before insertion or soaked in an antifungal solution at night.

Traumatic Ulcers (Denture Sore Spots)

An unstable or unretentive denture will cause tissue irritation or ulceration because of excess movement. Overextended denture flanges, bone spicules under the dentures, and foods—especially seeds trapped between the denture and mucosa—can also cause ulcerative lesions. The ulcers are small, painful, irregularly shaped lesions usually covered by a necrotic membrane and surrounded by an inflammatory halo. Treatment consists of correcting the underlying cause. Most lesions usually heal promptly.

Periodontium

Periodontal Disease

The most common disease of the periodontium is *periodontal disease*, a term generally used to describe specific chronic disorders that affect the gingiva, supporting connective tissue, and alveolar bone.[135] It is a chronic, progressive, and destructive condition, and its incidence and severity typically increase with age.[136–138] There is considerable question, however, about whether the increase in severity with age represents age-dependent pathology or the cumulative effects of a lifetime of intermittent destruction.[1]

Periodontal disease commonly develops in two stages, gingivitis and periodontitis. As with dental caries, the major etiologic factor is plaque, which accumulates more rapidly and heavily in elderly people.[136] If the plaque is not removed daily, it will calcify into calculus (tartar). Accumulation of plaque, food, bacteria, and calculi on the tooth surfaces between the tooth and gingiva produces a low-grade inflammation of the gingiva (gingivitis). This is clinically characterized by gingival redness, enlargement, tenderness, and bleeding. Although gingivitis develops more rapidly and with greater severity in older adults, it is reversible with adequate plaque control.[138]

If the inflammatory process is allowed to progress, there is formation of pus (pyorrhea) with or without discomfort or other symptoms. Without drainage, the accumulation of pus leads to acute swelling (periodontal abscess) and pain. When the inflammation extends to the underlying alveolar bone and connective tissue (periodontitis), it loosens the teeth and causes them to be extruded. However, once the teeth are lost, the inflammatory symptoms subside.

The diagnosis depends on a combination of findings, including localized pain, loose teeth, the presence of periodontal pockets, erythema, and swelling or suppuration. A severe case results in a foul odor, inflamed and ulcerated gums, fibrotic tissue, and bleeding. Roentgenograms can reveal the destruction of alveolar bone. Margins of overextended fillings often play a role as local irritating factors. Occlusal trauma, particularly from grinding and teeth-clenching habits, and systemic factors can contribute to periodontal disease, but they do not initiate the disease.[136,138]

The prevention of periodontal disease depends largely on plaque control through meticulous oral hygiene[136,138] Although there are indications that periodontal breakdown progresses slowly in elderly persons, progression of the disease can be retarded by oral hygiene and use of antimicrobial agents.[130,135,138,139] Local drainage and oxygenating mouth rinses (3% hydrogen peroxide in an equal volume of water) will usually reverse any acute symptoms and allow for routine follow-up procedures. In some cases, surgery to reduce excess gum tissue helps prevent the formation of periodontal pockets that predispose to periodontal infections. In advanced disease, extraction of teeth might be necessary.

It should be noted that periodontal disease has been linked as a risk factor to several age-related diseases, such as poor glycemic control in type 2 diabetes mellitus, metabolic syndromes, and cardiovascular diseases, including myocardial infarction and stroke. Current evidence has shown that chronic periodontal infection/inflammation might modulate systemic inflammatory mediators (cytokines) and metabolic biomarkers such as C-reactive protein, von Willebrand factor, and IL-1β that have been associated with endothelial dysfunction, atherosclerosis, diabetes, and other metabolic disorders.[140–143] The pathophysiology of periodontal diseases in cardiovascular and metabolic syndrome development still need further elucidation. However, the treatment of periodontal diseases can improve the inflammatory biomarkers and glucose level in blood samples of these patients.[140,142] The control of periodontal diseases could play a critical role for prevention and treatment of metabolic and cardiovascular diseases in the dentate population.

Necrotizing Ulcerative Gingivitis (Vincent's Infection, Trench Mouth)

Necrotizing ulcerative gingivitis is an acute, recurring, noncommunicable inflammatory disease of the gingiva resulting from local irritation and organisms in the normal oral flora that invade the gingival tissue when its resistance is lowered. The disease is characterized by redness, swelling, ulceration, bleeding, and pain. The yellowish-gray pseudomembrane that usually covers the ulcerated surface can be removed easily, leaving a raw, bleeding base. In severe cases, there is a fetid odor and foul taste in the mouth. Recurrent attacks can lead to bone loss. Treatment includes eliminating local irritants by careful and thorough oral hygiene procedures. Local anesthetics, as well as antibiotic therapy, can serve as adjuncts to treatment. Caustics are contraindicated.

Tongue

Fissured Tongue

The prevalence of fissured tongue, characterized by cracks on the dorsolateral surfaces of the tongue, increases progressively in each decade of adult life. Fissured tongue is found in varying degrees in approximately 5% of the population. It occurs in 60% of persons after age 40 years.

The fissures are deep, tend to collect food debris and microorganisms, and cause the tongue to be inflamed often. However, the tongue is usually pain free or only mildly tender, even if the fissures become secondarily infected from retained debris and microbes. Fissuring of the tongue sometimes is associated with deficiency of vitamin B complex, or it can be genetic. Another cause is correlation with long-standing glossitis. Treatment consists of brushing the tongue or rubbing it vigorously with a moistened washcloth to provide relief. The scarring is irreversible.

Glossitis

Inflammation of the tongue, usually manifested by considerable atrophy of the filiform papillae, creates a red, smooth appearance. It can be secondary to a variety of diseases, such as anemia, nutritional deficiency, drug reactions, systemic infection, and physical or chemical irritations. The diagnosis is usually based on the history and laboratory studies, including cultures as indicated.

Treatment is based on identifying and correcting the primary cause, if possible, and palliating the tongue symptoms as required. When the cause cannot be determined and there are no symptoms, therapy is not indicated.

Glossodynia, Glossopyrosis (Burning Tongue, Chronic Lingual Papillitis)

Glossodynia, or painful, burning, itching, stinging tongue, is a distressing symptom that predominantly affects older women.[144] It can accompany atrophy of the tongue papillae and is a prominent feature of the "burning mouth" syndrome. Involvement of the entire tongue or isolated areas, occurring with or without glossitis, can be the presenting symptoms of hypochromic or pernicious anemia, nutritional disturbances, emotional upset, hormonal imbalance, allergies, psychosomatic syndromes (grief, loneliness, despair), or other systemic disorders.[145] Smoking, xerostomia, medication use, and candidiasis can also be causative.

In most cases, a primary cause cannot be identified. Cultures are of no value because the offending

organisms usually are also present in the normal oral flora. Dental prostheses, caries, and periodontal disease are usually of no causative significance. Although certain foods can cause flare-ups, they are not the primary causes. Dentifrice ingredients are rare causes of burning and pain of the tongue.

Treatment is mainly empiric because causative factors usually are not identified. Important approaches include ruling out systemic conditions associated with these symptoms, changing the individual's drug regimen, and reassuring the patient that there is no evidence of infection or neoplasia. Ointments and mouth rinses are of no value.

Hairy Tongue

Hairy tongue is characterized by elongated, thick, densely matted, and stained filaments on the dorsum of the tongue that resemble hair. The filaments are hypertrophied or hyperplastic filiform papillae that can be stained yellow, brown, or black.

Normally, the developing papillary tissue cells slide off the tongue during mechanical stimulation. When desquamation is diminished, the papillae become elongated and provide a nidus for *materia alba* to accumulate, for stains to collect, and for bacteria and fungi to lodge and produce minor infections.[119] Common causes of staining are coffee, tobacco, medications, foods, and chromogenic microorganisms.

Hairy tongue is not a serious condition and is easily eliminated by improving tongue hygiene and promoting desquamation. If candidal organisms are present, the use of antifungal agents is indicated.

Macroglossia

Macroglossia (large tongue) can be congenital or acquired. It is significant in the elderly population because individuals who have been edentulous for many years might develop this condition. The marked use of the tongue to aid in mastication of food results in muscular hypertrophy, a common type of acquired macroglossia. If the patient is able to wear dentures, the tongue muscle might regress, with reduction in the size of the tongue.

CIRCUMORAL TISSUES

Lips

Angular Cheilitis (Cheilosis, Pseudocheilosis, or Perleche)

Angular cheilitis is a nonspecific inflammation at the oral commissure area bilaterally. It proceeds to a cracking of the angles of the mouth, with well-defined fissures present. The drooling of saliva often aggravates the condition.

The etiology of angular cheilosis is often complex. The combination of bone resorption, muscle atrophy, and tooth loss decreases the distance between the nose and chin, which causes the skin to wrinkle and fold around the mouth. The wrinkling can also accompany a change of bite with old, ill-fitting, or even new dentures. The wrinkled folds collect saliva, *C. albicans,* bacteria, and other contaminants that can cause the infection. Contributing factors include vitamin B complex deficiency, iron deficiency, or both.[3,24,29]

Treatment is directed toward unfolding the skin by the fitting of proper dentures; culturing and treating all infections; initiating measures of local hygiene; and, if necessary, giving iron and vitamin supplements.

Squamous Cell Carcinomas

A high risk of lip cancer is associated with the use of tobacco, particularly pipe tobacco, and exposure to ultraviolet radiation. Almost 95% of these cancers develop in the lower lip, where trauma and heat from the pipestem and exposure to the sun are greatest.[146] Atrophy of the lip, thinning of the lip border, and loss of elasticity are early clinical features. Carcinoma of the lip can appear as a crack in the lip surface, a crusting ulcer, or a tumorous growth. The prognosis is very good unless the lesion is extensive because metastases develop later and less frequently than from intraoral sites.[120,121]

Oral Musculature

Dysphagia

Dysphagia (difficulty in swallowing) can render a patient vulnerable to aspiration of saliva or oral intake. Indicators that a swallowing problem is likely to be present include dysarthria, poor control of oral secretions, inability to swallow spontaneously, drooling or gurgling aspirations or regurgitation through the nose, and frequent reflexive coughing.[147,148]

The causes of dysphagia can be neurologic, neuromuscular, or structural. In one study, a significant increase in swallowing dysfunction was seen among older persons taking prescription medications.[149] Conditions that alter the ability to form and prepare a bolus in the oral stage of swallowing can also cause dysphagia.[10]

Dyskinesia

Oral dyskinesia is a movement disorder characterized by severe, dystonic, involuntary movement of the facial,

oral, and cervical musculature.[150] The involuntary abnormal contractions, mainly of the tongue, lips, and mandible, occur frequently with age in patients who exhibit disturbances of the cerebral stroma or stromal changes of the extrapyramidal motor system.[150–152] Because the movements disappear when the mouth is opened wide, during sleep, or when the patient's attention is distracted, oral dyskinesia tends to be regarded as a disease of the central nervous system.[150]

Some studies have reported a close correlation between oral dyskinesia and poor oral conditions.[150,151] One study describing the clinical appearance of this disease in older adults found that its occurrence was associated with missing teeth and use of uncomfortable dentures.[150] It has also been demonstrated that the symptoms of oral dyskinesia respond favorably to dental treatment, such as extractions, new dentures, and adjustment of old dentures.[150,151]

Drug-induced oral dyskinesia, or tardive dyskinesia, is a permanent side effect of long-term neuroleptic (antipsychotic) drug therapy that does not resolve on withdrawal of the drug. The most common movements are tongue protrusion, licking and smacking of the lips, puffing of the cheeks, sucking and chewing, and facial grimacing.[153,154]

Trismus

Trismus is a condition in which tonic spasms of the masticatory muscles limit opening of the mouth. It can develop during or after radiation therapy if these muscles are included in the treatment field. Management is directed toward exercises and various prosthetic appliances to increase the opening capacity of the muscles.

OTHER CHANGES

Salivary Glands

Sialolithiasis

Sialoliths, or salivary stones, can form in any of the major or minor salivary glands or their excretory ducts. The most common manifestation of ductal stones, which do not generally cause complete obstruction, is enlargement of the gland and subsequent pain during eating. Both the glandular swelling and pain subside between meals as the entrapped saliva is gradually excreted.

Tumors

Benign and malignant tumors of the salivary glands are more common in older patients, and both the major and minor salivary glands are involved.[155] Overall, neoplasms arising from the minor salivary glands are relatively uncommon compared with those arising from the major salivary glands.[121,155] However, most tumors of the minor salivary glands are malignant.[155]

Both elderly men and elderly women appear to have an increased risk of salivary gland malignancies; however, little information about the causative factors exists. Radiotherapy is an infrequent etiologic factor.[156] Trauma, infection, stone formation, and the use of alcohol or tobacco are not associated with these tumors.[157]

The survival rates for patients with malignant salivary gland tumors are generally higher than they are for persons with most other oral cancers. Usually, diagnosis and treatment are rendered early, and metastasis occurs late in the course of the disease. Long-term follow-up is essential, however, because there is a high rate of recurrence.

Xerostomia (Dry Mouth, Decreased Salivary Flow)

Xerostomia, although not a disease concomitant with aging, is a symptom that is often evident in older patients. The main cause is use of xerogenic medications; other causes include vitamin deficiencies, dehydration, mouth breathing, stress, and a variety of systemic diseases and their therapies.

Without the antibacterial, cleansing, lubricating, remineralizing, and buffering actions of saliva, the individual with xerostomia is at increased risk of developing coronal and root surface caries; abrasion and erosion of tooth surfaces; periodontal disease; atrophic glossitis; traumatic injuries to the mucous membranes; mucosal lesions; infections of the pharynx and salivary glands; and dysfunctions of speech, chewing, swallowing, and taste.[24,158] In addition to the damage to teeth and supporting structures, problems with prostheses are also magnified when the mouth is dry. Saliva provides a thin, fluid film between the denture base and underlying soft tissues necessary for the retention and stability of dentures during function.[42,159] Additionally, saliva prevents the hard acrylic or metal surfaces from abrading the oral mucosa. Consequently, frequent denture problems and sores arise, and the patient complains of generalized intraoral soreness during mastication.

Patients might express one or all of the signs and symptoms associated with xerostomia in varying degrees of severity. Some of these include mouth dryness; a fissured tongue; glossodynia or glossopyrosis; candidiasis; rampant caries; oral soreness; sticking of food or lips to the teeth; cracking of lips; difficulty in speaking, chewing, and swallowing; a generalized

burning sensation; and ageusia, dysgeusia, or hypogeusia.[42,159] The mucosa becomes dry, rough, and sticky; bleeds easily; and is subject to ulceration or infection.

Prevention and management of xerostomia depends on its etiology (**Table 8-5**). With drug-induced xerostomia, the responsible drug might be able to be eliminated, reduced in dosage or frequency of administration, or replaced by a substitute drug. Management of xerostomia that is irreversible, such as radiation-induced xerostomia, is essentially palliative and accommodative. Small, frequent mouthfuls of water are palatable and inexpensive and moisten the mouth fairly well. To facilitate chewing and swallowing, most patients with xerostomia moisten and thin foods with sauces, gravies, milk, and other fluids.

Artificial saliva preparations provide relief by coating and lubricating the mucosa. Saliva substitutes containing fluoride and fluoride gels are helpful for patients with xerostomia who are at high risk for dental caries. For the lips and dentures, a constant coating of petroleum or water-based jelly and frequent oral application of artificial salivas should alleviate some of the problems. However, lemon glycerine swabs should be avoided because of their cariogenic and drying effects. Also, commercial mouthwashes should be avoided because they have a high alcohol content and dry the oral mucosa. Similarly, ingestion of alcoholic beverages should be minimized.[160]

Table 8-5 Prevention and Management of Xerostomia

Determine etiology
Alter medication regimen
Eliminate medication
Reduce dosage or frequency of administration
Replace with medication for another with less severe oral side effects
Alleviate complaints
Increase fluid intake (water or low-sugar beverages); avoid caffeinated drinks
Avoid dry, bulky, spicy, salty, or highly acidic foods
Avoid tobacco and alcohol intake
Humidify air
Use saliva stimulants (local and systemic agents)
Local agents
Sugarless hard candy or lozenges
Sugarless gum
Systemic agents
Pilocarpine drops
Oral pilocarpine (2.5–5.0 mg three times daily before meals)
Use artificial saliva preparations (containing fluoride)
Glycerine
Methylcellulose
Coat lips and dentures with petroleum jelly
Increase resistance to dental disease
Have frequent dental examinations
Use fluorides frequently
Modify diet
Control plaque

Sense of Taste

Taste acuity can be affected by oral pathologic conditions, dental diseases, olfactory deficits, medications, malnutrition, smoking, radiation therapy, neurologic deficits, and other systemic disorders.[5] Cues that can indicate alterations in taste sensitivity include decreased or increased appetite, excessive use of seasoning, and excessive use of sugar.

SYSTEMIC DISEASES AND MEDICATIONS AFFECTING ORAL HEALTH

The elderly population suffers from many concurrent acute and chronic diseases, some of which can have oral manifestations or adversely affect oral health. Because 86% of all elderly persons suffer from at least one chronic disease, oral health problems secondary to these diseases can be important.[161] Systemic diseases that affect the oral and circumoral structures are listed in **Table 8-6**.

Many of the most commonly experienced chronic disease conditions found in elderly individuals are symptomatically controlled with the proper use of medications. The increased use of medications with advancing age, therefore, is not surprising. Geriatric patients take more drugs because they have more chronic illnesses than do younger patients. Not only does the problem of multiple drug use among elderly people have serious implications related to pharmacokinetic and pharmacodynamic considerations with aging, but also many drugs have iatrogenic oral manifestations (**Table 8-7**).

In addition to the oral signs and symptoms from elderly people's use of properly prescribed and over-the-counter medications, recreational drug abuse has oral manifestations, including advanced

Table 8-6 Oral Manifestations of Systemic Diseases

Disease/Condition	Oral Manifestations
Achlorhydria	Tongue—glossitis
Adrenal insufficiency	Oral infections—increased risk Oral mucosa—pigmentation Taste—loss or distortion Wound-healing response—poor
Agranulocytosis	Gingiva—spontaneous bleeding Hemorrhagic tendency—petechiae Periodontal disease—high incidence Ulcerations—painful, persistent, necrotic
Alcoholism	Breath odor of alcohol Dental caries—high incidence Facial neuralgia, edema Hemorrhagic tendency—ecchymoses, petechiae Lips—angular cheilosis Oral cancer—increased risk Oral hygiene—poor
Alcoholism	Oral infections—increased risk Oral mucosa—jaundiced, ulcerated Parotid salivary glands—chronic swelling Periodontal disease—chronic (with frequent acute exacerbations) Taste—decreased sensitivity Teeth—attrition, erosion, loss Tongue—glossitis, ulcerated Wound-healing response—delayed Xerostomia
Alzheimer's disease	Dysphagia Oral hygiene—poor, neglected Taste sensitivity—decreased Xerostomia
Amyotrophic lateral sclerosis	Dysarthria Tongue fasciculations—atrophic
Anemia	Burning/sore mouth—mucositis/stomatitis Filiform papillae—atrophic Oral mucosa—pale, atrophic, thin, tender Tongue—glossitis, glossodynia Xerostomia
Anxiety disorders	Burning/sore mouth Dysphagia Xerostomia
Arthritis	TMJ involvement—limited jaw movement Biliary tract obstruction Bleeding—excessive, spontaneous Hemorrhagic tendency—petechiae, hematomas
Bipolar disorders	Depressive phase—oral hygiene neglected Facial pain syndromes due to mood swings Manic phase—self-inflicted mucosal abrasion
Bleeding disorders	Intraoral bleeding—ecchymoses, hematomas, petechiae Oral mucosa—jaundiced
Cerebrovascular accident	Chewing difficulty/inability Dysarthria Dysphagia

(*continues*)

Table 8-6 Oral Manifestations of Systemic Diseases (continued)

Disease/Condition	Oral Manifestations
	Facial drooping—affects denture fit Gag reflex—decreased Oral motor apraxia Oral sensation—decreased unilaterally
Chorea	Dysarthria Oral dyskinesia
Congenital heart disease	Cyanosis Intraoral hemorrhages, infections Leukopenia, polycythemia, thrombocytopenia
Congestive heart failure	Intraoral bleeding—ecchymoses, petechiae Lips—cyanosis, thinning of vermilion border Oral infections
Coronary arteriosclerotic heart disease	Oral or facial pain—referred
Crohn's disease	Aphthous ulcers—high frequency Burning/sore mouth Dental caries—high frequency Oral hygiene—poor
Cyclic neutropenia	Mucositis/stomatitis Oral infections—increased risk Periodontal disease—high incidence Ulcerations—aphthous type
Dementia	Bruxism Burning/sore mouth Dysphagia Facial pain—atypical Oral injuries—increased susceptibility Periodontal disease—accelerated Poor oral hygiene—chronic
Depression	Burning/sore mouth Dental caries—rapid progression Facial pain syndromes—numerous Oral hygiene—poor Periodontal disease—accelerated Tongue—glossodynia Xerostomia
Diabetes mellitus	Breath odor of ketone Burning/sore mouth—mucositis/stomatitis Candidiasis Dental caries—rampant Gingiva—inflammation Lips—angular cheilitis Mucomycosis Oral infections—increased susceptibility Oral paresthesias Periodontal disease—accentuated, abscesses Taste sensitivity—decreased Teeth—sensitivity Tongue—glossodynia Ulcerations Wound-healing response—delayed Xerostomia
Epilepsy	Gingiva—drug-induced hyperplasia Ulcerations—traumatic

(*continues*)

Table 8-6 Oral Manifestations of Systemic Diseases (continued)

Disease/Condition	Oral Manifestations
Gonorrhea	Gingivitis Oral abscesses/mucosal lesions/ulcerations Oral mucosa—erythematous Parotitis Pharyngitis/tonsillitis Stomatitis—generalized
Hepatitis	Intraoral bleeding Oral mucosa—pigmentation Taste—loss or distortion
Herpes zoster	Bone—osteoradionecrosis Neuralgia—trigeminal Oral mucosa—lesions, ulcerations, pain Teeth—devitalization, exfoliation
Hypertension	Neuritis
Hyperthyroidism	Dental caries—extensive Periodontal disease—progressive Tongue—tumors (midline of posterior dorsum)
Hypoparathyroidism	Candidiasis
Hypothyroidism	Candidiasis Taste—loss or distortion Teeth—malocclusion Tongue—macroglossia Xerostomia
Immunosuppression	Increased susceptibility to candidiasis; dental caries; infections; local and systemic; intraoral bleeding; periodontal disease; recurrent aphthous ulcers; tumor development
Leukemia	Bone—lesions Burning/sore mouth—mucositis/stomatitis Candidiasis Gingiva—hyperplasia, spontaneous bleeding Hemorrhagic tendency—ecchymoses, hematomas, petechiae Herpetic stomatitis Infections—increased risk Lymphadenopathy Oral mucosa—pallor, lesions Oral paresthesias Ulcerations—painful, persistent, necrotic
Leukopenia	Oral infections—increased risk
Liver disease	Bleeding—excessive, spontaneous Hemorrhagic tendency—ecchymoses, hematomas, petechiae
Lupus erythematosus	Burning/sore mouth Candidiasis
Lupus erythematosus	Mandible immobility Oral lesions—bullae, erosions Oral mucosa—sloughing TMJ deviation, pain with movement or palpation, joint sounds, locking or dislocation Tongue fissuring, atrophic papillae Ulcerations Xerostomia

(*continues*)

Table 8-6 Oral Manifestations of Systemic Diseases (continued)

Disease/Condition	Oral Manifestations
Lymphomas	Burning/sore mouth Candidiasis Cervical lymphadenopathy Extranodal oral tumors Hemorrhagic tendency—ecchymoses, petechiae Infections—increased risk
Malabsorption syndrome	Bleeding—excessive, spontaneous Candidiasis Hemorrhagic tendency—ecchymoses, hematomas, petechiae
Malignant hypertension	Facial paralysis
Multiple myeloma	Amyloid deposits in soft tissue Bone—lesions, pain Soft tissues—tumors Teeth—unexplained mobility
Multiple sclerosis	Dysarthria TMJ—pain with movement or palpation, joint sounds Trigeminal neuralgia Xerostomia—drug-induced
Muscular dystrophy	Mouth breathing Muscles—weakness, decreased biting force Tongue—hypertrophy
Myasthenia gravis	Chewing difficulty Dysphagia Gingiva—poor health Mouth breathing Muscles—weakness, inability to close mouth Tongue—flaccid
Narcolepsy	Candidiasis Xerostomia
Nephritis	Burning/sore mouth Xerostomia
Neurofibromatosis	Oral neurofibromatous lesions Oral paresthesias Soft tissues—pigmentation Tongue—macroglossia, enlarged lingual papillae
Organ transplants	Intraoral bleeding—increased susceptibility Oral infections—increased susceptibility Tumor development—increased susceptibility
Osteoarthritis	Bone—resorption TMJ—unilateral involvement, dysfunction
Paget's disease	Bone—progressive enlargement
Parkinson's disease	Chewing difficulty Drooling of saliva due to swallowing difficulty (not excessive production) Dysarthria Dysphagia Facial paresthesias, tremors Lips—angular cheilitis, tremors

(*continues*)

Table 8-6 Oral Manifestations of Systemic Diseases (continued)

Disease/Condition	Oral Manifestations
	Oral hygiene—poor Oral mucositis—stomatitis Tardive dyskinesia—drug-induced Teeth—involuntary bruxism Tongue—tremors Xerostomia—drug-induced
Pemphigus vulgaris	Burning/sore mouth Candidiasis Halitosis Hypersalivation Oral lesions, erosions—bleed easily, painful Ulcerations—raw, red, eroded
Pneumonia	Aspiration—increased susceptibility with dysphagia, poor dentition, poor oral hygiene
Polycythemia	Oral mucosa—cyanosis
Posttraumatic stress disorder	Bruxism Dental caries—increased incidence Oral hygiene—poor Periodontal disease—increased incidence Tongue—glossodynia
Progressive bulbar palsy	Chewing difficulty Dysarthria Jaw muscles—spastic
Radiation therapy to head/neck	Candidiasis Mucositis Muscles—dysfunction, trismus Oral infections—increased susceptibility Pulp—pain, necrosis Taste—lost or distorted Teeth—hypersensitivity, radiation caries Xerostomia
Renal disease/dialysis/transplants	Breath odor of urea Calculus—increased formation Candidiasis Dental caries—low incidence Gingiva—pale, undefined, bleeds spontaneously Oral infections—frequent retrograde infectious parotitis Oral mucosa—pallor, uremic stomatitis Renal osteomalacia/osteodystrophy Salivary flow—decreased Taste—metallic Teeth—mobility Tongue—macroglossia, glossodynia Ulcerations—ulcerative stomatitis Wound-healing response—poor
Rheumatoid arthritis	Bone—resorption Muscles—atrophic TMJ—dysfunction Xerostomia

(*continues*)

Table 8-6 Oral Manifestations of Systemic Diseases (continued)

Disease/Condition	Oral Manifestations
Sjögren's syndrome	Burning/sore mouth—mucositis/stomatitis Candidiasis Dental caries—increased susceptibility Lips—angular cheilosis, lesions Oral mucosa—lesions Parotid gland—enlargement Periodontal disease—accelerated Saliva—composition changes; increased sodium, potassium, manganese; decreased calcium Taste—loss, distortion Tongue—glossitis, glossodynia Xerostomia
Smokeless tobacco use	Gingiva—recession Halitosis Oral cancer—increased risk Oral mucosa—leukoplakia Periodontal disease—accentuated Smell sensitivity—decreased Taste sensitivity—decreased Teeth—abrasion; attrition, erosion, loss
Syphilis	Oral lesions—chancre, mucous patch, gums Tongue—interstitial glossitis
Temporal arteritis	Orofacial pain
Thrombocytopenia	Hemorrhagic tendency—ecchymoses, hematomas, petechiae
Tobacco smoking	Calculus—increased Gingivitis—increased Hairy tongue
Tobacco smoking	Halitosis Smell sensitivity—decreased Taste—loss or distortion Teeth—abrasion Wound-healing response—delayed
Tuberculosis	Lymph node involvement (scrofula) Ulcerations, especially on tongue
Urticaria (angioneurotic anemia)	Swelling—soft tissues
von Willebrand's disease	Hemorrhagic tendency—ecchymoses, hematomas, petechiae Intraoral bleeding—spontaneous

generalized periodontal disease, bruxism, numerous abscessed or missing teeth, poor oral hygiene, rampant caries, tooth attrition (secondary to bruxism), and xerostomia.[162–164]

IMPACT OF NUTRITIONAL STATUS ON ORAL HEALTH

Nutritional status has an important role in oral health. A sophisticated system of nutrient interaction is essential to the formation of healthy teeth and the maintenance of oral and circumoral tissues throughout life.[165–167] The systemic effects of nutrients on oral health, growth and development, cell integrity and renewal, proper function of the tissues and saliva, tissue repair, and resistance and susceptibility to oral diseases (**Table 8-8**) have been studied by very few researchers and need more attention and understanding. The local effects of food on plaque formation and the resultant oral disease processes, including coronal and root caries, gingivitis, and periodontitis, have been relatively well described.

Table 8-7 Drug-Induced Oral Manifestations

Candidiasis
- Antibiotics
- Antineoplastics
- Corticosteroids
- Diuretics
- Immunosuppressives
- Steroid inhalers

Contact hypersensitivity
- Iodine
- Menthol
- Topical analgesics
- Topical antibiotics

Erythema multiforme
- Anticonvulsants
- Antimalarials
- Barbiturates
- Busulfan
- Chlorpropamide
- Clindamycin
- Codeine
- Isoniazid
- Meprobamate
- Minoxidil
- Penicillins
- Phenolphthalein
- Phenylbutazone
- Propylthiouracil
- Salicylates
- Sulfonamides
- Tetracyclines

Fixed drug eruptions
- Barbiturates
- Chlordiazepoxide
- Sulfonamides
- Tetracyclines

Gingival hyperplasia
- Cyclosporine
- Nifedipine
- Phenytoin sodium

Glossodynia
- Diuretics

Hairy tongue
- Antibiotics
- Corticosteroids
- Sodium perborate
- Sodium peroxide

Hypersalivation/sialorrhea
- Antianxiety agents
- Anticholinesterases
- Apomorphine
- Iodides
- Lithium
- Mercurial salts
- Nitrazepam

Infections
- Antineoplastics
- Corticosteroids (high dose)
- Immunosuppressives

Intraoral bleeding/petechiae/purpura
- Antiarrhythmics
- Antibiotics (broad spectrum)
- Anticoagulants
- Aspirin
- Warfarin sodium

Lichenoid mucosal reactions
- Allopurinol
- Antihypertensives
- Beta blockers
- Chloroquine
- Chlorothiazide
- Chlorpropamide
- Dapsone
- Diuretics
- Doxorubicin hydrochloride
- Gold salts
- Mercurial diuretics
- Mercury compounds
- Minocycline
- Nonsteroidal anti-inflammatory agents
- Penicillamine
- Phenolphthalein
- Phenothiazines
- Phenytoin
- Quinidine
- Silver compounds
- Streptomycin
- Sulfamethoxazole
- Tetracyclines
- Tolbutamide

Lupus erythematosus (oral mucosa) reactions
- Gold salts
- Griseofulvin
- Hydralazine hydrochloride
- Isoniazid
- Methyldopa
- Penicillin
- Phenytoin
- Primidone
- Procainamide
- Streptomycin
- Sulfonamides
- Tetracyclines
- Thiouracil

Mucositis/stomatitis
- Antineoplastics
- Lithium
- Mercurial diuretics

Oral dyskinesias
- Buspirone

Orofacial neuropathies (numbness, tingling, burning of the face or mouth)
- Acetazolamide
- Antineoplastics
- Beta blockers
- Chlorpropamide
- Ergotamine
- Hydralazine hydrochloride
- Hypoglycemics (oral)
- Isoniazid
- Methysergide
- Nalidixic acid
- Nitrofurantoin
- Phenytoin
- Streptomycin
- Tolbutamide
- Tricyclic antidepressants

Pigmentation (soft tissue)
- Antimalarials
- Busulfan
- Chlorhexidine

Salivary gland enlargement
- Antipsychotics
- Insulin
- Iodides
- Isoproterenol
- Methyldopa
- Phenylbutazone
- Potassium chloride
- Thiocyanate
- Thiouracil
- Warfarin sodium

Salivary gland pain and/or swelling
- Antihypertensives
- Antithyroid agents
- Cytotoxic agents
- Ganglion-blocking agents
- Insulin
- Iodine
- Isoproterenol
- Oxyphenbutazone
- Phenothiazines
- Phenylbutazone
- Potassium chlorate
- Sulfonamides
- Warfarin sodium

(*continues*)

Table 8-7 Drug-Induced Oral Manifestations (continued)

Spontaneous oral bleeding
- Anticoagulants
- Antineoplastics

Tardive dyskinesias
- Butyrophenone antipsychotics
- Levodopa
- Phenothiazines
- Thioxanthene

Taste dysfunction
- Amphetamines
- Benzodiazepines
- Carbimazole
- Chlorhexidine
- Chlorpromazine
- Clofibrate
- Ethionamide
- Gold salts
- Griseofulvin
- Levodopa
- Lincomycin
- Lithium carbonate
- Methocarbamol
- Metronidazole
- D Penicillamine
- Penicillin
- Phenformin hydrochloride
- Phenindione
- Propranolol
- Quinidine
- Tranquilizers
- Vitamins (excessive use)

Tooth decay (rampant)
- Tricyclic antidepressants

Tooth discoloration
- Chlorhexidine
- Gentian violet
- Stannous fluoride
- Tetracyclines

Ulcerations
- Antiarrhythmics
- Antineoplastics
- Aspirin
- Gold salts
- Indomethacin
- Meprobamate
- Mercurial diuretics
- Methotrexate
- Methyldopa
- Naproxen
- D Penicillamine
- Phenylbutazone
- Potassium chloride
- Propranolol
- Spironolactone
- Thiazide diuretics
- Tolbutamide

Xerostomia
- Amphetamines
- Analgesics
- Anorexiants
- Antiallergics
- Antianxiety agents
- Antiarrhythmics
- Anticholinergics
- Anticonvulsants
- Antidepressants
- Antidiarrheals
- Antihistamines
- Antihypertensives
- Anti-inflammatory agents
- Antinauseants
- Antineoplastics
- Anti—parkinsonism agents
- Antipsychotics
- Antispasmodics
- Atropine
- Barbiturates
- Benzodiazepines
- Bronchodilators
- Central nervous system stimulants
- Congestive heart failure medications
- Decongestants
- Diuretics
- Ganglion-blocking agents
- Hypnotics
- Lithium
- Monoamine oxidase inhibitors
- Muscle relaxants
- Narcotics
- Nonsteroida! anti-inflammatory agents
- Phenylbutazone
- Scopolamine
- Sympathomimetics
- Tranquilizers

PLAQUE FORMATION

Plaque consists mainly of bacteria and a matrix produced by them that is composed primarily of carbohydrate, protein, salts, and water. From a dietary standpoint, carbohydrates have an important role in initiating plaque formation. Once plaque is present, carbohydrates from food and beverages can diffuse into it and be fermented by the plaque bacteria. The acid produced can dissolve tooth structure, thus leading to carious lesions. Although acids present in food and beverages can also diffuse into the plaque, the result is usually erosion of the tooth surface and not dental caries.

If fermentable carbohydrates are not part of the diet, the acid-producing activity of the plaque will be low. Plaque is still demonstrable in subjects eating a diet devoid of fermentable carbohydrates, but the plaque is thin and structureless.[168] In contrast, subjects eating a sucrose-rich diet have voluminous, turgid plaque formation.

The texture of the diet can also influence dental plaque. Diets containing soft foods increase plaque formation more than those composed of firmer foods. In a study of women on a low-calorie diet, the rate of plaque formation increased.[169]

Plaque initially forms along the tooth–gum margin and gradually spreads across the tooth surface as the bacterial matrix grows. Because dietary carbohydrates contribute to this supragingival plaque formation, they have been implicated as an etiologic agent in the resulting gingival inflammation.[68,170]

DENTAL CARIES

Although dental caries is generally accepted as primarily a microbial disease, diet plays a crucial

Table 8-8 Systemic Effects of Nutrients on Oral Health

Nutrient	Systemic Effect	Nutrient	Systemic Effect
Barium	Tooth decay resistance	Phosphorus	Bone formation/metabolism Tooth decay resistance Tooth formation/metabolism
Boron	Tooth decay resistance	Protein	Epithelial integrity Taste bud renewal Tooth formation Wound healing
Calcium	Bone formation/maintenance Muscle tone maintenance Nerve impulse transmission Tooth formation/maintenance	Selenium	Tooth decay promotion
Calcium–phosphorus balance	Bone maintenance Periodontal maintenance	Silicon	Bone formation
Copper	Bone formation/maintenance Collagen synthesis Periodontal maintenance Wound healing	Strontium	Tooth decay resistance
Fluorine	Tooth decay resistance	Sulfur	Bone maintenance
Folic acid	Epithelial integrity Wound healing	Vanadium	Bone maintenance Tooth decay resistance
Gold	Tooth decay resistance (mild)	Vitamin A	Epithelial integrity Tooth formation Wound healing
Iron	Epithelial integrity Periodontal maintenance	Vitamin B_1	Wound healing
Lead	Tooth decay promotion	Vitamin B_2	Wound healing
Lithium	Tooth decay resistance	Vitamin B_6	Wound healing
Magnesium	Bone formation/maintenance Tooth decay promotion Tooth formation Wound healing	Vitamin C	Epithelial integrity Periodontal maintenance Tooth formation Wound healing
Manganese	Cell membrane formation Tooth decay resistance	Vitamin D	Bone formation/maintenance Tooth formation
Molybdenum	Tooth decay resistance (mild)	Zinc	Epithelial integrity/metabolism Periodontal maintenance Taste bud renewal Wound healing
Nickel	Wound healing		

secondary role. The dietary component contributing most to the initiation and progression of the caries process is fermentable carbohydrates.[170] Biochemical, microbiologic, and animal and human clinical and epidemiologic studies support a causal relationship. Even root surface caries in human populations is enhanced by the ingestion of dietary sugars.[170]

Normally, before eating, the pH of tooth surface plaque exposed to saliva is close to neutrality (pH 6.5–7,0).[171] The ingestion of foods containing fermentable carbohydrates leads to acid production by the cariogenic plaque bacteria on tooth surfaces. The acids cause a rapid drop in pH that can result in demineralization of the tooth substance. If the plaque pH falls below the critical point of about 5.5 and remains there for an appreciable time, the food causing the decrease is likely to support caries initiation and progression.[172,173]

The greatest concentration of acid, or lowest pH, occurs in 5 to 15 minutes, but teeth are attacked by acids for 20 minutes or more.[171] Saliva has a buffering effect that helps to control acid production to some degree, and it contains proteins that act as antibacterial agents. However, in elderly persons, who might have reductions in salivary flow and therefore reduced buffering and antibacterial capacity, each acid attack is significantly prolonged.

Dietary Control

There is compelling evidence that dietary control of dental caries requires modification in the form, quantity, frequency, and timing of consumption of carbohydrates.[174,175] Sucrose traditionally has been regarded as the form of carbohydrate most detrimental to teeth.[174,175] However, recent research indicates that many of the common simple sugars (glucose, dextrose, fructose, maltose, and lactose) can contribute to the rapid formation of acid by dental plaque.[176] Some studies even suggest that complex carbohydrates, such as starches, have the potential to promote caries under certain conditions.[177,178]

Reducing the quantity of fermentable carbohydrates ingested deprives the potentially pathogenic plaque of necessary substrates for growth. It also limits the numbers of cariogenic microorganisms found in the dental plaque.[179,180]

Frequency of consumption is important because each encounter of bacteria with fermentable carbohydrates can result in acid production, tooth surface demineralization, and the formation of carious lesions.[181] There is a strong association between root caries lesions in adults and the frequency of fermentable carbohydrate intake.[181] Restricting between-meal snacks containing cariogenic carbohydrates is advised because frequent sugar consumption, especially between meals, is associated with increased dental caries activity.

The best time to ingest fermentable carbohydrates is with meals. Eating these foods at mealtime will produce less caries than eating the same foods between meals. One reason for this might be that saliva, the production of which is increased during meals, helps neutralize acid production and clears food from the mouth. This is not true for between-meal snacks. Recently, however, it has been established that increasing salivary flow rates after meals, as with sugarless gum chewing, helps reduce plaque acids that can cause caries.[182]

Cariogenicity

Cariogenicity refers to the potential that a specific food or diet has for dental caries formation. The local acidogenic activity of the food, not its nutrient content, largely determines its cariogenic potential.[183] Clinical trials to evaluate the cariogenicity of foodstuffs are expensive processes.[173] Studies must last 2 or 3 years because dental caries develop slowly and are not clinically discernible for many months.

A key determinant of cariogenicity is oral clearance time.[184] When sugar is consumed in foods that adhere to or between tooth surfaces, caries activity has been shown to increase.[183,184] However, if the fermentable carbohydrate source is eaten with a beverage or in liquid form, the time needed for oral clearance is reduced, resulting in a lower net cariogenic potential. Thus, solid or retentive sugar-containing foods are more cariogenic than sugar-containing foods that are liquid or nonretentive.[183] Likewise, fermentable carbohydrates eaten at meals are less cariogenic than the same ones eaten between meals.[175]

Another indication of cariogenicity is the change in plaque pH associated with food consumption.[180] This measure has been used by a number of investigators to monitor the cariogenicity of particular foods and has been found to relate to oral clearance time. Studies have shown that foods that adhere to the teeth depress the plaque pH for longer periods than foods that are removed from the teeth more quickly.[180,181,183,184]

The cariogenic potential of preparations of liquid medications is of particular concern for the geriatric patient. These medications frequently include high levels of sucrose, glucose, or fructose as sweeteners. Studies of patients taking sweetened liquid medications demonstrate a significant increase in dental caries, especially with long-term therapy.[185] Sweetened liquid iron supplements, cough syrups, antibiotics, and anticonvulsants have been shown to decrease plaque pH after ingestion.[185]

Artificial Sweeteners

Research has been focused on identifying and developing substances that serve as taste-competitive, noncariogenic sugar substitutes. Aspartame and saccharin are the two agents currently available. Cyclamate was banned by the Food and Drug Administration in 1970 because of concerns over its safety. That ban currently is being reconsidered.

Aspartame is noncariogenic, but it is not noncaloric.[186] However, its sweetness is of sufficient intensity (180 times sweeter than sucrose) that only small amounts are required, resulting in a very significant reduction in calories. Saccharin is 300 times sweeter than sucrose but is not metabolized by the body and is therefore noncaloric and non-nutritive.[187] Although it has been periodically labeled potentially

carcinogenic, studies to date support its safety for human consumption.

Sugar Alcohols

Technically, the sugar alcohols are not sugars, but they are closely related both chemically and biochemically. Because their degrees of sweetness, compared with those of sugars, are similar, they are used as sugar substitutes.

Sorbitol, mannitol, and xylitol have been used in sugarless chewing gums and candies. Sorbitol- and xylitol-sweetened products appear to be noncariogenic in clinical trials.[173,188] Apparently, xylitol is not metabolized by plaque microorganisms at all, and sorbitol is not metabolized rapidly enough to support an active carious process.

In one study, chewing sorbitol gum after consuming potentially cariogenic snacks helped in counteracting the adverse plaque pH measurements.[188] The investigator postulated that the gum not only stimulated salivary flow, which is known to have a high buffering capacity, but allowed the saliva to penetrate between the tooth surfaces to neutralize acid production by plaque microorganisms.

PERIODONTAL DISEASE

Nutrition has never been implicated as a primary etiologic agent in gingivitis or periodontitis. However, it does play a secondary role by influencing or altering the resistance of the periodontium to the noxious agents and irritants that have a primary etiologic role.[189] The importance of both diet and nutrition in maintaining effective host defense mechanisms to withstand periodontal microbial challenge is well established.[189]

Nutrient deficiencies can affect the rate and degree of periodontal disease rather than its initiation. Research suggests that the disease progresses faster and is more severe in patients whose diets do not supply the necessary nutrients.[189,190] However, there is insufficient evidence at this time to justify nutritional therapy as part of periodontal treatment.[167]

OTHER ORAL CONDITIONS

The role, if any, of diet and nutrition in edentulous ridge resorption, mucosal lesions, glossodynia, and taste perception is poorly defined, although research is evolving in the area of diet as a risk factor for oral cancer.[127,167] Positive associations have been found with increasing consumption of meats, liver, sodium, and retinol.[191,192] Intakes of vitamins A, C, and E, as well as consumption of raw fruits and vegetables, are associated with a reduced risk of oral cancer.[127,191–196]

A major reason for poor adaptation to dentures by elderly persons is reduced tissue tolerance resulting from an inadequate diet.[197] Thin and friable epithelium covering the edentulous area might not tolerate the forces imposed on it by the hard, unyielding base of the denture.

The composition of saliva is critically dependent on flow rate from the glands, and numerous studies have demonstrated that both the physical consistency and the nutritional quality of the diet influence the structure of the glands, as well as the flow rate of saliva.[198]

NUTRIENT INTAKE AND MALNUTRITION

Inadequate amounts of nutrients can result in fragile, friable oral tissues, with a loss of adaptability and tolerance to irritants and a loss of repair potential.[199] For many nutrient deficiencies, the oral cavity serves as an early warning system.

Because of the rapid tissue turnover and easy visibility of the oral mucosa, it is possible to identify signs of inadequate intake or improper absorption before other organ systems are affected.[199] Although not all nutrient deficiencies have oral manifestations, the most common ones are listed in **Table 8-9**. Oral signs indicative or suggestive of malnutrition are listed in **Table 8-10**.

IMPACT OF ORAL HEALTH ON NUTRITIONAL STATUS

There is general agreement that poor oral health is a risk factor contributing to malnutrition, weight loss, poor general health, and loss of strength.[200,201] Although the impact of oral health status alone on dietary intake and nutritional status of older adults is virtually unknown, any alteration in the anatomic structures or physiologic functions of the oral cavity can play an important role in deterring the elderly population from attaining or maintaining a proper diet and nutritional state.

Dietary intake, with respect to food selection, chewing, and swallowing, is integral to the health of the geriatric patient. Many factors influence food selection, including social customs, taste preferences, amount of preparation, and cost.[202] Chewing is influenced by the status of the oral cavity and the efficiency of the masticatory apparatus. Swallowing

Table 8-9 Nutritional Deficiencies and Related Oral Manifestations

Nutrient Deficiency	Oral Manifestations
Vitamin A	Candidiasis Gingiva—hypertrophy, inflammation Oral mucosa—keratosis, leukoplakia Periodontal disease Taste—decreased acuity Xerostomia
Vitamin B complex	Lips—angular cheilosis Oral mucosa—leukoplakia Periodontal disease Tongue—papillary hypertrophy, magenta color, fissuring, glossitis
Vitamin B_2 (riboflavin)	Filiform papillae—atrophic Fungiform papillae—enlarged Lips—shiny, red, angular cheilosis Tongue—magenta color, soreness
Vitamin B_3 (niacin) *(pellagra)*	Lips—angular cheilosis Mucositislstomatitis Oral mucosa—intense irritation/inflammation, red, painful, denuded, ulcerated Tongue—glossitis, glossodynia Tongue (dorsum)—smooth, dry Tongue (tip/borders)—red, swollen, beefy Ulcerative gingivitis
Vitamin B_6 (pyridoxine hydrochloride)	Burning/sore mouth Lips—angular cheilosis Tongue—glossitis, glossodynia
Vitamin B_2 (cyanocobalamin) *(pernicious anemia)*	Bone loss Burning/sore mouth—mucositislstomatitis Gingiva—hemorrhagic Halitosis Hemorrhagic tendency—petechiae Lips—angular cheilosis Oral mucosa—epithelial dysplasia Oral paresthesias—burning, numbness, tingling Periodontal fibers—detachment Taste—loss or distortion Tongue—beefy red, glossy, smooth; glossitis, glossodynia, loss of papillae Ulcerations—aphthous type Wound-healing response—delayed Xerostomia
Vitamin C *(scurvy or megavitamin C withdrawal)*	Blood vessels—fragility Bone—abnormal osteoid formation, fragility, loss Burning/sore mouth Candidiasis Gingiva—friability, raggedness, swelling, redness, hemorrhagic tendency Hemorrhagic tendency—petechiae, subperi-osteal Oral infections—decreased resistance Periodontal disease—increased suscepti-bility Teeth—marked mobility, spontaneous exfoliation Wound-healing response—delayed

(*continues*)

Table 8-9 Nutritional Deficiencies and Related Oral Manifestations (continued)

Nutrient Deficiency	Oral Manifestations
Vitamin D	Periodontal disease
Vitamin K	Candidiasis Gingiva—bleeding
Calcium	Bone—excessive resorption, loss of mineral, fragility, osteoporosis Hemorrhagic tendency Periodontal disease Teeth—mobility, early loss, edentulism
Copper	Bone—decreased trabeculae, decreased vascularity, fragility
Folic acid	Burning/sore mouth—mucositislstomatitis Candidiasis Filiform/fungiform papillae—atrophic, loss Gingiva—inflammation Lip—angular. cheilosis Tongue—glossitis Tongue (dorsum)—slick, bald, pale, or fiery Tongue (tip/borders)—red, swollen Ulcerations—aphthous type
Iron	Bleeding complications—increased risk Burning/sore mouth Candidiasis Dental caries—increased susceptibility Dysphagia Filiform papillae—atrophic Lips—angular cheilosis, pallor Oral infections—increased risk Oral mucosa—pallor Oral paresthesias Tongue—atrophic, pale; glossitis, glossopyrosis Ulcerations—aphthous type XerostomiaTongue (dorsum)—slick, bald, pale, or fiery Tongue (tip/borders)—red, swollen Ulcerations—aphthous type
Magnesium Iron	Bone—fragility Gingiva—hypertrophy Bleeding complications—increased risk Burning/sore mouth Candidiasis Dental caries—increased susceptibility Dysphagia Filiform papillae—atrophic Lips—angular cheilosis, pallor Oral infections—increased risk Oral mucosa—pallor Oral paresthesias Tongue—atrophic, pale; glossitis, glossopyrosis Ulcerations—aphthous type Xerostomia
Phosphorus Magnesium	Dental decay—increased susceptibility Periodontal disease Bone—fragility Gingiva—hypertrophy

(*continues*)

Table 8-9 Nutritional Deficiencies and Related Oral Manifestations (continued)

Nutrient Deficiency	Oral Manifestations
Protein Phosphorus	Bone—decreased repair Epithelium—fragility, burning sensation Lips—angular cheilosis Oral infections—decreased resistance Periodontal disease—increased susceptibility Wound-healing response—delayedDental decay—increased susceptibility Periodontal disease
Protein-calorie Protein	Bone loss. Candidiasis Necrotizing ulcerative gingivitis Periodontal diseaseBone—decreased repair Epithelium—fragility, burning sensation Lips—angular cheilosis Oral infections—decreased resistance Periodontal disease—increased susceptibility Wound-healing response—delayed
Water Protein-calorie	Burning/sore mouth Epithelium—dehydration, fragility Muscle strength—diminished Tong ue—glossopyrosis Xerostomia Bone loss Candidiasis Necrotizing ulcerative gingivitis Periodontal disease
Zinc Water	Candidiasis Dental caries—increased susceptibility Epithelial thickening Oral mucosa—atrophic Periodontal disease—increased susceptibility Smell acuity—decreased Taste acuity—loss or distortion Wound-healing response—delayed XerostomiaBurning/sore mouth Epithelium—dehydration, fragility Muscle strength—diminished Tong ue—glossopyrosis Xerostomia
Zinc	Candidiasis Dental caries—increased susceptibility Epithelial thickening Oral mucosa—atrophic Periodontal disease—increased susceptibility Smell acuity—decreased Taste acuity—loss or distortion Wound-healing response—delayed Xerostomia

Table 8-10 Oral Signs Suggestive of Malnutrition

Oral Area	Normal Appearance	Signs Associated with Malnutrition
Teeth	Bright; no caries; no pain	Dental caries; might be missing or erupting abnormally
Gums	Healthy; red; not swollen; no bleeding	Receding; spongy; bleed easily
Tongue	Deep red; not swollen or smooth	Scarlet or magenta color; smooth; raw; swelling; sores; atrophic, hyperemic, or hypertrophic papillae
Lips	Smooth; not swollen or chapped	Redness; swelling of mouth and lips
Face	Uniform color; smooth; pink; healthy appearance; not swollen	Lumpiness or flakiness of skin around mouth
Salivary	Face not swollen in gland areas	Parotid enlargement (swollen glands, cheeks)

Adapted with permission of the American Public Health Association from Christakis G., *Nutritional Assessment in Health Programs*, 7th ed., American Public Health Association, 1984, p. 19.

depends on adequate lubrication and moisture provided by the salivary glands, as well as sufficient functioning of the oral musculature to form and prepare a food bolus. Clearly, any factor that interferes with food selection, chewing, or swallowing can restrict food intake and thus affect nutritional status.

DENTITION STATUS

Dentition status, inasmuch as it contributes to masticatory efficiency, can exert potent effects on dietary intake. Research suggests that the number of occluding teeth, especially in the posterior segments of the mouth, is correlated with masticatory efficiency.[203] Masticatory efficiency is dependent not only on the number and condition of teeth present but also on the length of time spent in chewing a bolus of food and the force exerted when biting.[204]

Impaired masticatory efficiency and biting force have been associated with many oral conditions.[10,14,203,204] These include atrophy of orofacial musculature; oral dyskinesia; trismus; bone loss; tooth attrition, brittleness, mobility, pain, or loss; advanced carious lesions; TMJ dysfunction or dislocation; mucosal atrophy; generalized periodontal disease; gingival enlargement; and ill-fitting dentures.

One commonly held belief is that optimal masticatory efficiency allows an individual to select a wider variety of foods, which leads to a more nutritionally balanced diet.[203] It is also suggested that the loss of mechanical chewing efficiency leads to a preference for soft, easy-to-chew foods, which can increase the risk of nutritional deficiencies.[16,68,200,202–207] These foods tend to be high in carbohydrates, cholesterol, and calories but low in fiber, protein, iron, calcium, and essential vitamins. Such a diet routinely contains salt and saturated fats in unhealthy amounts for persons with heart disease and usually lacks vitamin K, which leads to calcium loss in bone.[208]

Edentulousness can affect masticatory function and dietary choice, but its influence on nutritional status is controversial. Some researchers have found that tooth loss is a strong predictor of inadequate nutrition, resulting from problems with biting, chewing, or swallowing foods.[209] Other investigators have found little evidence to indicate that adequate dentition is necessary for geriatric patients to maintain a satisfactory nutritional state.[210,211]

Even the incidence of malnutrition, weight loss, and gastrointestinal disturbances in the older adult appears to be unrelated to impaired masticatory function.[204,212] In these studies, the percentage of individuals with significantly reduced or inefficient masticatory ability was similar to the percentage of persons with and without overt signs of malnutrition or undernutrition. In addition, various changes in blood chemistry usually associated with malnutrition have not been routinely found in individuals with significantly reduced masticatory ability.[213] It would appear that replacing missing teeth with partial or complete dentures would improve chewing and limit the risk of nutritional problems. Indeed, the change from poor natural dentition or edentulousness to complete dentures is generally accompanied by improved chewing efficiency and nutritional status, but there are conflicting observations in the literature.[210,211,214]

Properly fitted dentures might allow one to choose from a wider selection of food textures. However, denture wearing has been reported to interfere with the ability to eat satisfactorily, talk clearly, and laugh freely.[202,205] Elderly denture wearers also

require more time to chew before swallowing than do those with natural teeth.[39]

It is well known that the denture wearer does not have the chewing efficiency enjoyed by the individual with natural teeth. Several studies have shown that significant differences in chewing ability occur among persons with intact natural dentition, individuals with partial prosthetic replacements, and individuals with complete dentures.[203–205,207] Dental studies have established that the chewing efficiency of an average complete denture wearer is only 15% to 25% of that of an individual with natural teeth.[202,203,213–217]

Even so, a chewing efficiency as low as 23%, a level attainable with just the 12 maxillary and mandibular anterior teeth, was sufficient to digest the 28 experimental foods in one study of masticatory efficiency and food assimilation.[218] Because the masticatory efficiency attained by the average denture wearer is in this range, most people with dentures should be able to chew food adequately for proper digestion.

The condition of dentures has a direct bearing on an individual's ability to chew. Well-fitting dentures in a healthy mouth can result in better chewing, swallowing, and digestion.[210,214,219] Problems with denture fit, bone shrinkage, and the gum tissues supporting the denture compromise masticatory function and can negatively alter dietary intake.[39] In fact, many denture wearers avoid foods that tend to slip under dentures or are too difficult to manipulate and chew.[209,210,214,215]

Other studies have reported significant variation in the masticatory performance of people who wear dentures.[220–223] Some individuals are barely able to comminute a test food, whereas others with similar prostheses have a relatively high degree of masticatory proficiency. Furthermore, approximately five times more effort is required for the average person wearing complete dentures to pulverize a test food to the same degree that a person with natural dentition can. This agrees with previous reports that impaired chewing ability is not usually improved by chewing food longer or by increasing the rate of chewing, but rather by ingesting foods that are softer and easier to chew or by swallowing larger particles.[224–226] Therefore, denture wearers can be more prone to accidental choking from improper mastication.[227]

Data from dietary surveys before and after the insertion of new dentures are inconclusive about associated changes in essential nutrient intake.[214,228–233] Before the insertion of new dentures, several essential nutrients were consumed in quantities significantly lower than the recommended daily allowance. After new dentures were placed, shifts in nutrient intake occurred, although the changes were not necessarily beneficial. Subjective evaluations, however, indicated improved chewing efficiency, which aided food digestion, particularly of fibrous foods.

Using dental implants to replace missing teeth or as support for dentures can improve oral function and are not contraindicated as a treatment of choice for geriatric patients.[234–236] One retrospective study showed that the success rate of 160 dental implants in a geriatric cohort (n = 47, median age 89 years) was 99–100%.[235] Patients with implant supported full (overdentures) or partial dentures usually have higher satisfaction, a better quality of life, and a stronger maximal bite force than those with conventional full dentures.[237–239] Medical and dental history, socioeconomic status, and treatment planning should be evaluated in geriatric patients before any dental implant procedure. Implant placement in a patient with uncontrolled diabetes mellitus might result in a failed implant.[240] Although bisphosphonates have not been shown to have a negative effect on dental implant outcomes, BRONJ cases have been reported in patients with dental implants.[234,241] Unfortunately, risk factors associated with dental implant–induced BRONJ are still unknown.[234] Special precautions for implant placement must be taken in medically compromised geriatric patients.

Self-Perceived Chewing Ability

Experimental subjects' evaluations of their own chewing ability have been examined as possible predictors of masticatory efficiency, but most reported results are conflicting.[203,215,217,233,242–244] There appears to be wide individual variation in the subjective assessment of chewing problems that is not always related to dentition status. For those with poor masticatory efficiency, the lack of a perceived problem is probably in part the result of selection of foods that are easy to chew or preparation of food in such a way as to facilitate chewing. In fact, perceived ease of chewing is related to subjective estimates of food preference.[203,216,217,245] In general, denture wearers give lower preference ratings to hard-to-chew foods than do persons with intact or even compromised natural dentition.

Dietary Control of Chewing Difficulties

For those with chewing problems caused by dentures or tooth loss, the key is to modify food selection habits and methods of preparing foods for easier chewing. Specific ways to overcome chewing difficulties are listed in **Table 8-11**.

Table 8-11 Dietary Control of Chewing Difficulties

- Drink fluids with meals to aid in chewing and swallowing.
- Chop, grind, or mechanically blend foods that are hard to chew.
- Add sources of dietary fiber (stems of vegetables, whole grains, skins of fruits and vegetables, and seeds or berries) that can be cooked, shredded, mashed, ground, or softened with liquids without affecting the fiber content.
- Shred or chop raw vegetables and use them in salads.
- Mash or strain cooked vegetables.
- Buy prechopped vegetables and meat.
- Prepare meats and vegetables in soups, stews, and casseroles.
- Trim meats to remove fat and tough fibers.
- Substitute softer, protein-rich foods such as fish, eggs, peanut butter, cheese, baked beans, ground meats, or yogurt for regular meat.
- Use melted cheese as a sauce on vegetables or toast to increase protein intake.
- Add extra nonfat dry milk powder to cream soups, cooked cereals, puddings, custards, creamed vegetables, casseroles, and milk beverages to increase protein and calorie content.
- Use cooked whole-grain cereals such as oatmeal or mixed grains.
- Add bran to hot cereals, baked breads, meatloaf, and casseroles.
- Use fruit juices in place of fruits. Most fruits can be pureed in a blender and the pulp added to juices.
- Avoid sticky foods that adhere to teeth and dentures.
- Use menus from cookbooks written for people with chewing problems.
- Most important, eat a variety of foods from the major food groups each day.

ORAL CANCER

Neoplasms in the oral cavity can interfere with chewing and swallowing because of both pain and infiltration of tissues. Antineoplastic drugs and radiation therapy can alter the character and volume of saliva. In addition, the balance of the oral flora is disrupted, allowing overgrowth of opportunistic organisms such as *Candida* species.

Many patients who undergo radiation therapy for oral cancer become nutritional casualties. Profound loss of appetite is an early and sustained reaction to radiation-induced soreness, xerostomia, taste loss, dysphagia, and nausea and vomiting.[246] Eating becomes a pleasureless and painful chore, and food selection is restricted to items that do not aggravate the oral discomfort, often at the expense of adequate nutrition. When prolonged and severe enough, lack of nutrients can precipitate a nutritional deficiency stomatitis.

ORAL PAIN

Oral pain can reduce food intake in both texture and amount. In fact, many patients experiencing dental or facial pain avoid certain foods.[247] As an example, mucositic tissues are sensitive to temperature and pressure, so a semisoft diet that is low in sucrose and citric acid is advised.[248]

Masticatory ability, biting force, and tongue movements are impaired in painful oral conditions, thus influencing the ability to chew many foods. Oral pain can also interfere with swallowing. Conditions that can cause oral pain are listed in **Table 8-12**.

Table 8-12 Oral Conditions That Can Be Painful

- Angular cheilosis
- Aphthous ulceration
- Benign mucous membrane pemphigoid
- Burning mouth syndrome
- Candidiasis
- Contact stomatitis
- Dental caries
- Denture stomatitis
- Erythema multiforme
- Glossodynia
- Glossopharyngeal neuralgia
- Herpes labialis
- Herpetic stomatitis
- Hypersensitive teeth
- Lichen planus
- Mucositis
- Necrotizing ulcerative gingivitis
- Oral cancer (advanced)
- Periodontal disease
- Pulpal infection
- TMJ dysfunction
- Traumatic ulceration
- Trigeminal neuralgia

Note: TMJ: temporomandibular joint.

SALIVA

Saliva is essential for taste perception, mastication, and swallowing of foods. It provides the environment for optimal functioning of taste buds and contributes to ingestion and digestion by forming a mucin-coated food bolus and adequate fluid volume to allow for ready passage along the chewing and swallowing surfaces. The bolus is then digested in the gastrointestinal tract.

When salivary flow is deficient, it causes various stresses on the hard and soft tissues of the mouth, leading to increased oral disease and dysfunction of chewing, swallowing, and taste.[39] The greater concentration of electrolytes in a diminished amount of saliva can result in a salty or metallic taste in the mouth. In addition, decreased ptyalin levels in the reduced salivary flow can affect digestion of chewed particles.

Most patients with xerostomia have difficulty eating solid and dry foods, which can contribute to changes in nutritional intake patterns.[42,249–251] Oral pain associated with sialadenitis or sialolithiasis can also impair oral intake. In response, elderly individuals reduce the intake of various foods or switch to foods more easily chewed.

To facilitate chewing and swallowing in severe xerostomia, food must be lubricated with artificial saliva or prepared in liquid or semiliquid form. Saliva substitutes have been shown to improve both chewing and swallowing.[250,252] Many patients moisten foods with sauces, gravies, milk, and other fluids.

TASTE AND SMELL SENSITIVITY

For the most part, taste and smell determine the flavor of foods and beverages.[253] Reduced acuity of either of these senses can significantly lessen the ability to enjoy food and thus decrease appetite. Declines in gustation and olfaction, whether with age, chronic disease, or drug use, decrease the flavor and palatability of foods and beverages. Because of this, the senses of both taste and smell are important in food selection and nutrient intake.[252,254]

Decreased taste sensitivity is compounded by dental disease or poor oral hygiene.[5] The causes can be physical, such as debris covering the taste buds, or chemical, such as taste fatigue from constant stimulation by decaying matter in the mouth.[45] Also, chronic dental or periodontal infections can result in the continuous discharge of purulent matter into the mouth, creating a constantly unpleasant taste. Routine oral hygiene has been shown to improve sensitivity to salty and sweet tastes and can improve the elderly patient's appetite.[255]

Saliva has modulating effects on taste sensitivity. A salty taste is detected only when the concentration is above salivary levels of sodium chloride. Saliva diminishes the effect of a sour taste as a result of buffering by salivary bicarbonate. Decreased salivation also alters the taste of many foods.[253]

Diminished taste also can result from altered taste perception.[253] It has long been suspected that denture wearers have a lowered ability to taste, and edentulous individuals experience a reduction in taste sensitivity after the insertion of complete dentures.[216,253,256] Perhaps the taste buds in the hard palate are more insensitive to taste, especially sour and bitter, when covered with dentures.[253]

CONCLUSION

The cumulative effects of aging, disease, and trauma contribute to the wide variety of oral health problems prevalent in the older adult. Although many of these problems can be neither prevented nor cured by diet alone, to ignore nutritional considerations in the oral disease process would be a serious error. Many of the oral problems mentioned previously are associated with dietary deficiencies, excesses, or practices that are detrimental to the oral and circumoral structures. It is imperative that dietary intake provide adequate nutrients to support oral health and function.

There are also strong associations among oral health status and food selection, chewing efficiency, and ability to swallow. Clearly, oral health problems that interfere with any aspects of these factors can restrict food intake and ultimately affect nutritional status. It is the position of the Academy of Nutrition and Dietetics that nutrition is an integral component of oral health.[257] Nondental healthcare providers would do well to screen older adults for oral risk factors that could negatively affect nutritional status.[258]

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