Chapter 17

ORAL HEALTH EFFECTS OF COMBAT STRESS

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The average American, if not prepared, will cope with distress by eating more junk food, drinking more alcohol, smoking more cigarettes, taking more over-the-counter drugs, or under-the-counter drugs. We are a society that copes with distress with some form of oral behavior.

—JO Prochaska, PhD, developer of the Trans-Theoretical Model of Behavior Change
(interview available at: mms://mediastream.buffalo.edu/content/nur/Part1a.wmv, accessed July 13, 2010)

INTRODUCTION

Approximately 5% to 10% of disease and nonbattle injuries among service members result from oral diseases or injuries.[1] The rate of dental emergencies is higher for units whose soldiers deploy with poor oral health. Oral diseases can cause impaired duty performance, work loss, restricted activity, poor diet, difficulty pronouncing words, inability to sleep, and excruciating pain. If they are not prevented or treated early, oral diseases can cause severe, life-threatening illness and may even require medical evacuation from theater. One recent study of US Army soldiers (regular Army, reserve, and National Guard) medically evacuated from the Central Command area of responsibility during 2003 and 2004 found that oral disease accounted for 42% of oral-facial evacuations.[2]

STRESS AND ORAL HABITS

Many Americans have a low level of health literacy and perceive oral health as less important and separate from general health.[3] Advances in dental research since the 1950s have shown that nearly all oral disease is preventable, yet many people continue to have a fatalistic view that "dental problems" are inevitable, of mysterious origin, and can be ignored as long as they are not unaesthetic or painful. Many service members join the military with these erroneous beliefs about oral diseases, viewing oral hygiene as mainly cosmetic and unnecessary during field training or operations.

The primary causes of oral diseases during deployment are bacterial or viral infections that are initiated or exacerbated by the effects of poor oral hygiene, poor diet, or substance use. Self-care and hygiene habits are often the first things to break down when a soldier is deployed to an austere environment or experiences overwhelming stress. Many of these soldiers also turn to harmful oral habits such as using tobacco or eating and drinking nutrient-poor beverages and foods that are high in simple carbohydrates.

According to the 2006 Department of Defense Survey of Health-Related Behaviors in the Reserve Component,[4] nearly 45% of service members admitted to eating as a way to cope with stress. Women were significantly more likely than men to eat as a coping strategy (51.8% vs 42.5%), while more men than women reported using cigars or smokeless tobacco (11.4% vs 3.6%). Reserve-component men and women were similar in their use of cigarettes (19.4%) and alcohol (24.9%) for coping. Results of the 2008 Department of Defense Survey of Health-Related Behaviors Among Active Duty Military Personnel[5] shows that men were significantly more likely to smoke cigarettes (28.6%) or drink alcohol (34.5%) for coping than were women (21.0% and 25.2%, respectively). Comparisons between the services show that more service members in the Marine Corps (39.9%), the Army (35.6%), and the Navy (33.7%) have a drink to cope with stress than do service members in the Air Force (25.0%). The same pattern is seen for smoking, with 33.2% of Marine Corps, 30.8% of Army, 27.9% of Navy, and 18.8% of Air Force service members lighting up a cigarette to cope with stress. Women, on the other hand, are still significantly more likely to use eating as a coping mechanism (56.2%) than are men (45.7%). Service members in the Navy and Army are significantly more likely to use eating as a coping mechanism (49.6% and 48.2%, respectively) than those in the Air Force or Marine Corps (45.1% and 44.5%, respectively). The combination of poor oral hygiene with harmful oral habits such as tobacco use and increased intake of refined carbohydrates causes oral disease (Figure 17-1).

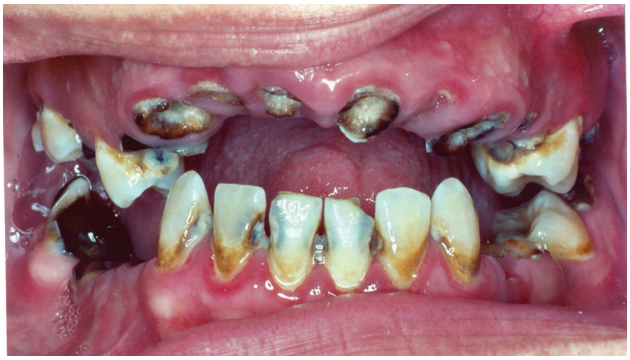

Figure 17-1. Plaque accumulation, tobacco staining, gingival inflammation, and generalized caries resulting from a combination of inadequate oral hygiene, tobacco use, and refined carbohydrate intake.

Changes in diet during deployment or other stressful situations can cause service members to suffer from dehydration and micronutrient deficiencies that can lead to skin conditions, stress fractures, anemia, and other conditions.[6–11] Because the oral epithelium has a high rate of cellular turnover, these same deficiencies can affect oral health by preventing the oral epithelium from renewing or repairing itself. As a result, the first signs of micronutrient deficiency can appear in the mouth, and may present as glossitis, angular cheilitis, stomatitis, and gingivitis. Undernutrition can also exacerbate oral infections and has been associated with increased progression of periodontal disease.[12] Vitamins A, C, D, E, B_2, B_6, B_{12}, niacin, and folic acid, and minerals such as zinc, iron, magnesium, and calcium are essential for repairing oral epithelium, maintaining periodontal attachment, preventing demineralization of bones and teeth, and ensuring an adequate amount of saliva of sufficient quality to protect the oral cavity. In addition to enabling taste, mastication, and digestion, saliva is critical for defense against oral diseases. Compromised saliva composition or flow impairs or eliminates its antifungal, antiviral, and antibacterial activities and prevents it from protecting the teeth from demineralization by acids from oral bacteria or foods and beverages.

STRESS AND DENTAL HARD-TISSUE DISEASES

When eating is used as a coping mechanism for stress, it often involves the frequent consumption of foods containing organic acids and simple carbohydrates such as sugar and starch. Between-meal consumption of these foods or beverages (ie, sports drinks, energy drinks, or soda) promotes dental caries and dental erosion.

Dental Caries

The majority of dental emergencies during deployment have been for conditions resulting from dental caries.[13–18] Exact incidence of new disease is unknown, but a pilot study[19] of soldiers deployed in Operation Iraqi Freedom for 6 months during 2003 found that the number of carious lesions increased by 156%, and the severity of disease, as measured by the number of tooth surfaces involved, increased by 183% over predeployment statistics.

Dental caries is caused when poor oral hygiene allows uncontrolled growth of bacteria in dental plaque on tooth surfaces (Figure 17-2). The bacteria rapidly metabolize the simple carbohydrates (starches or sugars) in the diet into destructive acids (lactic acid). Fermentable carbohydrate metabolism supports the colonization of more bacteria on the tooth surface, further increasing the amount of acid produced. Following exposure to fermentable carbohydrates, plaque pH falls below 5.5, the critical pH for maintaining the structural integrity of the mineralized dental tissue. It can take 30 to 40 minutes for the pH to rise again, depending on the flow rate and buffering capacity of the individual's saliva. As a result, the amount of bacterial acid produced in any 24-hour period mirrors the frequency and duration of exposure to fermentable carbohydrates more closely than the total amount of carbohydrate consumed.

Starches do not directly serve as substrate for oral bacterial fermentation. Starch granules in grains, vegetables, potatoes, and beans are damaged when they are subjected to heat and mechanical forces, producing a gelatinized starch. This starch is further broken down by salivary and bacterial amylases into sucrose, maltose, and maltotriose—substances that are available for bacterial acid production.[20] As a result, untreated whole grains and raw vegetables have lower caries-promoting potential than heat-processed foods like white breads, crackers, chips, snacks, and dry cereal. Foods that contain sugar plus starch destroy dental enamel more rapidly than pure sugar foods because the starch acts like digestible glue, keeping the sugar in close contact with the tooth surface for longer periods of time. Teeth that have low levels of fluoride in the enamel are more vulnerable to these acids, and decay can begin and progress quite rapidly. Without regular exposure to fluoride, sugar consumption is a strong indicator of caries risk.[21]

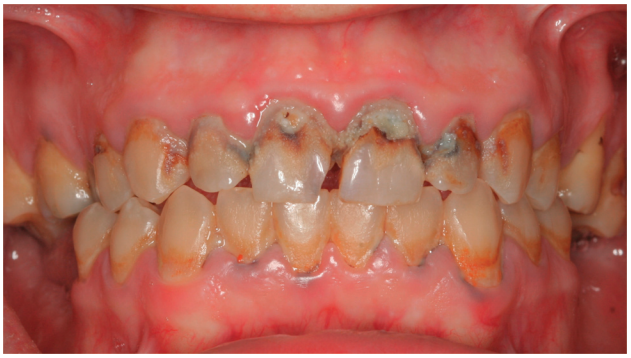

Figure 17-2. Extensive accumulation of bacterial plaque after cessation of oral hygiene. Gingiva is inflamed and several carious lesions are evident.
Photograph: Courtesy of Captain Andrew Marshall, US Army Dental Corps.

Dental Erosion

Dental erosion, or erosive tooth wear, is described as "the loss of dental hard tissue either through chemical etching and dissolution by acids of nonbacterial origin or by chelation."[22(p243)] The prevalence of dental erosion is increasing, particularly in younger populations.[23,24] Nonbacterial acids that cause erosion primarily come from two sources: gastric acids or diet. Caffeinated sodas, sports drinks, and energy drinks are commonly used in theater for hydration, to maintain alertness, or for simple enjoyment. These flavored beverages often contain polybasic organic acids such as citric acid, phosphoric acid, malic acid, and tartaric acid. Popular beverages that fall into this category include soft drinks, sports drinks, energy drinks, and sweetened, bottled tea blends. Other types of acidic drinks include apple or citrus fruit juice. Acidic foods include citrus, pineapple, and sour candies. Any of these acidic foods and beverages can accelerate the progression of dental caries. Organic acids accelerate the progression of decay by chemically eroding the dental enamel[25] (Figure 17-3). Polybasic acids have a unique buffering capacity and can maintain an acidic pH even with marked dilution. This high titratable acid level multiplies the actual number of hydrogen ions available for interaction with the tooth surface, and is thought to be an important reason for their erosive properties. The chemical structure of polybasic organic acids also gives them the ability to chelate calcium at higher pHs, so they can erode dental enamel even in a neutral environment.[26]

Exposure to gastric acids is caused by either gastroesophageal reflux disease (GERD) or eating disorders that involve self-induced vomiting. Exposure to gastric acids is one of the most destructive processes that can affect the teeth. Stomach acids demineralize the outer layers of tooth enamel, leaving behind the protein matrix, which is then easily removed with brushing. GERD is the most common gastrointestinal-related diagnosis given during office visits, and affects somewhere between 10% to 20% of the Western population.[27] The disorder causes erosion of the lingual and occlusal surfaces of the teeth, affecting the posterior teeth most severely. In the early stages, affected teeth exhibit a smooth, glazed appearance. Damage generally progresses to the development of a "dished out" appearance and the exposure of the dentin layer (Figure 17-4). Over time the affected teeth can become weakened and may develop thermal sensitivity.[28] Patients with GERD are usually prescribed proton pump inhibitors and must avoid eating certain foods, as well as sleeping within a few hours of eating, exercising, or bending over. Disruption of dietary or medication routines by high operations tempo or combat situations may cause recurrence of GERD symptoms and repeated exposure of the dentition to gastric acids.

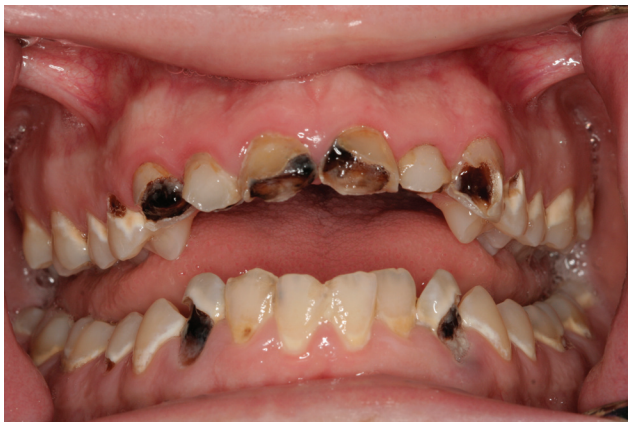

Figure 17-3. Generalized demineralization and loss of enamel on the facial surfaces of teeth just above the gingival margin in the absence of gingival inflammation is often an indication of frequent use of sugared beverages that contain organic acids.
Photograph: Courtesy of Captain Andrew Marshall, US Army Dental Corps.

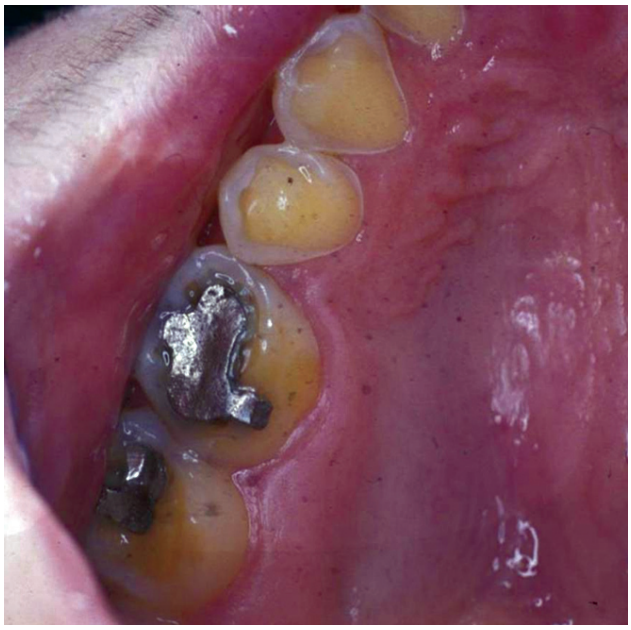

Figure 17-4. Erosion of enamel and exposure of the dentin layer in a patient with gastroesophageal reflux disease.
Photograph: Courtesy of Bennett T Amaechi, Associate Professor and Director of Cariology, University of Texas Health Science Center at San Antonio.

Eating Disorders

In most cases, dental hard-tissue damage is unlikely to signify anorexia or bulimia, except in those with an already established diagnosis who experience a recurrence under stress. Oral manifestations associated with eating disorders may include one or more of the following[29]:

- smooth erosion of the enamel or perimylolysis,
- traumatized oral mucosal membranes and pharynx,
- variations in the periodontium,
- enlargement of the parotid or submandibular salivary glands,
- xerostomia (dry mouth due to reduced salivary flow), and
- dental caries.

When vomiting occurs repeatedly over time, tooth enamel becomes thin and eroded, resulting in a smooth, glassy appearance. In the case of eating disorders, these changes are most commonly seen on the lingual, occlusal, and incisal surfaces of the maxillary teeth. When confined to these areas, the condition is termed "perimylolysis" (Figures 17-5 and 17-6). These areas of the teeth are more susceptible to erosion due to the combined chemical and mechanical effects of regurgitated gastric acids and their retention on the surface of the tongue. Erosion is usually not clinically detectible until vomiting behavior has occurred for about 2 years. However, the daily frequency of self-induced vomiting is one of the main determinants of the rate of progression and degree of dental erosion. As is the case with GERD, erosion may eventually affect the occlusal and facial surfaces of teeth, resulting in exposure of the underlying dentin and decreased vertical dimension (overclosure; Figure 17-7).

Patients with eating disorders may also develop diet-caused dental erosion in other areas of the teeth. Several studies have reported that people with restrictive anorexia tend to favor highly acidic, low-

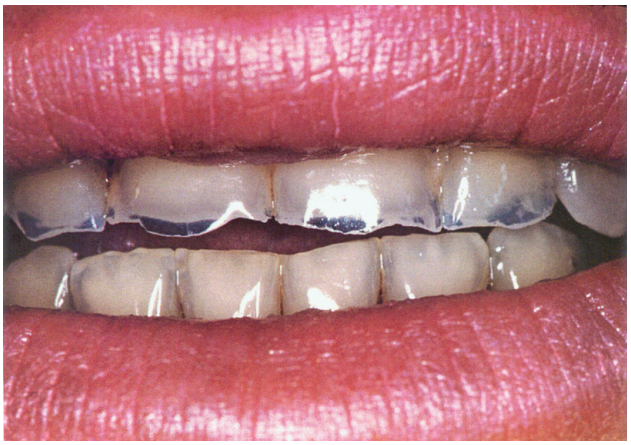

Figure 17-6. Translucency of the maxillary central incisors due to erosion in a patient with bulimia.
Photograph: Courtesy of cosmetic dentist Dr Craig Mabrito, Houston, Texas.

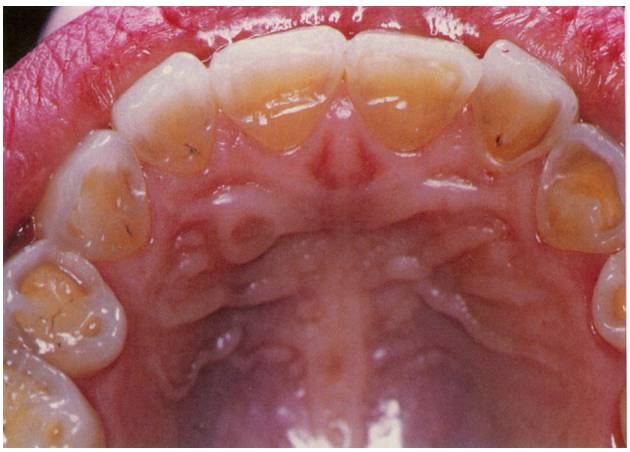

Figure 17-5. Erosion of the lingual, occlusal, and incisal surfaces of the maxillary teeth in a patient with bulimia.
Photograph: Courtesy of cosmetic dentist Dr Craig Mabrito, Houston, Texas.

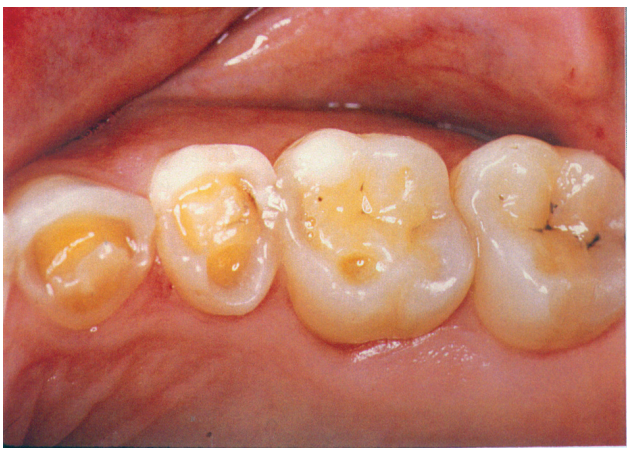

Figure 17-7. Occlusal wear and dentin exposure due to erosion in a patient with bulimia.
Photograph: Courtesy of cosmetic dentist Dr Craig Mabrito, Houston, Texas.

calorie foods, particularly raw citrus.[29–31] If exposure is frequent, these foods tend to cause erosion on the buccal or facial surfaces of the tissue, in contrast to perimylolysis (Figure 17-8).

Stomach acids may irritate the gingival tissue if exposure is frequent, but controlled studies of gingival inflammation in patients with eating disorders have produced conflicting results.[32,33] A more common cause of gingival inflammation in patients with eating disorders appears to be a lack of interest in oral hygiene, which can accompany depression. Also, alveolar bone support of the teeth may be compromised in some patients. Patients with severe disease experience early bone loss or osteoporosis as a result of changes in their estrogen and cortisol levels, and are probably at risk for accelerated alveolar bone loss.

Trauma to the oral mucous membranes or the oropharynx may also occur in patients who engage in binge eating or self-induced vomiting. The rapid ingestion of food associated with binge eating may cause trauma, as may the force of regurgitation. Objects used to induce vomiting may also injure the soft palate.

Salivary gland enlargement is seen in approximately 10% to 50% of patients who binge eat and purge by vomiting. The gland most frequently involved is the parotid gland, and swelling usually occurs 2 to 6 days after bingeing/purging behavior. The enlargement often becomes persistent as the eating disorder progresses. The swelling may give a square, widened appearance to the mandible. However, the involved gland is usually soft and painless upon palpation and has a patent duct, normal salivary flow, and an absence of inflammation, both clinically and histologically. Other histological characteristics include greater acinar size, increased secretory granules, fatty infiltration, and fibrosis. The exact cause of the enlargement is not known, but individuals who purge by methods other than vomiting do not experience salivary gland enlargement. Hypothesized reasons include autonomic stimulation of the glands by activation of the taste buds or cholinergic stimulation of the glands during vomiting.[29]

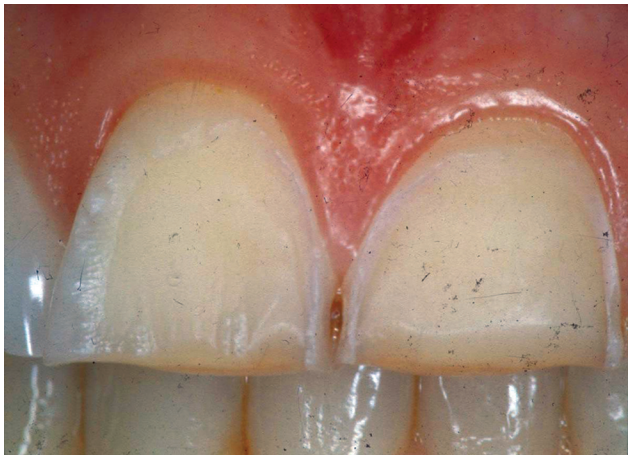

Figure 17-8. Erosion of the facial enamel and exposure of the underlying dentin.
Photograph: Courtesy of Bennett T Amaechi, Associate Professor and Director of Cariology, Department of Community Dentistry, University of Texas Health Science Center at San Antonio.

Antidepressants, which are frequently used to treat anorexia nervosa, usually cause xerostomia, and patients with xerostomia are more susceptible to developing carious lesions. Risk for dental caries may also be increased in patients whose bingeing episodes frequently involve high-calorie, high-carbohydrate foods. As a general rule, however, patients with eating disorders who primarily practice restriction or self-induced vomiting do not experience an increase in caries rates.[29]

Prevention

Nearly all dental caries is preventable with the use of good personal health habits (proper diet and nutrition, oral hygiene, and avoiding substance use). Daily oral hygiene is essential for the removal of harmful microorganisms and maintenance of oral health. Soldiers must be trained in proper field oral hygiene. Brushing after meals with fluoride toothpaste is the most effective caries prevention method. Soldiers who have been diagnosed with GERD should attempt to maintain or reestablish medication and dietary restrictions.

To prevent tooth decay, soldiers must use combat stress control techniques to help cope with the stresses of deployment, rather than relying on harmful dietary habits. Techniques such as talking, exercise, quick relaxation, deep relaxation, and cognitive exercises can all be used to relieve combat stress without harming oral health.[34,35]

Use of gum or mints that contain xylitol as the first ingredient, three to five times a day, between meals or after snacks also prevents dental decay.[36] Xylitol is a naturally occurring sweetener found in fruits, vegetables, and some other plants. It has about half the calories of sugar but is just as sweet. Xylitol prevents harmful bacteria from using starchy or sugary food particles to create acids that cause dental caries. It works synergistically with fluoride to prevent decay and promote remineralization of damaged tooth structure. Xylitol gum is distributed in theater dining facilities in the accessory pack of the MRE (meal,

ready-to-eat). Chewing xylitol gum for about 5 minutes regularly after meals and snacks should help prevent decay.

For both oral health and weight management, free sugar intake from foods and beverages should be less than 10% of energy intake (less than 41 g/day).[37] On average, people who consume over 55 grams of sugar a day are at high risk for dental caries. The frequency of free sugar intake should be less than five times a day. Cariogenic foods and beverages can be combined with protective foods (cheese, tea, protein, high-fiber foods, etc) to prevent dental caries.[38] Soldiers should be encouraged to drink fluoridated water for hydration between meals, and limit consumption of foods and drinks that contain harmful polybasic organic acids to mealtimes. Soldiers can also mitigate the effects of foods and beverages that have a high organic-acid or refined-carbohydrate content by rinsing their mouths with water after ingestion.

Patients who experience xerostomia are at increased risk for dental caries, and should be advised to use artificial saliva preparations to lubricate the oral tissues, avoid cariogenic foods or drinks, and use sugarless or xylitol-containing candies or mints. Sucking on these items can stimulate increased salivary flow, which may buffer oral acids. Increased saliva flow also increases the concentration of calcium, phosphate, and hydroxyl ions, which may aid remineralization of early carious lesions. Xylitol may also be beneficial because of its bacteriostatic properties.

Treatment

Patients should be referred to a dentist for treatment of dental caries. Soldiers who experience recurrence of GERD symptoms for any reason should be seen by a dentist as soon as possible. To prevent chemical damage to dental hard tissues by stomach acids, dentists may fabricate a thin plastic stent that covers the dentition. The stent is to be worn at night or at times when reflux symptoms are most likely to occur. Early referral of eating-disorder patients to a psychiatric therapist can reduce the risk of further damage to the teeth and the oral cavity. Patients should be referred to a dentist for an evaluation of dental erosion, salivary flow rate, and oral mucosa condition. Comprehensive dental procedures should not be performed until vomiting behavior is significantly improved or the patient has recovered completely; until then, proper home care is the best treatment.

All patients whose oral cavity is exposed to gastric acid should be counseled on several important principals of home care[29,33]:

- Toothbrushing should never be performed immediately after the mouth is exposed to stomach acid because demineralized enamel has been shown to be vulnerable to removal by abrasive forces.
- Following acid exposure, patients should rinse with a buffering or alkaline solution to neutralize the acid and allow the saliva to remineralize the teeth, thereby reducing damage from demineralization. Options include 0.5% sodium fluoride sodium bicarbonate in water, liquid antacids, slightly alkaline mineral water, or plain water.
- If acid exposure happens repeatedly on a daily basis, a neutral sodium fluoride mouth rinse or prescription fluoride gel may be necessary to prevent dental erosion.
- Acidic foods and drinks should be avoided, including citrus, pineapple, and lemon candies; apple or citrus fruit juice; alcohol (particularly white wine); and drinks that contain polybasic organic acids such as citric, phosphoric, malic, and tartaric.[10,11]
- Patients who experience xerostomia should be advised to use artificial saliva preparations to lubricate the oral tissues, avoid cariogenic foods or drinks, and use sugarless or xylitol-containing candies or mints. Sucking on these items can stimulate increased salivary flow, which may buffer oral acids.

STRESS AND PERIODONTAL DISEASES

Gingivitis

Neglect of oral hygiene is common in stressful operational environments. Failure to properly remove plaque from the teeth and gums for a week or more usually results in the development of gingivitis in response to bacteria (Figure 17-9). The previously mentioned evaluation of soldiers deployed to Iraq showed that these service members experienced a significant worsening of their average periodontal screening and recording scores after 6 months of deployment.[19] If left untreated, gingivitis associated with tenderness and bleeding gums may discourage soldiers from performing normal oral hygiene.

Acute presentations of gingivitis can be managed by improved oral hygiene and the use of an antimicrobial mouth rinse. Soldiers with extreme gingival tenderness may be encouraged to reestablish oral hygiene proce-

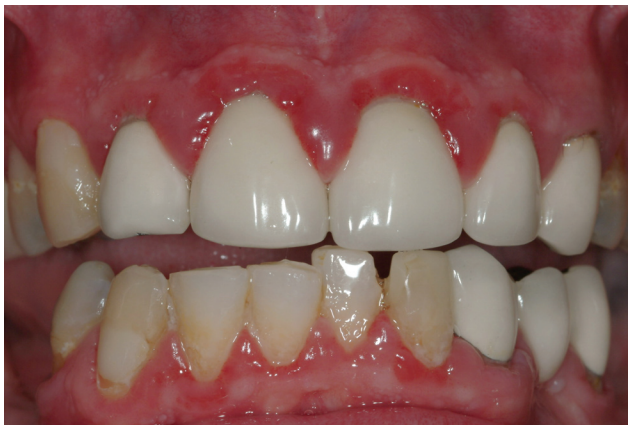

Figure 17-9. A case of gingivitis.
Photograph: Courtesy of Colonel Dave Reeves, US Army Dental Corps, Consultant to The Surgeon General for Periodontology, and Chief, Periodontics, Fort Hood, Texas.

dures using viscous lidocaine on their toothbrushes rather than a dentifrice.

Periodontitis

Untreated gingivitis may progress to periodontal disease (Figure 17-10). The risk of periodontal disease has been studied for both men and women (Figure 17-11), and several risk factors have been identified,[39–42] such as gram-negative anaerobic bacteria, smoking, diabetes mellitus, a genetic tendency to produce increased levels of proinflammatory mediators, and use of hormone-mimicking medications.

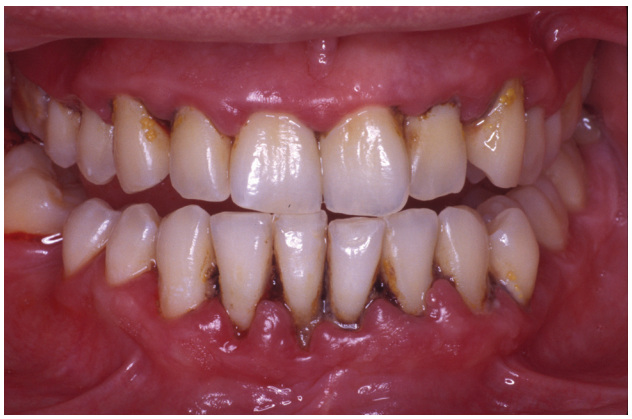

Figure 17-10. Gingival erythema, edema, and recession in conjunction with blunting of the interdental papilla are evident in this patient with periodontal disease.
Photograph: Courtesy of Lieutenant Colonel Georgia dela Cruz, US Army Dental Corps, Falls Church, Va.

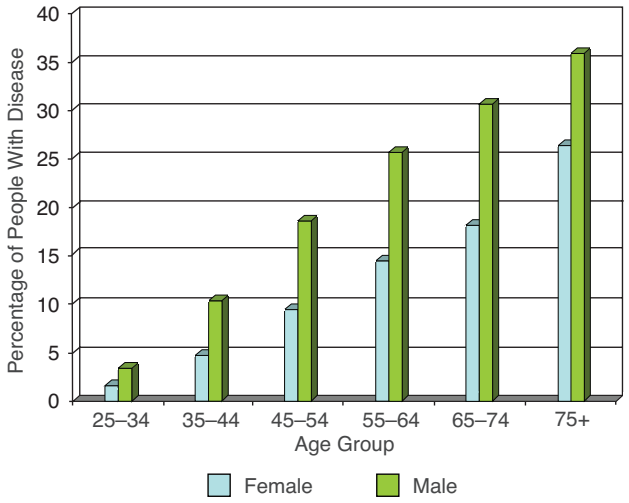

Figure 17-11. Prevalence of periodontal disease. Males are more likely than females to have at least one tooth site with 6 mm or more of periodontal loss of attachment.
Adapted from: US Department of Health and Human Services. *The Surgeon General's Report on Oral Health*. Washington, DC: USDHHS; 2000: 65.

Periodontitis has been described as "a complex disease in which disease expression involves intricate interactions of the oral biofilm with the host immunoinflammatory response and subsequent alterations in bone and connective tissue homeostasis."[43(p1560)] Classification of periodontal disease is based on two clinical factors: inflammation and loss of supporting tissue.[44] Inflammation features typically seen are redness, edema, and bleeding on probing. Loss of supporting tissue is usually measured as increased probing depth, decreased attachment level, or alveolar bone loss. Even with the presence of periodontal pathogens, the pathogenesis of periodontitis cannot occur without immune and inflammatory responses, which are in turn shaped by both intrinsic (eg, genetics, age, systemic disease) and extrinsic (eg, toxins, tobacco, plaque) host factors[45] (Figure 17-12). How much bone loss occurs depends largely on the level of inflammatory mediators present in gingival tissue.

Alveolar bone destruction occurs after activation of the pathogenesis pathways and the penetration of inflammatory mediators deep into gingival tissue, near alveolar bone. Several proinflammatory cytokines are responsible for bone resorption, such as interleukin-1, -6, -11, and -17; tumor necrosis factor-α; leukemia inhibitory factor; and oncostatin M.[45] When an inflammatory response occurs, periosteal osteoblasts are stimulated by proinflammatory cytokines and other mediators. Once the osteoblasts are stimulated,

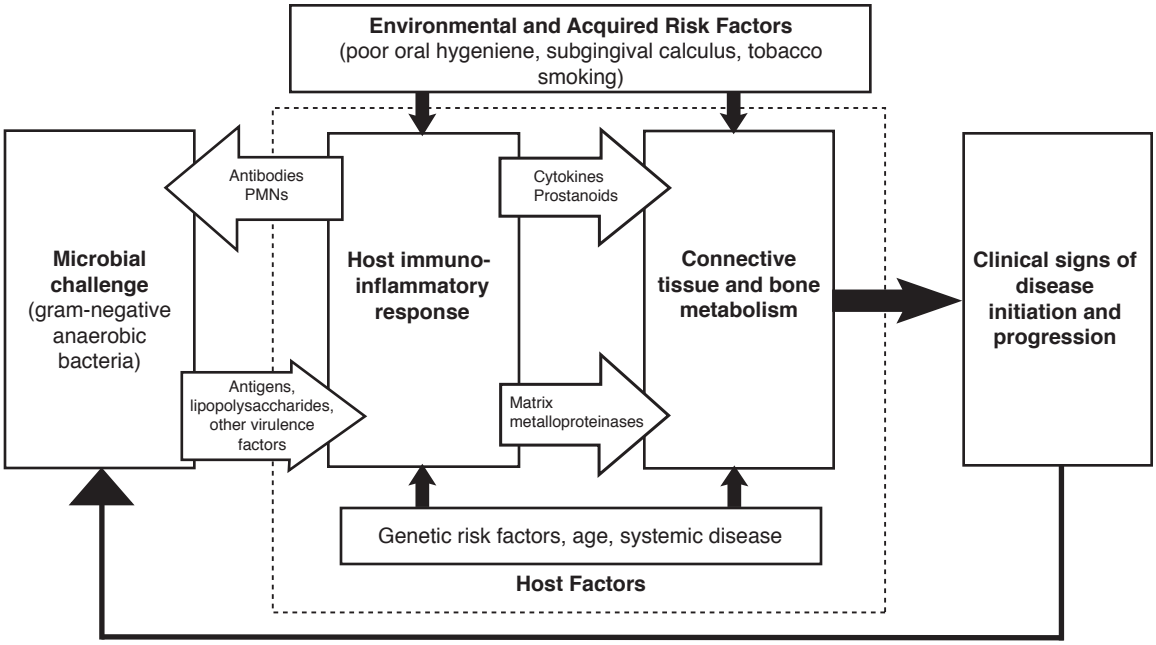

Figure 17-12. Model of periodontitis depicting the multifactorial nature of periodontal disease. A change in any one factor can result in clinical signs of the disease.
PMN: polymorphonuclear leukocytes
Data sources: (1) Page RC, Kornman KS. The pathogenesis of human periodontitis: an introduction. *Periodontology 2000*. 1997;14:9–11. (2) US Department of Health and Human Services. *The Surgeon General's Report on Oral Health*. Washington, DC: DHHS; 2000. (3) Gluck G, Morganstein W. *Jong's Community Dental Health*. 5th ed. St Louis, Mo: Mosby; 2003: 185–187.

changes in the overall cell surface occur that increase the expression of a protein called "receptor activator of nuclear factor-κ B ligand (RANKL)."[45] RANKL can be found on the surface of many other cells, including fibroblasts, T lymphocytes, and B lymphocytes. In the noninflammatory state, there is a balance between osteoclastic and osteoblastic processes determined by the proportion of RANKL. However, during an active inflammatory response, the proinflammatory mediators increase the expression of RANKL while at the same time decreasing other surface protein production in osteoclast precursor cells, triggering the formation of mature osteoclasts. Alveolar bone resorption by these osteoclasts leads to destruction of the supporting structures of the teeth, which, in the absence of intervention, leads to tooth loss.[45]

Women's risk of developing gingival inflammation or periodontal problems may be increased by the higher levels of estrogen and progesterone associated with the menstrual cycle and use of oral contraceptives.[46,47] Gingival tissues may become tender and swollen, and may bleed during brushing. Human gingiva has specific high-affinity estrogen receptors and can function as an estrogen target tissue. The stratified squamous epithelium of the oral mucosa and gingiva responds to ovarian hormone levels, with alterations in maturation and keratinization. Estrogen is involved in the regulation and maintenance of collagen synthesis and has been associated with gingival hyperplasia.

Estrogen and progesterone also promote changes in the microcirculatory system of the gingiva. The endothelial cells and pericytes of the venules swell, granulocytes and platelets adhere to the vessel walls, microthrombi form, and perivascular mast cells are disrupted.[46] The microvasculature proliferates and becomes more permeable, causing gingival edema and increasing the flow of gingival crevicular fluid. The resulting fluid also contains elevated levels of sex hormones, polymorphonuclear leukocytes, and increased levels of prostaglandin E_2. Anaerobic bacteria (eg, *Bacteroides melaninogenicus, Prevotella intermedia*, and *Porphyromonas gingivalis*) may be present and proliferate under these conditions.

The increase in bacteria production is a result of two factors: (1) some bacteria associated with gingival inflammation are able to metabolize steroid hormones and use them for energy production, thereby directly increasing their numbers; and (2) increased estrogen and progesterone levels also depress T-cell responses

and decrease neutrophil chemotaxis and phagocytosis. This impaired immune response allows bacteria in the gingival crevice to proliferate without restraint.

Proliferating bacteria can cause increased levels of bacterial endotoxins, which can increase inflammation and trigger bone loss from the periodontium. Oral contraceptives, especially those containing progesterone, have been associated with an increased risk of periodontal bone loss.[47,48] No studies have been done on Depo-Provera (medroxyprogesterone acetate [the Upjohn Company, Kalamazoo, Mich]) and periodontal bone loss.

Necrotizing Ulcerative Gingivitis

When soldiers are placed under severe stress, increased cortisol levels can compromise immune function. Under these conditions, gingivitis can progress to acute necrotizing ulcerative gingivitis, an extremely painful inflammation accompanied by necrosis of the interdental gingiva and a fetid odor (Figure 17-13).

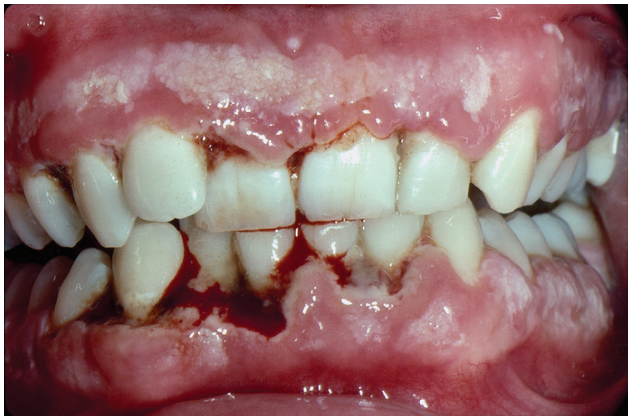

Figure 17-13. Gingival edema, erythema, and cratering of the interdental papilla and purulent exudate are visible in this patient with acute necrotizing ulcerative gingivitis. Photograph: Courtesy of Dr Carl Allen, Professor and Director, Oral and Maxillofacial Pathology, Ohio State University College of Dentistry.

Prevention

Noncommissioned officers should ensure that the oral hygiene routine of all troops includes the following:

- toothbrushing, once daily at a minimum, preferably twice daily, with fluoride toothpaste to prevent dental caries and gingival problems;
- flossing daily, which is also effective in preventing gingival or periodontal problems; and
- rinsing several times a week with an antimicrobial mouthwash containing thymol or chlorhexidine gluconate (for those with chronic periodontal conditions).

Treatment

Acute presentations of necrotizing periodontitis should be referred to a dental professional. In addition to reestablishing oral hygiene procedures with viscous lidocaine hydrochloride and an antimicrobial mouthwash, acute periodontal disease usually requires the removal of plaque-retentive factors, such as calculus or defective restorations, from the crown and root surfaces of the teeth.

TEMPOROMANDIBULAR DYSFUNCTION

Stress can produce temporomandibular dysfunction (TMD) symptoms when it causes patients to clench or brux their teeth more frequently, either at night or during the day. Masticatory muscle spasms and pain may result. Other known causes of TMD symptoms include injury to the temporomandibular joint from blunt force to the face, arthritis, joint overload, or repetitive loading (usually because of bruxism or grinding of the teeth).

Although the overall incidence of the condition is low, TMD affects both men and women. According to a 1994 triservice recruit comprehensive oral health survey,[49] approximately 3.5% of female recruits were found to have some type of orofacial pain or limited mandibular movement sufficient to require referral or treatment for TMD. The incidence for women was significantly higher than that for men (1.5%). Civilian studies[50,51] have consistently shown an increased incidence of TMD (1.5- to 2-fold higher) in women compared with men, and most patients treated for TMD are women (80%). Age plays a strong role in women. Symptoms begin after puberty and peak during the reproductive years, with prevalence highest among women aged 20 to 40. Gender and age distributions of TMD expression strongly suggest a link to the female hormonal system. Some studies[51–53] have shown that women who use oral contraceptives may be at increased risk compared with women who do not. TMD pain levels increase during menstruation.[54,55] Current studies investigating the relationship of increased risk with the presence of estrogen receptors in the temporomandibular joint structures (particularly the synovial

lining cells, the articular disc, and the chondrocytes) have produced contradictory results.[56,57]

Prevention

TMD can be prevented by avoiding opening the mouth too wide during eating or yawning, and avoiding frequent, prolonged masticatory activity associated with pervasive oral habits such as jaw clenching, gum chewing, cheekbiting, or nailbiting.

Treatment

Acute open lock (an internal joint derangement that prevents closing the mouth) can cause extreme pain and should be immediately referred to a dental provider for reduction of the dislocation. If the provider is unable to reduce the dislocation due to severe muscle spasm or extreme pain, then the patient must be referred to an oral and maxillofacial surgeon for sedation prior to reduction of the dislocation. An acute closed lock (where the mouth is prevented from opening) warrants an immediate referral for evaluation by an oral and maxillofacial surgeon or an orofacial pain specialist because early intervention beyond conservative therapy (such as arthrocentesis) may be necessary. For all other cases, conservative therapy for an acute condition should focus on reducing joint loading and inflammation. Attempts should be made to relax the muscles as much as possible. The patient should use ice or cold packs for the first 24 to 48 hours, adhere to a soft diet, and avoid gum chewing. After the first 24 to 48 hours, the patient should apply moist heat several times a day. Gentle stretching exercises of the mastication muscles should be performed after heat application. The patient should continue to avoid gum chewing and adhere to a soft diet for several days to limit masticatory activity.

Pharmacological interventions should begin as soon as possible with analgesics, preferably nonsteroidal antiinflammatory analgesics. Patients with more severe TMD may also require short-term use of skeletal muscle relaxants. Because low-dose tricyclics improve sleep, they may be effective in decreasing pain from nocturnal bruxism. If symptoms recur, the patient should initiate moist heat application, stretching therapy, and use of nonsteroidal antiinflammatory drugs. Normally, this treatment prevents the development of more severe problems.

Ideally, TMD patients should be referred to a dentist for a comprehensive evaluation of contributing factors from diet, occlusion, oral habits, and stress. However, because no national standards exist for TMD curricula at the predoctoral level, finding a knowledgeable practitioner may be difficult.[58] Initial evaluation for acute TMD pain from trauma must rule out fractures, tears, and articular disc displacement. Examination of the occlusion and oral habits may show whether the patient would benefit from wearing an appliance (eg, a bite plate) every night to prevent or minimize the effects of jaw movements during sleep.

If symptoms do not resolve, patients should be referred to an orofacial pain specialist, who can determine if the patient would also benefit from cognitive-behavioral skills training and biofeedback (eg, relaxation techniques) to decrease muscle jaw tension, decrease stress, increase awareness, and prevent diurnal tooth grinding or clenching incidents. Studies have shown that early intervention using a biopsychosocial approach is effective in reducing TMD pain and decreasing the progression of the disorder to chronic stages, as well as decreasing depression, increasing positive coping behaviors, and significantly reducing TMD-related healthcare costs.[59,60]

SUMMARY

The stress of deployment can cause serious oral health problems. Soldiers should be trained in stress-reducing techniques to avoid these problems (as well as the many other negative effects of combat stress). However, once problems occur, they should be treated with self-care techniques; more serious conditions necessitate attention from oral care practitioners.

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