A proposed categorization of carious lesions for the purpose of conservative management places lesions into three categories: lesions where no treatment is advised, lesions where preventive care is advised, and lesions where restorative treatment is advised (Pitts and Longbottom 1995). This approach, using caries as an infectious disease paradigm, resulted in a marked reduction of operative procedures in Danish schoolchildren (Thylstrup et al. 1995) and has been proposed as a means to preserve tooth structure and maximize appropriate care in the United States (Ismail 1997).

New imaging and laser technologies are emerging as tools for early diagnosis and prompt treatment of dental caries. For example, quantitative light-induced fluorescence is showing promise (de Josselin de Jong et al. 1996) for dental caries diagnosis. Two different methods, the quantitative infrared laser fluorescence method and electrical conductance measurements, are currently commercially available. At present, these methods are being used to augment conventional diagnostic tools but are not yet part of routine practice. However, they could potentially be used for close monitoring of the lesions and for patient motivation (Angmar-Månsson et al. 1996). Laser treatments for soft tissue surgery have been used in dentistry in recent years. Currently, in vitro studies are under way for the application of lasers for hard tissues, specifically to prevent dental caries by altering tooth mineral and inhibiting progression of artificial caries-like lesions (Featherstone et al. 1998, Kantorowitz et al. 1998).

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

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

The evidence base for the survival of the endosseous dental implants, an implant that is placed directly into a tooth socket, is extensive and has been recently reviewed (Cochran 1996, Fritz 1996). The predictability of endosseous dental implants in fully and partially edentulous patients has been clearly demonstrated in longitudinal studies (Albrektsson 1988, Albrektsson et al. 1988, Buser et al. 1991, Spiekermann et al. 1993). Many implant designs and surfaces have shown high success rates (often exceeding 95 percent in good-quality bone and 85 percent in poorer-quality bone, such as the posterior maxilla) (Buser et al. 1988, Cochran 1996, Fritz 1996).

Rehabilitation of lost tooth structure or even the whole tooth itself may be revolutionized in the next century, based on discoveries of the natural repair and regeneration mechanisms the body uses. The new sciences of biomimetics and tissue engineering combine engineering principles and materials science with rapidly growing knowledge of the progenitor cells and molecules that give rise to specific tissues such as skin, bone, teeth, and cartilage. Already it is possible to generate new cartilage and bone of a prescribed shape to replace tissue lost from injury or disease (Reddi 1995). Eventually, it may be possible to use a patient's own oral cells and cell products to generate new tooth enamel, dentin, and cementum for the natural repair of carious lesions.

**Periodontal Diseases**

Periodontal diseases are caused by microbial infections, and are plaque-related complex diseases like dental caries, presenting as several clinical variants...
(see Chapter 3). The mildest form is gingivitis, characterized by inflammation of the gingiva with a marked loss of gingival collagenous material (Page and Schroeder 1976, Schroeder et al. 1973). In a more advanced disease, periodontitis, there is involvement of the soft tissue and bone that support the teeth. If untreated, periodontitis may progress and result in abscesses, mobile teeth, and tooth loss. Periodontitis also may be associated with certain systemic diseases and conditions (see Chapter 5).

Gram-negative anaerobic bacteria in plaque are implicated as causative agents in periodontitis. However, host immune system factors, specifically, a chronic inflammatory response, are now considered to be the primary determinants of disease progression and outcome (Page 1998). The disease process is very similar across the different types of periodontal disease and involves interactions between infectious agents and their virulence factors and host defense mechanisms, operating within a context of environmental, acquired, and genetic risk factors specific to a given individual. Figure 8.5 illustrates the pathogenesis of these diseases (Page and Beck 1997).

**Risk Assessment.** Sufficient knowledge of demographic and systemic risk factors and indicators has been acquired to guide clinical decisions in the management of periodontal diseases (Genco 1996, 2000, Page and Beck 1997, Papapanou 1998). Table 8.7 provides an overview of the strength of the associations of local and systemic factors with destructive periodontal diseases (Genco 1996, 2000). Table 8.8 presents the odds ratios derived from studies that investigated the likelihood of developing periodontal disease given a specific risk factor, indicator, or marker/predictor (Jeffcoat et al. 1997, Page and Beck 1997). The presence of pathogenic bacteria, poor oral hygiene, tobacco smoking, diabetes mellitus, and preexisting periodontal disease are some of the factors that contribute to the likelihood of disease presence, progression, and treatment outcomes.

A systematic identification of risk factors, indicators, and predictors has been proposed as the first step in diagnosing and managing periodontal diseases (Genco 1996, Page and Beck 1997, Papapanou 1998). Clinicians can weigh the known risks for individual patients and devise treatment plans appropri-

---

**FIGURE 8.5**

A new paradigm for the pathobiology of periodontitis

![Diagram](http://example.com/image.png)

### TABLE 8.7
The strength of association of local and systemic factors with destructive periodontal disease

<table>
<thead>
<tr>
<th>Factor</th>
<th>Case Report Studies</th>
<th>Case-Control Studies</th>
<th>Cross-sectional Studies</th>
<th>Longitudinal Studies</th>
<th>Intervention Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific bacteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P. gingivalis</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>B. forsythus</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>P. intermedia</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>Yes</td>
<td>NR</td>
<td>Yes</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Age</td>
<td>Yes</td>
<td>Yes</td>
<td>No (to 7th decade)</td>
<td></td>
<td>NR</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 2</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes (treatment reduces glycosylated hemoglobin)</td>
</tr>
<tr>
<td>Type 1</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
<td>Yes</td>
<td>NR</td>
</tr>
<tr>
<td>Smoking</td>
<td>NR</td>
<td>Yes</td>
<td>Yes</td>
<td>NR</td>
<td>Yes (smokers heal poorly)</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Stress, distress, coping</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Polymorphonuclear disorders</td>
<td>Yes</td>
<td>Yes</td>
<td>NR</td>
<td>Yes (case series)</td>
<td>NR</td>
</tr>
<tr>
<td>Genetic factors (IL-1 polymorphisms)</td>
<td>NR</td>
<td>Yes</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Dietary calcium</td>
<td>NR</td>
<td>Yes</td>
<td>Yes</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Preexisting periodontal disease</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Note: NR = not reported, or not relevant.


### TABLE 8.8
Risk of periodontal disease

<table>
<thead>
<tr>
<th>Strength of Association With</th>
<th>Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographic characteristics</td>
<td></td>
</tr>
<tr>
<td>Age, 35-44 years</td>
<td>alveolar bone loss 7.60</td>
</tr>
<tr>
<td>Age, 65-74 years</td>
<td>alveolar bone loss 24.08</td>
</tr>
<tr>
<td>Risk factors</td>
<td></td>
</tr>
<tr>
<td>Smoking, light</td>
<td>periodontal disease 2.05</td>
</tr>
<tr>
<td>Smoking, heavy</td>
<td>periodontal disease 1.48</td>
</tr>
<tr>
<td>Poor oral hygiene</td>
<td>periodontal disease 4.75</td>
</tr>
<tr>
<td>P. gingivalis</td>
<td>periodontal disease 7.28</td>
</tr>
<tr>
<td>A. actinomycetemcomitans</td>
<td>periodontal disease 2.50</td>
</tr>
<tr>
<td>Clinical measurement</td>
<td></td>
</tr>
<tr>
<td>Bleeding on probing</td>
<td>progression of periodontitis 2.7</td>
</tr>
</tbody>
</table>


Most recently, putative genetic markers for susceptibility for oral disease have been studied. In particular, a specific genotype of the polymorphic IL-1 gene cluster has been shown to be associated with severe periodontitis in nonsmokers (Kornman et al. 1997). IL-1β is of interest because the proinflammatory cytokines are key regulators of the host immune response to microbial infection and extracellular matrix catabolism and bone resorption. Functionally, this polymorphism is associated with high levels of IL-1 production, and high levels of IL-1 have been associated with progressive periodontal breakdown (Cavanaugh et al. 1998).

A consensus has been reached by a specialty organization that all patients in general and specialty care should be screened for periodontal disease (AAP 1996). The recommended approach is to apply the Periodontal Screening and Recording examination (PSR). Related screening tests include the Community Periodontal Index of Treatment Needs (CPITN) (Ainamo et al. 1982) and the Basic Periodontal Examination.

**Diagnosis.** The strengths and weaknesses of the range of tests and methods used to diagnose periodontal
diseases are presented in Table 8.9. Most diagnostic tests for periodontal diseases rely on a physical examination to note any swelling, redness, gingival bleeding, or tooth mobility. Periodontal probing, radiographs, and microbiologic and histological examinations of biopsied tissue provide important additional information. These tests indicate the presence, extent, and severity of gingival and periodontal tissue destruction; they do not indicate the cause of disease or whether it is quiescent or actively progressing.

Gingival inflammation may be assessed using a variety of methods, including bleeding on probing and the use of indices such as the gingival index (Loe and Silness 1963) to grade redness and bleeding. In adult periodontitis, the absence of inflammation is associated with a lack of disease progression, but the presence of inflammation does not indicate inevitable progression to destruction (Armitage 1996, Halazonetis et al. 1989, Okamoto et al. 1988). Longitudinal studies have also been conducted in patients who participate in maintenance programs. The absence of gingival bleeding, especially at recall visits, has been shown to be a valid indicator of gingival health in these patients (Lang et al. 1986).

Measurement of probing depths (also termed pocket depths) is an integral part of the periodontal examination. Longitudinal studies have shown that shallow probing depths and minimal loss of attachment are associated with lack of disease progression. The mere presence of a pocket does not herald progressive periodontitis at that site. Although teeth with moderate to deep probing depths are at higher risk for additional destruction, a single examination cannot determine the fate of the tooth with certainty (Armitage 1996, Halajee et al. 1983, Halazonetis et al. 1989, Okamoto et al. 1988).

Radiographs are used to obtain a visual image of the bony support around a tooth or dental implant. They are an essential tool in planning complex prosthetic reconstructions, as well as a necessary diagnostic aid in assessing periodontal progression.

At least 15 different organisms have been associated with adult periodontitis. The 3 species most

<p>| TABLE 8.9 |
| Strengths and weaknesses of tests and methods used to diagnose periodontal diseases |</p>
<table>
<thead>
<tr>
<th>Application</th>
<th>Strengths</th>
<th>Weaknesses</th>
<th>Type of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Periodontal screening and recording (PSR)</td>
<td>All patients in every practice</td>
<td>Cost-effective, quick, easy; detects patients with periodontal disease.</td>
<td>Does not provide a tooth-by-tooth assessment for later comparison during maintenance. A full periodontal examination is needed for this purpose.</td>
</tr>
<tr>
<td>Probing pocket depths</td>
<td>All patients</td>
<td>Shallow probing depths are associated with lack of future disease progression.</td>
<td>Moderate to deep pockets in single probing depth examination will not distinguish with certainty which teeth will undergo progressive periodontal destruction.</td>
</tr>
<tr>
<td>Gingival inflammation</td>
<td>Assessed in all patients</td>
<td>Absence of inflammation is associated with a lack of future progression. In treated patients, bleeding on probing is associated with an increased risk for progressive loss of attachment.</td>
<td>Presence of inflammation will not distinguish with certainty which teeth will undergo progressive periodontal destruction.</td>
</tr>
<tr>
<td>Radiographic evidence of bone loss</td>
<td>At-risk patients as determined by PSR screening or periodontal examination</td>
<td>Absence of bone loss is associated with a lower risk of future progression.</td>
<td>Presence of bone loss on a single radiograph will not distinguish with certainty which teeth will undergo progressive periodontal destruction.</td>
</tr>
<tr>
<td>Microbial/plaque tests</td>
<td>High-risk or refractory patients</td>
<td>Absence of supragingival plaque is associated with lack of disease progression.</td>
<td>At this time, routine testing offers limited benefit in adult periodontitis.</td>
</tr>
<tr>
<td>Biochemical profiles in gingival crevicular fluid</td>
<td>Not yet determined</td>
<td>A number of biochemical markers may identify individuals at risk.</td>
<td>At present, there are no specific biochemical profiles that characterize specific periodontal diseases.</td>
</tr>
</tbody>
</table>
strongly linked are *Porphyromonas gingivalis*, *Bacteroides forsythus*, and *Treponema denticola*. *Actinobacillus actinomycetemcomitans* is most strongly linked to early-onset periodontitis (Haffajee and Socransky 1994). No single bacterial species has been shown to satisfy Koch’s postulates (Moore 1987, Socransky and Haffajee 1992), leading some investigators to suspect that periodontitis is a mixed infection (Ranney 1993). As a result, diagnostic tests for periodontal diseases have included assessments of the presence and amount of several putative microbes in the subgingival plaque.

Routine bacterial testing of patients with adult periodontitis is not usually necessary and indeed is not supported by the preponderance of the evidence (Armitage 1996, AAP 1996). In formulating treatment programs for special patient populations and as a research tool, however, the tests can be very helpful. Such patients include those refractory to previous therapy, patients with rapidly progressive or early-onset periodontitis, and certain medically compromised patients.

The traditional method for assessing the subgingival flora is by culturing samples extracted from the site of infection. Culturing allows the clinician to determine the antibiotic sensitivity of the organisms, but it is technique-sensitive: scrupulous care is required when sampling the periodontal pocket. This is especially true for microbes that are strict anaerobes, because they are killed by even brief exposure to air. The requirement that bacteria have time to grow also precludes chairside testing.

With the advent of molecular biology, bacterial species can be identified by their DNA (Moncla et al. 1988, Savitt et al. 1988, 1990) or by unique antigenic components (Zambon et al. 1986). Either method will detect putative periodontopathic bacteria quickly and with a high degree of sensitivity and specificity, usually above 90 percent. The tests do not indicate whether there is actual disease, however. Nor do the tests reveal anything about the antibiotic sensitivity of the detected bacteria. Because DNA is very stable, the tests can be applied to nonliving plaque samples, simplifying the collection process. Kits are available that allow DNA testing to be performed in the dental office; otherwise the samples are sent to a reference laboratory.

Other tests are available for the detection of groups of putative periodontopathic bacteria (Loesche 1986). The BANA test detects a trypsin-like enzyme that is present in *P. gingivalis*, *T. denticola*, and *B. forsythus* (Loesche et al. 1990). Somewhat less accurate than the tests described above, the BANA test is 92 percent sensitive and 70 percent specific in detecting these groups of bacteria.

Once a periodontal infection is established, telltale metabolic changes occur in the body as a result of inflammation, injury, or death of tissue. A sample of fluid exudate from the gingiva (gingival crevicular fluid) in an affected pocket can be analyzed for these changes. They include elevated levels of prostaglandin E₂ (Cavanaugh et al. 1998, Offenbacher et al. 1986), interleukin 1 and interleukin 6 (Cavanaugh et al. 1998, Geivelis et al. 1993, Masada et al. 1990, Tsai et al. 1995), tumor necrosis factor (Rosciano et al. 1990), β-glucuronidase (Lamster et al. 1994, 1995), aspartate aminotransferase (Chambers et al. 1991, Persson and Page 1992), elastase (Armitage et al. 1994, Palcanis et al. 1992), and collagenase (Lee et al. 1995). Most of these analyses are based on inserting a filter paper strip into the isolated pocket to collect the fluid and testing for the metabolite of interest. A positive result usually indicates that inflammatory or destructive pathways have been triggered, but provides no clues concerning the etiologic factor or factors. Because of differences in experimental designs in the clinical studies, it is difficult to compare the sensitivity and specificity of each metabolite in detecting disease.

**Prevention.** Because periodontal diseases are plaque-associated infections, prevention and management of the early signs of these diseases depend on effective plaque control. This can be accomplished using both mechanical and chemotherapeutic approaches (Table 8.10). The prophylaxis performed in the dental office on periodontally healthy patients reduces plaque and removes stains and calculus. How often patients should be recalled for such preventive procedures is based on an assessment of risk factors such as the patients age, oral hygiene, personal habits (e.g., smoking and diet), and a medical history indicating a heightened risk of infection (such as noted with diabetes or HIV infection) (Hancock 1996, Mealey 1996).

Chemical plaque control has become an important part of the clinician’s armamentarium and may be prescribed for patient care at home (Table 8.10). Reviews of the literature by Hancock (1996) and Drisko (1996) provide detailed supporting evidence. Significant reductions in gingival inflammation have been demonstrated for chlorhexidine, triclosan copolymer when used in conjunction with a fixed combination of essential oils, and stannous fluoride. The magnitude of gingival inflammation reduction was greatest for chlorhexidine. The evidence supporting
these effects includes multiple randomized, double-blind, controlled clinical trials.

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

The concept of management of a patient's risk factors as part of treatment is reasonably well documented for individuals who smoke and those who are diabetic and may be important for other risk factors such as stress (Genco et al. 1999) and low dietary calcium (Nishida et al. in press). Several studies have shown that treatment of periodontal disease in smokers is not as successful as in nonsmokers (Grossi et al. 1996). Thus, the management of smoking as a risk factor will contribute to the success of periodontal therapy. Furthermore, it appears that treatment of diabetic patients with periodontal disease may require more intense therapy since several studies have shown that antibiotic therapy is successful not only in reducing periodontal disease, but also in reducing glycated hemoglobin (Grossi and Genco 1998).

Professional plaque removal typically employs scaling and root planing, in which hardened deposits of plaque and other debris are removed from the periodontal pocket and the tooth root surface is smoothed over. The effectiveness of scaling and root planing has been demonstrated repeatedly in longitudinal, cohort, and randomized clinical trials and was reviewed by Cobb (1996). Demonstrated benefits include decreased gingival inflammation, decreased probing depth, and facilitation of maintenance of clinical attachment level. The evidence indicates that similar results may be obtained with ultrasonic and sonic instruments as with manual instruments. Regardless of the methods used, meticulous attention to detail is required to achieve optimal results (Cobb 1996).

**TABLE 8.10**

<table>
<thead>
<tr>
<th>Periodontal diseases: mechanical therapy and chemotherapeutics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category of Treatment</td>
</tr>
<tr>
<td>-----------------------</td>
</tr>
<tr>
<td>Professional mechanical therapy—used in the treatment of gingivitis and periodontitis</td>
</tr>
<tr>
<td>Chemical plaque control with mouthrinses and dentifrices</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Irrigation</td>
</tr>
<tr>
<td>Sustained release antibiotics</td>
</tr>
<tr>
<td>Systemic antibiotics</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

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Topical administration of antimicrobial agents contributes to the control of gingival inflammation (Table 8.10). Supragingival irrigation (e.g., applying a jet of water under pressure) may be used as an adjunct to toothbrushing and has been shown to aid in the reduction of gingival inflammation. However, no clear substantial long-term benefits for the treatment of periodontitis have been shown if irrigation is applied subgingivally.

Surgical therapy is employed to provide access to root surfaces and bony defects for debridement and root planing. Surgery can facilitate regeneration, augment the gingiva, and promote root coverage (Table 8.11). It is also necessary in placing dental implants.

Palcanis (1996) reviewed the evidence regarding surgical therapy. The overall goal is to make plaque control easier for the patient, thereby reducing disease progression. Many surgical techniques are available. Extensive randomized clinical trials and longitudinal studies form the basis of the evidence for the efficacy of these procedures (Kaldhal et al. 1996, Knowles et al. 1979, Pihlstrom et al. 1983, Ramfjord et al. 1987). All procedures decrease pocket depth, and, with the exception of gingivectomy, all increase clinical attachment level. A caveat to be noted, however, is that procedures designed to reduce probing depth may increase gum recession, exposing the root and possibly compromising aesthetics. Thus, selection of a particular surgical procedure must always be based on the individual needs of the patient. Regardless of the approach selected, maintenance is important to long-term success.

Systemic administration of antibiotics, including the tetracyclines, metronidazole, spiramycin, and clindamycin, has been extensively studied and reviewed (Drisko 1996). The risk of generating antibiotic resistance in bacteria precludes the use of systemic agents in treating simple gingivitis (AAP 1996). Similarly, systemic antibiotics should not be used for the routine first-line treatment of common forms of adult periodontitis (AAP 1996, Drisko 1996). The preponderance of evidence from well-controlled, randomized, blinded clinical trials indicates that the agents do not offer sufficient benefit to overcome risks of either drug sensitivity or the emergence of antibiotic-resistant pathogens.

The situation is different in cases of aggressive forms of periodontitis, such as early-onset, rapidly progressive, or refractory periodontitis, which affect less than 10 percent of periodontitis patients. Randomized, double-blind clinical trials, as well as longitudinal assessments, indicate that the use of systemic antibiotics can slow disease progression in these patients (AAP 1996, Drisko 1996).

To circumvent the problems of systemic therapy, investigators have applied antimicrobial agents directly into the pocket. Antimicrobials incorporated into either resorbable and nonresorbable inter-pocket delivery systems have been studied in randomized, double-blind, controlled clinical trials and are now FDA approved and on the market (Goodson et al. 1991, Jeffcoat et al. 1998). When used as an adjunct to scaling and root planing, gains in clinical attachment level and decreases in probing depth and gingival bleeding were demonstrated. Because

<table>
<thead>
<tr>
<th>TABLE 8.11</th>
<th>Periodontal disease: selected surgical procedures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category and Goal</td>
<td>Procedures</td>
</tr>
<tr>
<td>Pocket therapy—provides access to root surfaces and bony defects, reduces probing depths, facilitates plaque control, and enhances restorative and cosmetic dentistry</td>
<td>Modified Widman Flap to provide access to roots and bony defects for debridement</td>
</tr>
<tr>
<td></td>
<td>Apically repositioned flap with or without bony recontouring</td>
</tr>
<tr>
<td></td>
<td>Gingivectomy</td>
</tr>
</tbody>
</table>

 Certain systemic tetracyclines, notably doxycycline, are safe and effective in low doses for prevention of bone loss associated with periodontitis. Doxycycline hyclate (20-mg capsule) is approved for twice-a-day use for up to 9 months for this indication. At these low doses, the doxycycline appears to reduce the elevated collagenase activity, rather than function as an antimicrobial.
these delivery systems are relatively new, there is a paucity of evidence addressing their long-term effectiveness.

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

Freeze-dried demineralized bone represents one of the most frequently used and well-studied bone graft materials in periodontics. Freeze-dried demineralized bone is an allograft material, harvested, prepared, and demineralized prior to grafting. The demineralization step is important because it retains the activity of bone morphogenetic proteins—compounds in the graft material found to be essential for new bone formation (Urist 1965, Urist and Iwata 1973). Case reports and controlled clinical trials have demonstrated the bone-forming potential of such material, with some variability in the amount of bone fill achieved (Garrett 1996). Because allografts are derived from donor tissues, proper collection, handling, and storage are...
TABLE 8.12b
Odds ratios for protective factors for oral and pharyngeal cancers

<table>
<thead>
<tr>
<th>Diet high in fruits, by quartiles of intake</th>
<th>Both Sexes</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>First (lowest intake)</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Second</td>
<td>0.6</td>
<td>0.6</td>
<td>0.6</td>
</tr>
<tr>
<td>Third</td>
<td>0.4</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>Fourth (highest intake)</td>
<td>0.4</td>
<td>0.4</td>
<td>0.4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Years since quit smoking cigarettes</th>
<th>Both Sexes</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never smoked</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>0 (never)</td>
<td>3.4</td>
<td>3.4</td>
<td>3.4</td>
</tr>
<tr>
<td>1 to 9</td>
<td>1.1</td>
<td>1.1</td>
<td>1.1</td>
</tr>
<tr>
<td>10 to 19</td>
<td>1.1</td>
<td>1.1</td>
<td>1.1</td>
</tr>
<tr>
<td>20+</td>
<td>0.7</td>
<td>0.7</td>
<td>0.7</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lip cancer: daily use of lip protection (mostly colored lipstick)</th>
<th>Both Sexes</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>≤1 per day</td>
<td>3.2</td>
<td>3.2</td>
<td>3.2</td>
</tr>
<tr>
<td>&gt;2 per day</td>
<td>7.3</td>
<td>7.3</td>
<td>7.3</td>
</tr>
</tbody>
</table>

Note: First row for each factor is the referent group. Dashes indicate not studied or too few to analyze.

*Blot et al. 1988.
*Pogoda and Preston-Martin 1996.

Essential to ensure viability and prevent contamination with viruses or other pathogens (Melloni et al. 1995).

Alloplasts represent a class of synthetic resorbable or non-resorbable graft materials. When evaluated in controlled clinical trials, they demonstrated improvements in probing depth and attachment level (Garrett 1996). Histology, however, indicates that, in general, synthetic grafts act primarily as space fillers, with little, if any, regeneration.

In the 1980s a number of investigators explored a procedure called guided tissue regeneration. The idea was to employ either a resorbable or non-resorbable membrane at the diseased site that would selectively allow passage of cells able to regenerate periodontal attachment apparatus and bone, while prohibiting migration of non-regenerative cells such as fibroblasts. The evidence for the efficacy of guided tissue regeneration ranged from randomized controlled clinical trials to case reports (Garrett 1996). Although less evidence is available for resorbable membranes than for non-resorbable membranes, significant improvements in clinical attachment levels have been shown compared to debridement alone. Most favorable results are reported for bone loss between the roots of mandibular tooth defects (Class II furcations). Less favorable results were reported in maxillary molar and Class III (through and through) furcation defects (Garrett 1996).

**Oral and Pharyngeal Cancers**

Oral and pharyngeal cancers, like other neoplastic diseases, are caused by mutations in cell regulatory genes. The mutations contribute to carcinogenesis by promoting uncontrolled cell growth, suppressing the function of tumor suppressor genes, promoting the growth of new blood vessels (angiogenesis) to nourish the growing tumor, or facilitating metastasis. A model depicting the genetic progression for oral and pharyngeal cancers has been proposed (Califano et al. 1996).

Potentially malignant lesions can present in a variety of ways and can include erythroplakia or leukoplakia (red or white flat lesions, respectively), ulceration, failure of a wound to heal, lymphadenopathy, induration, dysphagia, and tissue growth. Erythroplakia lesions are considered to have a higher rate of malignancy than leukoplakia (Silverman 1990). *Candida albicans* infection of a leukoplakia lesion appears to increase the risk of malignant transformation (Field et al. 1989, Scully 1995). In addition, infections with strains of human papillomavirus (HPV) and herpes simplex virus (HSV) have been implicated in the etiology of oral cancers. Other potentially predisposing factors include chronic iron deficiency anemia, erosive lichen planus, oral submucous fibrosis, and actinic keratosis.

**Risk Assessment.** Risk assessment for oral and pharyngeal cancers (Table 8.12a) includes an evaluation of the patient's exposure to tobacco and alcohol and an examination to identify suspicious lesions and conditions thought to predispose to cancer. All tobacco products (see Chapters 3 and 10) have been associated with oral and pharyngeal cancers. The risk of oral cancer is increased 6 to 28 times in current smokers. Alcohol is also a risk factor for oral cancer, and combined with tobacco use accounts for 75 to 90 percent of oral cancer in the United States (Blot et al. 1988, Vokes et al. 1993). A dose-response relationship has been demonstrated for cigar smokers, and the overall risk of cancer is 7 to 10 times higher among users than for those who never smoked (NCI 1998). In addition, individuals who have had oral cancer are at increased risk for a second primary...
cancer, and this risk is higher than that for other cancers (Boice et al. 1985, Winn and Blot 1985).

Other factors, such as infection with HPV or HSV, as noted earlier, and use of high-alcohol-content mouthwashes, also have been associated with oral cancers (Flaitz and Hicks 1998, Sugerman and Shillitoe 1997, Winn et al. 1991). Lip cancer is associated with exposure to the sun (Pogoda and Preston-Martin 1996).

Biomarkers—measurable alterations in molecules derived from human tissues or fluids—are being developed to identify those at risk for oral cancer and to identify which patients may benefit from specific treatments (Lippman et al. 1993, Patterson et al. 1996, Sidransky 1997). These markers may also be able to predict oral cancer recurrence or the occurrence of new primaries (Shin et al. 1996).

Diagnosis. At present, the diagnosis of oral and pharyngeal cancers involves a systematic extra- and intraoral physical examination to identify lesions and conditions that may be precancerous or indicate a predisposition to cancer (USDHHS 1998). Biopsies are essential to confirm the clinical observations. The sensitivity and specificity of a brush biopsy have been established in a multisite clinical trial (Sciubba 1999). Imaging technology may also be employed to determine the extent of the lesion. Although the sensitivity and specificity of the physical examination have not been established in clinical studies, it is clear that persons with localized oral and pharyngeal cancers have a better prognosis than persons whose cancers were detected with regional or distant spread (Ries et al. 1999). A thorough examination for oral and pharyngeal cancers is recommended by the American Cancer Society annually for individuals over 40 and for individuals who are exposed to known risks (Murphy et al. 1995).

Prevention. Primary prevention of oral and pharyngeal cancers involves avoiding known carcinogenic agents (Blot et al. 1988, Vokes et al. 1993), primarily tobacco in any form and excessive use of alcohol. In addition, the use of lip balms with ultraviolet radiation blockers is recommended (see Table 8.12b). A high dietary intake of fruits and vegetables may reduce oral and pharyngeal cancer risk by as much as 30 to 50 percent (McLaughlin et al. 1988). Dentists, physicians, and nurse practitioners among others are in a critical position to counsel patients on tobacco and alcohol use, pointing out that tobacco cessation lowers the risk for oral and pharyngeal cancers (Blot et al. 1988). Physicians, dentists, and other health care professionals have been shown to be effective in increasing tobacco cessation rates (Cohen et al. 1989, Oelke et al. 1991, Stevens et al. 1995, Wilson et al. 1988). A prime reference for use in national and international antismoking efforts is the Clinical Practice Guideline on Smoking Cessation (Number 180) developed by the Agency for Health Care Policy and Research (Fiore 1997).

Treatment. Diagnosis of cancer at an early stage can be followed by prompt and conservative treatment of the affected tissues. Some early lesions may be successfully treated with excisional biopsy, more advanced cases will require additional surgery, radiation, and/or chemotherapy (Shah and Lydiatt 1995, Vokes and Athanasiadis 1996). Preservation of function and appearance is emphasized. Advanced cancers require follow-up reconstruction and rehabilitation to improve function and aesthetics.

Birth Defects

There are hundreds of genetic diseases and syndromes as well as congenital anomalies that affect the craniofacial, oral, and dental tissues. However, some craniofacial anomalies may be spontaneous and manifest only at the time of birth. Chapter 3 describes a number of these disorders. Rapidly advancing knowledge of the genetics of development and of mutations associated with specific birth defects is aiding in the development of screening tests for genetic disorders and identifying high-risk individuals and families.

A complete diagnosis of the craniofacial disorder may involve a multidisciplinary team of experts in imaging, genetics, and other areas. Similarly, long-term management of the disorder, often extending to adulthood, generally calls for a team of specialists, including physicians and dentists, surgeons, nurses, rehabilitation experts, speech pathologists, psychologists, and social workers. Quality of life considerations, including social and psychological effects of birth defects such as cleft lip and palate, are taken into account (see Chapters 3 and 6).

Prevention. Primary prevention involves minimizing exposure to known teratogens, and genetic counseling as appropriate. The importance of educating parents or potential parents on behavioral risk factors, especially tobacco and alcohol use, the teratogenic potential of certain prescription drugs, and the need for adequate nutrition in the perinatal period is emphasized. In a study by Tolarova and Harris (1995), supplementation of the diet by multivitamins and folic acid during the periconceptional period (i.e., before, during, and after conception) markedly
diminished the occurrence of cleft lip and palate in a high-risk group. Unfortunately, only about 29 percent of women of childbearing age consume recommended amounts of these essential nutrients (Werler et al. 1999). The evidence associating moderate to severe periodontal disease in pregnant women with low-weight preterm births warrants attention to the importance of maintaining optimal oral health in pregnancy. The oral care clinician can contribute to birth defect prevention not only by treating oral disease, but also by providing educational messages to patients to promote the birth of healthy, full-term babies.

Treatment. A number of birth defects may not be apparent at birth because they are not manifested until later in development. One example is the ectodermal dysplasias (EDs), disorders characterized by abnormalities of skin, hair, sweat glands, and teeth. Dentists are essential in the management of care for children with these disorders, who must be repeatedly fitted with dentures throughout childhood. More recently, clinical studies have demonstrated that fitting ectodermal dysplasia patients as young as 12 years old with dental implants not only is effective, but also provides greater functional utility and satisfaction (Guckes et al. 1998, Kearns et al. 1999). As with other complex craniofacial anomalies, management by a multidisciplinary team is the best approach, with experts able to advise on the various oral, skin, and sweat gland complications.

Mutations have recently been identified for several forms of ED, including the anhydrotic form (absence of sweat glands). Ultimately, the development of genetic diagnostic tests can confirm the diagnosis in the child and permit counseling of parents.

Chronic Craniofacial Pain and Sensorimotor Conditions
A variety of problems involving pain and other sensorimotor abnormalities affect the craniofacial complex. These conditions can include burning mouth syndrome, trigeminal neuralgia, various facial palsies, postherpetic neuralgia affecting branches of the trigeminal nerve, temporomandibular disorders, fibromyalgia, and disorders of taste and olfaction. Some of these are infectious in origin (e.g., postherpetic neuralgia and some taste disorders); some are traumatic (e.g., some cases of temporomandibular disorder); and for others, the cause or causes are unknown (see Chapter 3). Patients with facial palsies and trigeminal neuralgia are generally referred to neurologists for treatment. Disorders of taste and smell also require neurological consultation as well as brain imaging because they can be symptomatic of brain tumors.

Pain relief may also improve function and can be combined with adjunctive measures such as the use of hot or cold compresses and behavioral treatments such as relaxation and imaging therapy to reduce muscle tension. The variety of pain medications has greatly increased in recent years. They include aspirin and other nonsteroidal anti-inflammatory drugs, tricyclic antidepressants, new antiepileptic drugs, the selective serotonin re-uptake inhibitors, and the more potent opiate family of drugs.

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

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

Temporomandibular Disorders
Among the common types of craniofacial pain likely to be seen by oral care providers are temporomandibular disorders, characterized by symptoms of pain and dysfunction in and around the temporomandibular joints or the masticatory muscles.

Temporomandibular disorders may occur as a result of injury, arthritis, or fibromyalgia or for unknown reasons. Approaches used to obtain a differential diagnosis of these conditions can range from a physical examination that may include palpation and measuring the mouth opening, to the use of complex imaging and instrumentation, including procedures such as arthroscopy (Table 8.13) (Clark et al. 1993, NIH 1996, Rao 1995, Rao et al. 1990).

Diagnosis of temporomandibular disorders is based on the physical examination and a complete medical and dental history, including information about hearing, speech, and swallowing problems,
as well as pain and dysfunction. This information can be complemented by data from imaging and other diagnostic tests (Clark et al. 1993, NIH 1996). Evaluation encompasses examination of oral-facial tissues, musculature, and neurological function.

Particular attention is paid to measures of the range of motion, mouth opening, existence of any parafunctional conditions (e.g., clenching, grinding), and the presence of joint or muscle tenderness and cutaneous hyperalgesia. Features of the reliability studies on the examination methods have been reviewed (Clark et al. 1993, Mohl et al. 1990). Psychosocial assessments using validated instruments can determine the extent to which pain and dysfunction diminish the patient's quality of life (Dworkin 1994) and can suggest appropriate treatments (NIH 1996, Travell and Simons 1983, Zarb 1994).

The evidence base for the efficacy of treatment modalities is severely limited and has resulted in a wide range of diagnostics and therapies. Treatments range from conservative and reversible approaches to joint surgical procedures. At present the evidence is insufficient to warrant prophylactic intervention for management of these disorders (NIH 1996).

Currently available epidemiological evidence suggests that temporomandibular disorders can frequently resolve over time and that conservative, reversible approaches are the treatments of choice. Ideally, the practitioner and the patient should work together to develop a treatment plan that is evidence-based and patient-centered, taking into consideration all etiologic factors, the level of pain and dysfunction the patient is experiencing, and their impact on the patient's quality of life (see Chapter 3).

### Mucosal and Autoimmune Diseases

Microbial infections and autoimmune disorders contribute to a range of mucosal and gingival conditions. The physical examination may be sufficient to identify the lesions associated with herpes virus infections (cold sores), papillomaviruses (oral warts), or fungal infection (commonly, infection by *Candida albicans*), with definitive diagnosis confirmed by cytology, biopsy culture, or in situ hybridization. The patient's history and immune status can supply additional information indicating risk factors, including the presence of systemic diseases. Selected mucosal diseases and associated clinical findings are described in Chapters 3 and 5. Tissue biopsy is critical for the diagnosis of many mucosal diseases. In addition, oral sites may be convenient biopsy sites for autoimmune diseases such as Sjögren's syndrome, which have both a systemic and an oral-facial component. Although the evidence base in terms of randomized, double-blind, controlled clinical trials for the treatment of oral manifestations of mucocutaneous disorders is limited, treatment options generally depend on the severity and extent of the disease. Because many of the available drugs may have significant side effects, evaluation of the risk-benefit ratio for the patient is always of great importance. Coordination of care with other health care providers is warranted.

### FACTORS AFFECTING FUTURE HEALTH CARE PRACTICES

The last decades of the twentieth century were witness to major improvements in the prevention, diagnosis, and treatment of oral diseases—a trend that will continue to accelerate the paradigm shift in the management of oral diseases from repair of damaged tissues to the control of infections. In addition, modification of risk factor exposures will result in improvements in health and in the management of disease. A closer look into factors that will affect the future of oral health care requires an overview of the current state of guidelines for oral care and the status of evidence-based practice. The approaches used to determine the evidence for practice and the development of guidelines for care are an emerging field of activity. Education in the health professions is already emphasizing the
Evidence-based Practice

During the 1990s, "evidence-based medicine" emerged as both popular phraseology and practice philosophy. The origins of evidence-based medicine go back to mid-nineteenth-century Paris and earlier, yet the approach is still a relatively young discipline that is now rapidly evolving (Sackett et al. 1996). Evidence-based medicine has been defined as the integration of "individual clinical expertise with the best available external clinical evidence from systematic research" and with patients' choices (Sackett et al. 1996). The skills required include defining a clinical problem, critically appraising the relevant literature, and deciding whether and how to integrate this information into practice (Evidence-Based Medicine Working Group 1997). Evidence-based medicine is neither a "cookbook" nor an ivory tower approach (Sackett et al. 1996).

The philosophy is being adopted across a range of disciplines, leading to the terms "evidence-based dentistry" and "evidence-based nursing," among others. The practice of evidence-based dentistry "incorporates the judicious use of the best evidence available from systematic reviews, when possible, with knowledge of patients' preferences and clinicians' experiences to make recommendations for the provision of the right care, for the right patient, and at the right time" (Ismail et al. 1999).

The reliance on evidence using systematic reviews of the literature has led to initiatives in the United States, Canada, and Europe to enhance the conduct and use of systematic reviews. The Agency for Healthcare Research and Quality (AHRQ) created 12 evidence-based practice centers in 1997 to conduct systematic reviews and develop evidence reports. The Cochrane Collaboration and the Centre for Reviews and Dissemination at the University of York are examples of prominent activities in the United Kingdom to support systematic reviews. The Cochrane Oral Health Review Group, one of 50 specialty review groups within the Cochrane Collaboration, has a number of systematic reviews completed or under way of interest to oral health practitioners (see Table 8.14) (Tavender 1999). In Canada, considerable contributions to the field have been made by McMaster University and the Canadian Coordinating Office for Health Technology Assessment.

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

Clinical Practice Guidelines

The development of clinical practice guidelines is one of the intended outcomes of evidence-based reviews. The classic definition for clinical practice guidelines describes them as "systematically developed statements to assist practitioner and patient decisions about appropriate health care for specific clinical circumstances" (Field and Lohr 1992). A 1995 review of the status of clinical practice guidelines in dentistry (Shugars and Bader 1995) found that a limited number of guidelines were available and that few extant guidelines met all of the desirable attributes for guidelines identified by the Institute of Medicine (Field and Lohr 1992). Most notably, a majority of guidelines were based on consensus among selected professionals, with little evidence of support from the scientific literature. Since 1995, the number of dental practice guidelines has grown slowly but steadily. Table 8.15 lists selected sets of guidelines that, taken together, represent an estimated 50 percent of all current published dental guidelines intended for national distribution. When 36 national dental organizations representing clinical aspects of dentistry and dental practice were surveyed in early 1999, 12 of 22 responding organizations indicated that they had developed guidelines (J.D. Bader, personal communication, 1999), 8 of which are listed in the table. Some of these guidelines have not been widely distributed, are not published in the scientific literature, nor are available on the sponsoring organization's Web site. Not shown are guidelines developed by care delivery organizations for use in their clinical practices. These are generally not available for public or external professional scrutiny.

The table provides information on two important characteristics of clinical practice guidelines: the extent to which they are evidence-based and their level of specificity, which will determine their clinical applicability. Clinical applicability is a key feature of
practice guidelines, and it is heightened as the amount of specific information in a guideline is increased to identify patient and condition characteristics to which the guideline applies. As the "evidence-based" concept gains popularity, there is a growing expectation that clinical practice guidelines will reflect systematic evaluation of the relevant literature and will present an evaluation of the strength of the evidence for each recommendation (Ismail et al. 1999). Such information, which ensures the content validity of the guidelines, not only helps practitioners and patients understand exactly what is and is not known about the effectiveness of proposed treatments, but also identifies research needed to evaluate current practice. A less comprehensive but still useful approach to identifying the scientific support for clinical interventions is the traditional approach of citing specific studies and reviews when discussing specific treatments. Practice guidelines without explicit linkages to the literature, such as those supported only by selected citations not linked to specific statements and those without any citations, can still be useful, but users have less assurance that the content is valid.

As the expectations for the content and use of clinical practice guidelines mature, "perhaps the main task of guideline development [will be] to summarize the strength of the evidence for the effectiveness of a given clinical practice in relation to risks and costs" (Fletcher and Fletcher 1998). Thus, practice guidelines will need to be revised considerably to incorporate the literature on treatment outcomes as they become available. Currently, information on both the effectiveness of specific dental treatments and the range of outcomes examined is extremely limited (Bader and Shugars 1995). For example, none of the guidelines in the table address patient preference or patient utility issues. Dental organizations have the opportunity to address these information gaps as practice guidelines are developed. Professional dental organizations are the most likely developers, but other organizations can also be vigorous participants. Finally, organizations developing guidelines should also develop a plan for their dissemination, evaluation, and revision. The existence of guidelines does not ensure that appropriate treatment decisions will be made. Passive distribution of clinical practice guidelines is generally ineffective in altering practice, whereas more active, multifaceted interventions can achieve some measure of desired change (Bero et al. 1998).

**Science and Technology Contributions**

During the past several decades, there have been major improvements in the prevention, diagnosis, and treatment of oral diseases. Enhanced disease prevention and health promotion will require the participation of all health professions, especially in addressing common risk factors such as tobacco, alcohol, and inappropriate dietary practices. The field of diagnostic tests for oral diseases should continue to expand, enabling clinicians to analyze the risk of disease and disease progression for individual patients. Full assessment of the strengths and weaknesses of new diagnostic tests and evaluation of when they are best used will be key to proper interpretation of the results, permitting tailored referrals and treatments. Treatment options for individual patients are increasing, including the regeneration of lost bone and connective tissue. Restorative materials are continuously improving, resulting in safe, effective, and aesthetic restorations. The growing field of biomimetics should continue to revolutionize oral health and oral health care. Development of bio-compatible restorative and implant

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**TABLE 8.14**

Systematic reviews on oral health topics conducted by the Cochrane Collaboration Oral Health Review Group

<table>
<thead>
<tr>
<th>Interventions for treating oral leukoplakia</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevention of oral mucositis or oral candidiasis for patients with cancer receiving chemotherapy (excluding oral and pharyngeal cancers)</td>
<td>Protocol</td>
</tr>
<tr>
<td>Treatment of oral candidiasis for patients receiving chemotherapy or radiotherapy</td>
<td>Protocol</td>
</tr>
<tr>
<td>Treatment of oral mucositis or its associated pain for patients receiving chemotherapy or radiotherapy</td>
<td>Protocol</td>
</tr>
<tr>
<td>Topical fluoride for preventing dental caries in children and adolescents</td>
<td>Protocol</td>
</tr>
<tr>
<td>Pit-and-fissure sealants for preventing decay in the permanent teeth of children and adolescents</td>
<td>Protocol</td>
</tr>
<tr>
<td>Guided tissue regeneration for periodontal intrabone defects</td>
<td>Protocol</td>
</tr>
<tr>
<td>Orthodontic treatments for posterior cross-bites</td>
<td>Protocol</td>
</tr>
<tr>
<td>Interventions for treating oral lichen planus</td>
<td>Protocol</td>
</tr>
<tr>
<td>Potassium-nitrate-containing dentifrice for dentin hypersensitivity</td>
<td>Protocol</td>
</tr>
</tbody>
</table>

*Status: Protocol indicates the introduction, objectives, materials, and methods for reviews currently being prepared.*

*Source: Tavender 1999.*

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Materials will continue, as well as development of new biologically engineered substitutes for lost bone, connective tissue, and diseased articular disks, to name several possibilities. Harnessing other basic science knowledge will enable the development of new therapies such as genetically engineered growth factors. Improved understanding of the genetic risk factors, limitation of exposures to teratogens, and attention to diet may markedly diminish the occurrence of congenital anomalies such as cleft lip and palate.

Science is continuing to reveal the intricacies and complexities of disease etiology and pathogenesis. In turn, the classification and diagnosis of diseases and conditions will improve and lead to tailored treatment options. The recent efforts to understand and define early childhood caries are an example of this evolutionary process (Ismail et al. 1999). This example further demonstrates that disease definitions are important for population-based research (Drury et al. 1999, Kaste et al. 1999).

<table>
<thead>
<tr>
<th>TABLE 8.15</th>
<th>Characteristics of selected dental clinical practice guidelines</th>
</tr>
</thead>
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<tr>
<td><strong>U.S. professional dental organizations</strong></td>
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<tr>
<td>American Dental Association</td>
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<tr>
<td>dental practice parameters (1996)</td>
<td></td>
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<tr>
<td>American Association of Endodontists</td>
<td></td>
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<tr>
<td>appropriateness of care guidelines (1994)</td>
<td></td>
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<tr>
<td>American Association of Orthodontists</td>
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<tr>
<td>clinical practice guidelines (1996)</td>
<td></td>
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<tr>
<td>American Association of Oral and Maxillofacial Surgeons</td>
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<tr>
<td>parameters of care (1995)</td>
<td></td>
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<tr>
<td>American Academy of Pediatric Dentistry</td>
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<tr>
<td>Guidelines (1998)</td>
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<tr>
<td>American Academy of Periodontology</td>
<td></td>
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<tr>
<td>parameters of care (1996)</td>
<td></td>
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<tr>
<td>American College of Prosthodontists</td>
<td></td>
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<tr>
<td>parameters of care (1996)</td>
<td></td>
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<tr>
<td>American Cleft Palate-Craniofacial Association</td>
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<td>parameters (1993)</td>
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<td><strong>U.S. government agencies</strong></td>
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<tr>
<td>U.S. Preventive Services Task Force</td>
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<tr>
<td>counseling to prevent dental and periodontal disease (1996)</td>
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<tr>
<td>Office of Medical Applications of Research</td>
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<tr>
<td>management of temporomandibular disorders (1996)</td>
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<tr>
<td>Food and Drug Administration</td>
<td></td>
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<tr>
<td>selection of patients for x-ray examinations (1987)</td>
<td></td>
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<tr>
<td><strong>Other organizations</strong></td>
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<tr>
<td>American Heart Association</td>
<td></td>
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<tr>
<td>prevention of bacterial endocarditis (1997)</td>
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<tr>
<td>Canadian Task Force on Preventive Health Care</td>
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<tr>
<td>prevention of periodontal disease (1994)</td>
<td></td>
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<tr>
<td>Canadian Paediatric Society</td>
<td></td>
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<tr>
<td>the use of fluorides in infants and children (1996)</td>
<td></td>
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<tr>
<td>Task Force on Periodontal Regeneration of Intrabony Pockets</td>
<td></td>
</tr>
<tr>
<td>periodontal regeneration of intrabony defects (1995)</td>
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</tr>
</tbody>
</table>

*Refers to publication date of collected set of guidelines.
*Corporate sponsorship.
One area critical to the ability of dentists to adopt new treatment modalities or diagnostic techniques is the development of diagnostic codes. In contrast to medicine, these diagnostic codes currently have no impact on reimbursement. However, the development and introduction of such codes are essential for the conduct of needed outcomes research, and their widespread use is necessary for practice-based research. Such codes permit the documentation of preexisting conditions, monitoring of disease progression, and provision of surveillance data in public health programs. A pilot study in a Canadian public health program has proposed and implemented a set of diagnostic codes (Leake et al. 1999). The American Dental Association has undertaken the development of a comprehensive set of diagnostic codes, expected to be released in the near future.

**Broadening the Base for the Provision of Oral Health Care**

Further biologic, scientific, and technological advances and changes in the organization of health care delivery will continue to alter future professional and individual health care practices (see Chapters 9 and 11). The increased knowledge of risk factors, the importance of monitoring disease progression and treatment effects, and the ability to diagnose conditions and intervene earlier will necessitate increased involvement of all health professionals in oral health care and may reflect changes in care provision and referral patterns. Management of conditions such as oral and pharyngeal cancers, cleft lip/palate, and chronic pain requires multidisciplinary teams. The promotion of oral health and the prevention of oral disease are at a turning point. A systematic approach to integrate the scientific findings into evidence-based assessments will provide clearer guidance to all health care professions and the public. To capitalize on the rapidly emerging science base, the active participation of a full range of health care providers and individuals and the community is needed.

**FINDINGS**

- Achieving and maintaining oral health require individual action, complemented by professional care as well as community-based activities.
- Individuals can take actions, for themselves and for persons under their care, to prevent disease and maintain health. Primary prevention of many oral, dental, and craniofacial diseases and conditions is possible with appropriate diet, nutrition, oral hygiene, and health-promoting behaviors, including the appropriate use of professional services. Individuals should use a fluoride dentrifice daily to help prevent dental caries and should brush and floss daily to prevent gingivitis.
- All primary care providers can contribute to improved oral and craniofacial health. Interdisciplinary care is needed to manage the oral health—general health interface. Dentists, as primary care providers, are uniquely positioned to play an expanded role in the detection, early recognition, and management of a wide range of complex oral and general diseases and conditions.
- Nonsurgical interventions are available to reverse disease progression and to manage oral diseases as infections.
- New knowledge and the development of molecular and genetically based tests will facilitate risk assessment and management and improve the ability of health care providers to customize treatment.
- Health care providers can successfully deliver tobacco cessation and other health promotion programs in their offices, contributing to both overall health and oral health.
- Biocompatible rehabilitative materials and biologically engineered tissues are being developed and will greatly enhance the treatment options available to providers and their patients.

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Achieving optimal oral, dental, and craniofacial health requires a commitment to self-care and preventive behaviors as well as the receipt of appropriate professional care. Community-wide approaches to support oral health and the role of professional services are covered in Chapters 7 and 8, respectively. Although the services provided by dental practitioners are the first that come to mind when people consider the nation's resources to address the diseases and disorders that affect the craniofacial complex, the dental component is augmented by two other components—medicine and public health. These three do not constitute a single system of care, but serve as individual components variously involved in the promotion of health and the provision of services to individuals and families, communities, and the population at large. The linkages and overlaps among the components mirror those between oral and general health described elsewhere in this report (e.g., Chapters 2, 3, 5, and 6), and may also play a role in the disparities noted in Chapter 4.

As has been noted in previous chapters, data regarding the contributions to oral health care made by the medical and public health components are not nearly as available as those that describe the contributions made by dental practitioners. Most of this care is provided by dentists in private practice. Expenditures for their services represented over 96 percent of the estimated $53.8 billion spent on dental care in 1998, or 4.7 percent of the $1.1 trillion spent on all health care in the United States that year (HCFA 2000b). Although they surely undercount the contributions of the medical and public health components, these expenditures indicate the burden that oral diseases and conditions place on the American people, as well as their willingness to invest in the prevention, treatment, and rehabilitation of oral conditions—a reflection of the value they place on oral health.

There have been notable achievements in oral health over the years, among them the dramatic and continuing reduction in the prevalence of dental caries in sizable population groups (see Chapter 4). This has led to an impressive decline in tooth loss, with the result that the majority of Americans can now expect to retain their natural teeth over their lifetimes. At the same time, all three components have participated in the revolutions in biomedical and behavioral sciences and technology that have deepened our understanding of the biological, environmental, behavioral, and genetic origins of many oral, dental, and craniofacial diseases. Americans today can benefit from oral health services that are among the best in the world. Moreover, as new and improved preventive, diagnostic, and treatment measures emerge (see Chapter 8), they create further opportunities for improving the nation's oral health.

COMPONENTS OF PROFESSIONAL CARE

The dental, medical, and public health contributions to oral health differ dramatically in their size, focus, financing, and resources. Following is a brief description of each component and their areas of overlap.

The Dental Component

Comprehensive oral health care in America is largely supplied by a private dental care system composed of dentists, dental hygienists, dental assistants, laboratory technicians, and other professional staff in independent dental offices. The estimated numbers of active dental personnel are presented in Table 9.1. Of the 156,500 professionally active dentists in the United States in 1997, 91.7 percent were in private practice (ADA 1998a). Women constitute 14.4 percent of the total, and minorities 11.1 percent (ADA
Provision of Oral Health Care

1998a). Other professionals, who are educators, biomedical and behavioral science researchers, techni-
cians, manufacturers of dental products, and admin-
istrators, complement this workforce.

In contrast to medicine, where only 40 percent of physicians were in primary care practices in 1990, approximately 80 percent of dentists are general practitioners (ADA 1998a). The remainder qualify as specialists in one or more of the nine disciplines formally recognized by the American Dental Association: orthodontics, oral and maxillofacial sur-
gery, oral and maxillofacial radiology, periodontics, pediatric dentistry, endodontics, prosthodontics, dental public health, and oral and maxillofacial pathology. More than half of these specialists are orthodontists or oral surgeons (ADA 1998a). A small number of dental practitioners focus on special interest areas such as anesthesiology or oral medicine. There has been little change in the approximately 4 to 1 ratio of general dentists to specialists in the past 10 years.

New technologies and changing patterns of disease are broadening the scope of dental practice. The average general practitioner and staff now engage in more preventive services than in years past (ADA 1990). A reduction in the number of amalgam and resin restorations per patient per year from 1980 to 1995 has also been noted (Eklund et al. 1997). Although dentists perform fewer extractions and restorations, preserving the teeth of an aging population has increased the need for crowns and periodontal treatment.

Since oral health is an integral part of total health, most dentists provide primary care services to their patients. In addition to educating patients on oral health care, dentists and their staff may counsel patients on tobacco and other substance use and cessation, nutrition, and dietary practices. In addition, information that dentists obtain from a patient's history and from screening and diagnostic tests may suggest the presence of systemic disease, warranting a referral of the patient to other health care professionals.

Dental services are provided in a practice model that is different from that used by the medical profession. Most private dental practices consist of one dentist (68.7 percent) or two dentists (19.6 percent). The remaining practices (11.7 percent) are group practices of three or more dentists who share expenses and revenues. This distribution of dentists by practice size, along with the number of hours worked per week, has remained remarkably constant over the years (ADA 1998a). The size, number, and location of dental practices are important determi-
nants of availability of care and accessibility to services, as well as of the unit cost of care. Figure 9.1 shows the association between the availability of dentists and state mean per capita income (Burt and Eklund 1999). Dental care is also provided in dental schools and public health clinics, hospitals, nursing homes, and other institutional settings. These are sometimes the principal source of oral health care for communities and special population groups with limited access to health care.

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<th>TABLE 9.1</th>
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<tr>
<td>Estimated numbers of active oral health personnel, United States, selected years</td>
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<tr>
<td>Dentists</td>
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<tr>
<td>Dental hygienists</td>
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<tr>
<td>Dental assistants</td>
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<td>Dental laboratory technicians</td>
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Note: NA = not available.

Source: HRSA 1999.

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<th>FIGURE 9.1</th>
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<tr>
<td>The association between state mean per capita income and the population-to-dentist ratio, by individual state, United States, 1995</td>
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The Medical Component

In the context of oral, dental, and craniofacial health care, the medical component includes dentists, physicians, nurses, and allied health professionals whose services are provided through hospitals, nursing homes, ambulatory care facilities, and health professional offices. Data on the nature and extent of such oral health services, as well as on the number of non-dental professionals who supply them, are limited. For some conditions—particularly developmental anomalies, injuries, infectious diseases, pain syndromes, and oral and pharyngeal cancers—the medical component provides comprehensive care, often working in tandem with dental specialists and general practitioners. For example, physicians and oral and maxillofacial surgeons may plan treatments together and operate on individuals born with cleft lip/palate to repair the clefts as the children age. Orthodontists, pediatric dentists, prosthodontists, and other dental specialists, speech and hearing therapists, plastic surgeons, neurologists, radiologists, nutritionists, psychologists, other health professionals, and social workers are also part of the craniofacial team.

Collaboration and coordination between physicians and dentists are needed to provide integrated medical and oral health care for cardiac patients and those undergoing chemo- and radiation therapy or implant and organ transplant procedures. Nondental health care personnel in long-term and geriatric care facilities may be the principal sources of oral health care given to residents.

Although most hospitals have dental personnel on staff to handle emergency situations, emergency room physicians and other hospital personnel are often called on to initiate treatment of acute oral-facial injury or pain of dental origin, with referrals to dentists for follow up. Also, patients with chronic oral-facial pain conditions are sometimes treated by family practice, internal medicine, or neurology physicians, sometimes with referral to dental or other medical specialists.

The Public Health Component

Federal agencies under the jurisdiction of the U.S. Department of Health and Human Services (USDHHS) and the U.S. Departments of Defense, Veterans Affairs, Agriculture, Education, Transportation, and Justice, among others, serve public health needs in diverse ways. These agencies may include units or programs specifically dedicated to oral health, as well as components that collect, organize, or make available information or services related to oral health as part of general health programs. For example, the National Institutes of Health (NIH) is the primary federal agency supporting biomedical and behavioral research and research training. Assurance of the safety of foods, cosmetics, drugs, and devices is provided through the regulatory authorities of the Food and Drug Administration (FDA). The Centers for Disease Control and Prevention (CDC) focuses on state-based programs for monitoring and preventing disease and, through the National Center for Health Statistics, orchestrates the collection of nationally representative health information and population data. The Agency for Healthcare Research and Quality (AHRQ) uses evidence-based practice centers to evaluate literature relevant to the management of diseases and conditions, conducts national expenditure and care utilization surveys, and supports research directed at understanding health care systems. The Medicaid, Medicare, and newly enacted State Children's Health Insurance Program (SCHIP) programs are directed by the Health Care Financing Administration (HCFA), which funds a variety of care services prescribed by law or regulation.

Several federal agencies provide direct services to specific, often disadvantaged populations or to military personnel and their dependents. The U.S. Departments of Defense, Transportation, and Veterans Affairs, the U.S. Department of Justice's Bureau of Prisons, and the USDHHS's Indian Health Service (IHS) and Health Resources and Services Administration (HRSA) provide oral health care directly to selected populations. Oral health education also is provided through the U.S. Department of Agriculture's Women, Infants and Children (WIC) program. In addition, HRSA provides funds for health professional education and administers the Ryan White Comprehensive AIDS Resources Emergency Act. States, counties, and cities also support dental programs for disadvantaged populations under federally mandated and funded Maternal and Child Health Programs or as part of Medicaid or the State Children's Health Insurance Program. They may also provide direct support through tax.