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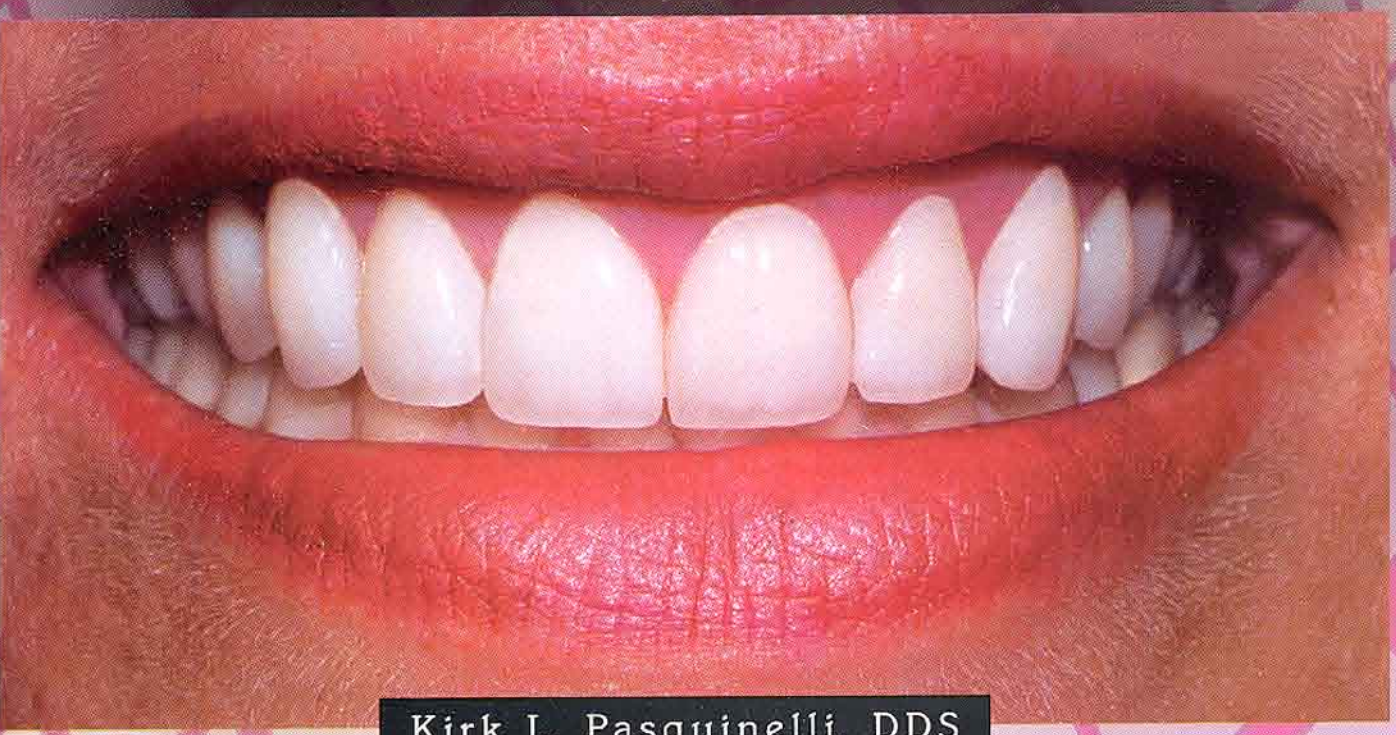
Perio PLASTIC SURGERY



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DEPARTMENTS

FEATURES

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CDA's Corporate Alphabet Soup

Jack F. Conley, DDS

For a period after unification of the northern and southern California dental associations in 1974, the new California Dental Association was relatively simple in structure.

In 1980, however, a time of phenomenal growth began with the start-up of The Dentists Insurance Company (TDIC) as California dentistry first recognized the value of for-profit subsidiaries as a benefit to the membership. With TDIC, CDA was ensuring the availability of an insurance product at reasonable cost and with dentist oversight. Prior to that time, professional liability coverage for dentists had been available only from commercial companies and was under their complete control. Besides dentist control, the other bonus to members from TDIC is the revenue that is ultimately returned to CDA and the dividends to members who are policyholders.

The alphabet soup continued to add ingredients as leadership saw merit in the potential revenue from programs that would enhance operations of the association and reduce the percentage of out-of-pocket membership dues required to fund the growing list of CDA membership services. There was a restructure earlier this decade that added a holding company (CDAHCI) for oversight. Over time, this growth contributed to what has become a baffling slate of ingredients in the CDA alphabet soup to the average CDA member. CDAHCI (California Dental Association Holding Company, Inc.), TDC (The Dentists Company), TDCIS (The Dentists Company Insurance Services), TDCMS (The Dentists Company Management Services), and TDIC currently make up a confusing landscape for CDA members who have had occasion to make contact with some of the above entities regarding programs and services.

Some of that confusion should soon be resolved once the necessary business and legal actions are completed on a couple of important changes that were approved by the CDA Board of Trustees at its summer meeting. Final approval would come from the 1999 House of Delegates in the fall. If simplification and clarification is a purpose, the House should not balk on these proposals.

One action will merge TDC with TDCIS. That will drop one ingredient from the mix. A major part of the confusion comes from these two entities and TDIC. The second action, once the two have been merged, will change the name of TDCIS to 1201 Financial & Insurance Services. With the former TDC entities under one umbrella and with a new name that meets the requirements of the California Department of Insurance, there should be no further confusion with TDIC. Whether or not the new entity becomes an alphanumeric soup (1201 FIS), we will have to wait to see.

This brings us to a most important part of the structure, CDAHCI, otherwise known as the Holding Company. The Holding Company Board, chaired by CDA Executive Director Timothy Comstock,

played a role in this simplification process by requesting that the subsidiary boards consider name changes that would eliminate the confusion that existed among members, volunteer leaders, vendors, and even staff.

The Holding Company has also been reviewing its statement of purpose and will take an active role in its responsibility to oversee the many business activities of CDA. Coupled with the organizational review currently being conducted for CDA, members can be assured that every effort is being made to provide oversight over business activities and eventually to incorporate efficiencies in operations that will strengthen the organization and provide them, the members, with the highest level of benefits. Further, it is our belief that efforts to simplify the structure so that members will better understand what their organization is doing on their behalf will also strengthen their support of their association and its progress.

Subsidiaries of the California Dental Association

CDAHCI -- California Dental Association Holding Company, Inc.

TDC* -- The Dentists Company

TDCIS* -- The Dentists Company Insurance Services

TDCMS -- The Dentists Company Management Services

TDIC -- The Dentists Insurance Company

* CDA's Board of Trustees has approved a merger of TDC and TDCIS into one entity to be named 1201 Financial & Insurance Services.

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Drinking in the Success

By David G. Jones

Fluoridated drinking water, maligned by its opponents as anything from poison to a nefarious political plot, has been chosen by the Centers for Disease Control and Prevention as one of 10 great public health measures of the century.

The CDC, in observance of National Public Health Week, determined that fluoridation's scientifically proven oral health benefits place it among other significant public health measures.

According to CDC, since the beginning of the 20th century, the average American life span has lengthened by more than 30 years, 25 of which are attributable to advances in public health.

"Fluoridation of water supplies has meant less tooth loss, less pain and suffering, and less time lost from school or work, and all that adds up to a major improvement in our quality of life due to better oral health," says Scott Presson, DDS, chief of the CDC's Program Services Branch, Division of Oral Health, in Atlanta. "Now members of each generation are taking more teeth with them as they move into later years, so seniors are increasingly able to smile at their grandchildren with their real teeth rather than false ones."

Presson said that the significance of fluoride's inclusion in the list is that it recognizes the important contribution of oral health to general well-being and a satisfactory quality of life.

"From the beginning, when science discovered fluoride's role in the prevention of tooth decay, to its application in public health programs to extend fluoridation to a large portion of the U.S. population, it has been a public health success story," he says.

Presson also emphasized that a message sometimes obscured by antifluoridation rhetoric is the benefit to all ages -- not children only.

"When older adults retain more teeth, their gums can recede making the roots at risk for root caries, so fluoridation can help prevent tooth decay even in adults and elders," he says. "So the decision to fluoridate was the right one in 1945, and it's still the right decision in 1999."

Michael W. Easley, DDS, an assistant associate professor at the State University of New York and a nationally recognized fluoride expert, says the CDC's statement is consistent with the many endorsements received from public health and scientific organizations over the past 50 years.

"It also reminds me of what former Surgeon General Luther Terry said in the 1960s," Easley says. "He was the one who issued the first surgeon general's report linking smoking and cancer, and he talked about the 'four horsemen' of public health -- chlorination, pasteurization, vaccination, and fluoridation."

In dentistry, fluoridation also reflects the hard work of members of the profession, according to one of the nation's top dental experts.

"It celebrates the dedication of the dental profession to promote oral health and reduce disease," says Harold Slavkin, DDS, director of the National Institute of Dental and Craniofacial Research in Bethesda, Md. "In 1995, before California's fluoridation legislation, approximately 110 million Americans benefited from daily consumption of fluoridated drinking water. Since one out of every eight Americans lives in California, current efforts to fluoridate more California water districts would have a significant effect on our nation's public health progress."

Easley emphasizes that before fluoridation's introduction, people sometimes died of complications of oral disease.

"We couldn't prevent caries with fluoridation before 1945, so a lot of people got caries that proceeded to major systemic disease that in quite a few cases killed people," he said. "It was a life or death issue for many in the old days."

Timothy R. Collins, DDS, chair of the California Fluoridation Task Force, believes the CDC's announcement further underscores what organized dentistry and fluoridation supporters have long said.

"This is one of the most important public health measures that has come along," he says. "Now we have a very clear statement as to how it relates to other public health measures, and it's another way to demonstrate fluoridation's benefits to the public. It's safe, effective, economical, and is the golden bullet that prevents tooth decay."

A state lawmaker who in 1995 put her political will behind a bill calling for fluoridation of many of the state's municipal water systems lauds the CDC's high-profile endorsement.

"After helping to ensure that fluoride will become a reality for more Californians, it's wonderful to see that one of the nation's top health agencies has recognized its benefits for public health," says Sen. Jackie Speier, D-Hillsborough, author of AB 733. "This reinforces the fact that fluoridation is safe and effective in preventing dental disease."

Slavkin says promotion of fluoridation must continue while scientists work to develop safe vaccines for at-risk children to reduce or eliminate dental caries.

"Such a strategy worked for smallpox and polio, and we feel it could also work for dental caries," he says.

The CDC's Morbidity and Mortality Weekly Report is profiling the 10 public health achievements in a series of reports published through December. Fluoridation will be examined in an October issue.

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The CDC's list of 10 great public health achievements of the 20th century:

1 Vaccination
2 Motor vehicle safety
3 Safer workplaces
4 Control of infectious diseases
5 Decline in deaths from heart disease and stroke
6 Safer and healthier foods
7 Healthier mothers and babies
8 Family planning
9 Drinking-water fluoridation
10 Recognition of tobacco use as a health hazard

Fitting Into the Publicity Picture

By Dell Richards

Most dentists give back to their communities through charitable involvement. Volunteerism usually goes hand-in-hand with the profession.

If a dentist takes the time to belong to a nonprofit organization, he or she may want to maximize its potential. Here are a few ways to get the most from one's involvement:

- A professional shouldn't be shy about passing out business cards during introductions at meetings and events.
- Whenever introducing oneself -- or being introduced -- a dentist can casually mention his or her profession and office location: "I'm Dr. So-and-So. My dental office is just down the street at the plaza."
- The dentist should let patients know about his or her involvement with the organization. The nonprofit affiliation should be included on all marketing materials, brochures, fliers and other printed material. Affiliations should be listed vertically in small type, not in a paragraph of brochure copy.
- Information about the nonprofit's drive or fund-raiser should be included in the dental office newsletter.
- The dentist should become acquainted with the nonprofit's public relations chief and find out how to become involved in providing publicity or support for events. Sometimes a call from a professional can make a potential story more impressive to a reporter. The pitch should be kept short and to the point. Two sentences will do. The reporter may need a source again in the future and will remember someone who was quotable.
- If the dentist feels he or she has received more than he or she has given -- or learned something profound -- from the experience of volunteering, the dentist should call the editor of the local daily or weekly newspaper and ask to write a column about the lesson. Small papers are usually interested in stories that help recruit volunteers for nonprofits.

Dell Richards is owner of Dell Richards Publicity in Sacramento.

Filling Comparison is Study's Target

A five-year study funded by the National Institute of Dental and Craniofacial Research will compare dental amalgam and alternative filling materials in an effort to measure the mercury exposure in children with amalgam fillings and evaluate any associated health effects, writes Mary Tavares, DMD, MPH, in the winter 1999 issue of Forsyth Dental Center.

Subjects were recruited from six clinical sites within two geographic areas in New England: Boston/Cambridge and Maine. In one of the treatment areas, subjects were randomized to receive amalgam restorations. In the other, subjects received composite/glass ionomer.

According to Tavares, some dental scientists have long wondered whether the very low levels of mercury from dental amalgam could cause problems. High levels of mercury exposure, Tavares writes, have been alleged to be associated with tremors, loss of memory, insomnia, fatigue, headaches, irritability, slowed nerve conduction, appetite loss and kidney disorders.

There is no evidence that any of these health effects occur because of the low levels of mercury exposure from dental fillings, Tavares notes. However, because of the concern that small amounts of mercury vapor are being released from amalgam fillings with such activities as chewing or brushing, and that this mercury might accumulate in the tissues of the body, the NIDCR funded the study to thoroughly investigate any possible effects of mercury exposure from dental amalgam in children.

Check Cycle for Clues

By Marios P. Gregoriou

Business activity tends to go in cycles, and understanding the stages of a business cycle can provide clues that may help investors identify favorable opportunities.

While some stocks tend to be immune to economic swings, others perform better or worse during different stages of the business cycle.

The business cycle can provide insight, but it is important to realize that the U.S. economy rarely follows this cycle precisely, and that time spent in each cycle varies. In addition, the economy does not always expand to its fullest levels, nor does it always dip into recession. It does, however, tend to proceed through six typical stages. The following are some general guidelines to which market sectors are inclined to benefit in each cycle.

Stage 1: Economic Slowdown

In a period of economic slowdown, utilities and financial company stocks

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usually react favorably as it becomes clear to investors that the economy is sluggish. Long-term interest rates peak, and shorter rates begin to fall as the Federal Reserve implements strategies to stimulate the economy.

Thus, investors often buy interest-rate sensitive stocks. Utilities, which generally have high debt levels, benefit as rates decline. Financial companies also benefit as rates (and therefore their cost of funds) decrease and loan demands increase.

Stage 2: Anticipated Recovery

With anticipated economic recovery, consumer stocks typically rise, as low interest rates encourage consumers to spend more. Stock prices are generally very low during this stage of the economic cycle.

Stage 3: Mid-Cycle Recovery

In a mid-cycle recovery, interest rates begin to go up, and early signs of inflation emerge. At this stage, stocks generally perform better than bonds. Industrial companies, such as those producing electrical equipment, machinery and construction come into favor.

Stage 4: Full Expansion

During the full expansion stage of the business cycle, interest-rate sensitive stocks generally peak by the time the cyclical expansion is fully under way. Opportunities may appear among companies that benefit during higher inflation and in higher interest-rate environments, such as chemical technology and energy stocks.

Stage 5: Economic Peak

At the economic peak of the cycle, the major stock market indexes may dip below their 12-month moving averages. Basic materials companies (including chemicals and metals) and energy stocks are often favored by investors since inflation is probably peaking during this cycle.

Stage 6: Economic Decline

When the business cycle reaches economic decline, investors attempt to protect their portfolios as the economy slows by moving back into "early-cycle" stocks, beginning with consumer noncyclicals, which are companies that sell products and services whose demand is not tied to the economic cycle.

The Cycle Is Not Precise

While tracking the business cycle can be a useful barometer for investors, keep in mind that the business cycle is not precise. In addition, pinpointing particular stages of economic activity is not always easy, and sometimes even economists cannot agree on exactly where the economy stands in any particular cycle.

Marios Gregoriou is associate vice president financial adviser with Morgan Stanley Dean Witter in Sacramento. This article is published for general information purposes and is not an offer or solicitation to sell or buy any securities or commodities. Any particular investment should be analyzed based on its terms and risks as they relate to specific circumstances and objectives.

Word of Mouth

Lack of coverage about oral cancer in the popular press provides a partial explanation of the public's lack of knowledge and misinformation about oral cancer, write Maria T. Canto, DDS, MPH; Yogo Kawaguchi, DDS, PhD; and Alice M. Horowitz, PhD, in the Journal of Public Health Dentistry.

The public's lack of awareness of oral cancer was documented in a 1990 National Center for Health Statistics survey that found that fewer people perceived smoking as a risk factor for oral cancer compared with other medical conditions, and just 13 percent knew that regular alcohol use increases the risk for oral cancer.

;More than half of the articles (56 percent) identified spit tobacco as the major risk factor. Far fewer mentioned either cigarettes (32 percent) or cigars (12 percent). Less than 50 percent mentioned warning signs for oral cancers, and less than half (42 percent) discussed preventive measures. Only one-third (32 percent) recommended cessation of tobacco use.

The authors note that previous press studies have shown that most magazine articles on cancer discuss breast cancer and skin cancer. The authors surmise the reasons for that are the strong involvement of advocacy groups and the fact that the treatment for these cancers could be cosmetically disfiguring to the patient.

Some Pain is in the Perception

Pain caused by treatment provided by a dentist who is perceived as caring is likely to have less psychological impact than pain from treatment by a dentist who is perceived to be cold and controlling, according to an article in the March 1999 Journal of Dental Research.

The article "Age of Onset of Dental Anxiety" discusses a survey of 6,630 residents of Etobicoke, one of five municipalities within metropolitan Toronto. The results of the survey challenge the view that dental anxiety is invariably a fear with childhood origins. It also addresses the causes of fear and who is most likely to be affected.

According to the study, about 16 percent of the subjects were dentally anxious and characterized by several factors: physiological responses during dental treatment, including increased breathing rate, increased heart rate, and nausea; avoidance of dental care, including canceling appointments or failing to keep appointments; and fears about the dentist-patient relationship regarding communication, belittlement, lack of control, and trust.

The study separated fear into two categories: exogenous (fear caused by conditioning) and endogenous (fear caused by overall vulnerability to anxiety disorders).

The researchers report only half of dentally anxious people became so in childhood. One-fifth reported onset of dental fear in adolescence, and almost one-third reported onset in adulthood.

Many of the people surveyed reported that their fear began when they had their first traumatic dental experience. However, 42.4 percent of the adult-onset subjects admitted being fearful about all aspects of life, compared with 27.1 percent of the adolescent-onset subjects and 21.6 percent

of the child-onset subjects. The fact that many people became fearful much earlier or much later than their first traumatic dental experience shows that the relationship between traumatic experience and dental anxiety is not simple. However, traumatic experiences were more likely to give rise to dental anxiety if they occurred early in an individual's dental care history than if they were preceded by a series of relatively painless dental visits.

The exogenous conditions that cause fear in children differ from those that cause fear in adults. According to the article, children who became fearful did so after a dental experience that caused pain, fear, or embarrassment. Most fearful children (55 percent) also had family members who were fearful. In contrast, adults were not affected by painful or embarrassing experiences -- frightening experiences alone induced exogenous dental anxiety in adults. Child-onset subjects were more fearful of invasive dental procedures, and adolescent- and adult-onset subjects were more negative concerning dentists' behaviors.

Implants Take Root

The average number of dental implants surgically placed by dentists who perform the procedure nearly tripled from 1986 to 1996, a new ADA survey shows. In 1986, the average number of procedures per implant-placing dentist was 17.7. By 1996, this average had climbed to 51.5.

In 1997, about 12 percent of all responding dentists reported that they had ever surgically placed a dental implant. When broken out by dental specialty, those most likely to have performed the procedure were oral and maxillofacial surgeons (97.5 percent have placed an implant), periodontists (64.1 percent) and prosthodontists (31.8 percent.)

The number of implants placed by oral and maxillofacial surgeons who perform the procedure rose from an average of 34.3 implants in 1992 to 62.4 in 1996. General practitioners who perform the procedure placed an average of 17.2 surgical dental implants in 1992 and an average of 21.0 in 1996.

When asked to indicate their current level of formal implant training, even if they had never placed a dental implant, in 1997 about six in 10 dentists (59.5 percent) reported having had some type of formal training in the procedure.

Some Assembly Required

A majority of the "blueprint of human beings," the human genome sequence, will be completed in the coming year, which will enable researchers to determine hereditary factors, causes, and treatments for major diseases -- such as heart disease, diabetes and common cancers -- according to Francis Collins, MD, PhD, director of the National Human Genome Research Institute at the National Institutes of Health.

The U.S. Human Genome Project officially began in 1990 as a 15-year program to find the estimated 80,000 human genes and determine the sequence of the 3 billion DNA building blocks that underlie all of human biology and its diversity. The complete set of instructions for making an organism is called its genome. It contains the master blueprint for all cellular structures and activities for the lifetime of the cell or organism. For each organism, the components of the slender DNA threads encode all the information necessary for building and maintaining life, from simple bacteria to remarkably complex human beings.

Dr. Collins expects a working draft of 90 percent of the human genome sequence, what he calls the "book of life -- the blueprint of human beings," to be completed in about a year, not 2005 as originally expected.

Proposal for Research Online Draws Fire

A proposal from the director of the National Institutes of Health to post scientific research on the Internet has drawn criticism that doing so would devalue scientific journals.

Dr. Harold Varmus, director of the NIH, counters that posting research online would let scientists exchange information much faster by eliminating the delay created by the publishing process.

But others counter that the proposed web site would be detrimental to journals.

"It would make the journal -- the paper journal, and also the journal web site -- merely archival, redundant," says Dr. Marcia Angell, executive editor of the New England Journal of Medicine. "Insofar as that happened, it would weaken the journals and maybe even destroy them."

The proposed E-biomed site would have two archives. One would accept nearly everything. Submissions would be rejected only if two reviewers found them "extraneous or outrageous."

The other archive would include only papers that have been accepted for publication by journals, but would post them immediately upon acceptance, rather than waiting until publication.

Those against the proposed archives believe they will lower the quality of scientific research and lead to a bloated taxpayer-supported site that few will visit.

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Periodontal Plastic Surgery

Kirk L. Pasquinelli, DDS

ABSTRACT As the demand for esthetic dentistry has increased, dentistry has developed techniques to meet this demand. Periodontal plastic surgery has been part of this effort. This article outlines the scope of periodontal plastic surgery procedures to aid the dental team in diagnosis and treatment of esthetic dental cases.

AUTHOR

Kirk L. Pasquinelli, DDS, is an assistant clinical professor in the Department of Stomatology and Division of Periodontology at the University of California at San Francisco. He also maintains a private practice limited to periodontics and implants in San Francisco.

Until relatively recently, periodontal therapy was limited to the prevention, diagnosis, and treatment of diseases of the supporting and surrounding tissues of the teeth. Periodontal surgical procedures were typically resective in nature. The goals of these procedures were to debride the roots and increase the cleansability of the teeth by reducing pocket depths and modifying furcation defects, often via root removal. The value of this form of therapy on the overall retention of teeth is high, and it remains valid as a treatment modality.1 The unfortunate consequences of this mode of therapy include increased root exposure and decreased papillary height due to apical repositioning of the osseous crest and free gingival margin. As society has become increasingly focused on individual beauty and the retention of youth as measures of self-worth, these side effects of periodontal surgery are no longer acceptable to the majority of patients or practitioners due to the negative effects in the esthetic zone.

The past 20 years have seen an increasing focus on esthetic procedures in all areas of clinical dentistry, and periodontics is no exception.2 The field of periodontics is continually expanding as regenerative procedures are developed in an attempt to replace missing hard and soft tissues and to prevent esthetic compromise.

Periodontal plastic surgery has as its primary goal the restoration or enhancement of the esthetic component of the supporting and surrounding tissues of the teeth or their substitutes. This can be accomplished by reshaping the existing tissues to a more pleasing form as well as by grafting or implanting natural or synthetic devices and materials to replace missing tissues or teeth.

The majority of periodontal plastic surgery procedures are undertaken to treat or prevent the following conditions and can be classified as such:

- Marginal recession (root coverage);
- Ridge deficiency (ridge augmentation);
- Ridge collapse after extraction (ridge preservation);

- Excessive or asymmetrical gingival display and biologic width invasion (crown lengthening); and
- Esthetic defects around dental implants (hard and/or soft tissue grafting).

The field of periodontal plastic surgery has become broad in scope. It includes procedures in which autogenous and non-autogenous materials are used for surgical augmentation of deficient areas, as well as the surgical reshaping of autogenous tissues to improve their appearance. For the sake of brevity, this article will present only techniques that manipulate the patient's autogenous tissues and exclude procedures utilizing membranes or sources of tissue other than the patient. The article will further focus on soft tissue grafting versus hard tissue grafting. The intent of this article is to present an overview of these periodontal plastic surgery techniques in order to expand multidisciplinary treatment planning options for the dental team.

Diagnostic and Treatment Planning Considerations

The oral esthetic zone consists of three components:The lips, which delineate that portion of the mouth that is on display;

- The gingiva, which frames and defines the shape of the individual teeth; and
- The teeth, which are the ultimate focus for an observer's assessment of color, contour, position, and shape.

Treatment planning for an esthetically pleasing smile involves bringing the three components of the esthetic zone into harmony. All dentists learn this "ideal" relationship when they are taught to set denture teeth and wax denture bases.

In Western culture, this so-called ideal setup has the following characteristics3 (**Figure 1**). On smiling, the upper lip line follows the level of the gingival margins of the maxillary teeth and exposes the entire length of the teeth and up to 3 mm of gingiva. The lower lip line follows the incisal edges of the maxillary teeth. The gingival heights of the maxillary central incisors mimic one another. The lateral incisor gingival margins are slightly coronal to that of the central incisors and are bilaterally symmetrical. The cuspid margins are at the same level as the central incisors and equal to each other. The tissue margins extending to the distal are more coronally positioned than the cuspid margins, are symmetrical from side to side, and rise superiorly as they proceed distally. The interdental embrasures are filled with tissue to the contact points. The incisal edges of the maxillary central incisors are even with the cusp tips of the canines, and the incisal edges of the lateral incisors are slightly apical to this line. The buccal cusp tips of the maxillary posterior teeth rise slightly as they proceed to the distal as a result of the Curve of Spee. As the teeth extend posteriorly and laterally, they fill the vestibules to the corner of the smile.

When a patient is concerned about deviations relative to this ideal position of the teeth or gingiva, dental therapy can be used to correct these variations and more closely approach the ideal. A comprehensive treatment plan to address the patient's concerns may require interplay between several areas of clinical dentistry. It is incumbent upon the clinician to recognize the possibilities and limitations of restorative dentistry, periodontics, orthodontics, orthognathics, and implantology in the multidisciplinary treatment of these cases.

Adjunctive Periodontics for Esthetic Dentistry

The color and shape of the periodontal tissues greatly influence the esthetics of the smile. The health of the tissue, as well as the type of tissue (mucosa, keratinized gingiva, or palatal masticatory mucosa) and the presence of dark objects in the alveolus or soft tissues (implants, alloy, metal crown margins, or dark roots), influences color. Gingival shape is also contingent upon the health of the tissue, as well as the position of the free gingival margin; the volume and height of the papilla; and, in the absence of teeth, the volume and height of the ridge.

Root Coverage

Root exposure resulting from apical recession of the marginal tissues can create esthetic concerns for a patient (**Figure 2**). As the length of the teeth increase, there is loss of gingival symmetry as well as increased sensitivity, susceptibility to caries, and concern over the retention of the teeth. Restorative coverage of the root can reduce sensitivity or treat caries but cannot decrease the length of the clinical crown, restore the lost periodontal support, or prevent future recession.

The clinical goals of root coverage procedures are to replace the tissues lost due to recession, effect an attachment of the restored tissues to the root of the tooth, reduce thermal and touch sensitivity, discourage future recession, and improve the esthetics of the area when the grafted tissues blend with the adjacent tissue color, texture, and contour (**Figure 3**).

Three forms of root coverage have been presented in the literature. The free autogenous graft (thick gingival grafts[4,5] and connective tissue grafts[6,7]), pedicle flaps (lateral[8] and coronal[9]), and guided tissue regeneration with both nonresorbable[10] and resorbable[11] membranes.

Pedicle flaps and guided tissue regeneration have been shown to be viable root coverage procedures, but

Figure 1. Smile illustrating harmony between the components of the oral esthetic zone (Restoration by Dr. Michael Hack).

Figure 2. Patient with a high smile that exposes a discrepant architecture of the gingival margins due to recession.

Figure 3. Same patient as in Figure 2 after connective tissue grafts have been done for root coverage. Symmetry has been restored to the gingival margins. Replacement of the restoration on tooth No. 5 is planned for an improved color match.

Figure 4a. Palatal donor site for a connective tissue graft. The graft has been removed and a strip of connective tissue approximately 1.5 mm wide has been left coronal to the donor site to aid in primary closure.

Figure 4b. The connective tissue graft free of epithelium.

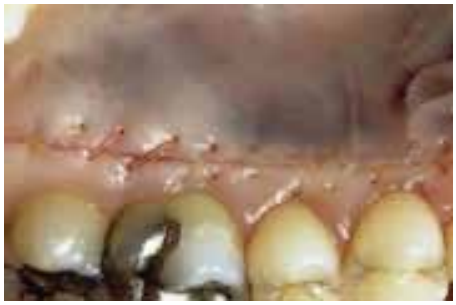

Figure 5a. Primary closure of the palatal donor site with 5-0 gut sutures.

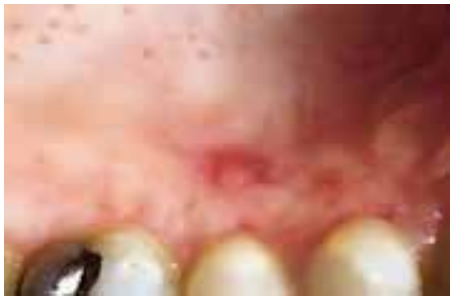

Figure 5b. One-week healing of the palate illustrating typical slight connective tissue exposure with minimal discomfort for the patient.

both techniques have limitations that reduce their clinical applicability. Free autogenous grafts exhibit a very high level of clinical utility. Compared to pedicle flaps and guided tissue regeneration procedures, free autogenous grafts are much less dependent upon the characteristics of the adjacent tissues for success, these grafts can create a localized thickening of the alveolar housing to aid in the prevention of future recession, and several adjacent teeth with recession can be treated simultaneously.

Nabers[12] introduced the free gingival graft in 1966, and Sullivan and Atkins[13] made further refinements in 1968. As originally described, the graft was palatal masticatory mucosa (epithelium and connective tissue) approximately 1 mm thick. This type of graft was found to be unpredictable for covering roots due to sloughing of the grafted tissue over the avascular root surface. The thin grafted tissue bridging the root could not maintain tissue viability for the period of time necessary to establish a new collateral blood supply. In the early 1980s, Miller[4] and also Holbrook and Ochsenbein5 described a technique to graft thicker tissue, approximately 2 mm, from the surface of the palate over the exposed root. This thicker tissue could survive the early lack of nutrition to the area over the root. This allowed the re-establishment of a vascular complex in the graft and retention of the tissue bridging the avascular root surface. Good biologic results were reported with this technique.[14] However, these grafts tend not to blend with the adjacent tissues and are readily identified as thicker and lighter in color. This can cause an esthetic compromise. In 1985, Raetzke[6] and then Langer and Langer,[7] described the use of connective tissue grafts for root coverage. In this technique, the epithelial component is eliminated from the graft, and palatal connective tissue is transplanted into an envelope-like pouch prepared at the recipient site. This pouch provides a dual blood supply to the graft from the superior and inferior connective tissue surfaces in contact with the graft. The retained superior flap also maintains the esthetics of the original tissues and acts as a source for the epithelial cells that migrate over the exposed portion of the connective tissue

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graft. These grafts are very successful in covering the root and blending with the adjacent tissues for a highly esthetic result.

The connective tissue donor site uses a trapdoor approach on the palate to harvest a connective tissue graft free of epithelium. A single horizontal incision is made on the palate parallel to the free gingival margin and approximately 3 mm apical to the margin (**Figure 4**). The incision can be extended from the second molar to the nasopalatine papilla if necessary to allow improved access to the connective tissue. With the primary flap elevated, a connective tissue graft approximately 1.5 mm thick is removed and trimmed as necessary to fit the recipient site. Sutures are placed for primary closure of the palatal access flap. The palate is protected by a custom Omnivac stent for two weeks postoperatively. Utilizing this technique, the palate heals with minimal discomfort or complications (**Figure 5**).

The roots of the teeth to be covered are prepared by thorough odontoplasty for debridement and reduction of the facial height of contour. This is done using hand instruments and finishing burs. The roots are then polished with a nonfluoride prophy paste. Chemical modification of the roots is then done with tetracycline or citric acid to remove the smear layer and expose collagen fibrils of the dentin matrix. This will allow subsequent interdigitation of these fibrils with those in the connective tissue graft.[15]

The recipient site is prepared by creating parallel horizontal incisions that extend one papilla width beyond the affected teeth on the mesial and distal (**Figure 6**). The distance between these incisions is 1.0 to 1.5 mm to allow slight coronal positioning of the flap over the graft. A split thickness dissection is carried apically far enough to allow free movement of the flap in the coronal direction. The connective tissue graft is slid between the primary flap and connective tissue and sutured into place with a single 6-0 stay suture in each papilla. Seven-0, 8-0 and 9-0 sutures are used as necessary as secondary and tertiary sutures to facilitate primary closure and graft stability. A periodontal dressing covers the recipient site for one week.

Evolution of the surgical technique to include the surgical microscope and microsurgical instrumentation has increased the precision of these procedures. Microsurgery techniques decrease trauma to the tissues and allow improved surgical closure, thereby improving the outcome and reducing patient discomfort at the donor and recipient sites.[16]

The question remains, does the technique just create a pocket where there had previously been recession. If not, then what sort of attachment occurs between the graft and the root surface? Several authors have shown clinical probing depths consistent with attachment of the graft to the root surface.[17] Histologic case reports of an autogenous graft[18] as well as guided tissue regeneration[19] have shown formation of new bone and connective tissue attachment on the root in the area previously exposed to the oral cavity.

Ridge Augmentation and Preservation

The position of the free gingival margin of a tooth can be corrected with a connective tissue graft, as shown in the prior section. The root of the tooth acts as support for the grafted tissue as well as for the alveolar bone and soft tissue housing. Removal of a tooth results in collapse of the alveolus and causes a shift of what had been the free gingival margin in an apical and lingual direction.[20] Esthetic restoration of missing teeth with pontics or implants often will require reconstruction of this lost tissue prior to placement of the prosthesis. Ridge augmentation techniques have been developed that allow predictable replacement of alveolar tissues lost after the removal of teeth. Ridge preservation techniques, performed simultaneously with tooth removal, can prevent the natural collapse of the ridge and will limit the loss of bone and soft tissue.

Ridge Augmentation

Collapsed ridges can be built up in a variety of ways: soft tissue grafts,[21] bone grafts,[22] guided bone regeneration,[23] alveolar distraction osteogenesis,[24] and combinations of these techniques. The anatomy of the defect and the restorative plan aid in the selection and sequence of treatment options. Seibert[25] categorized ridge defects based on anatomy:

- Class I, buccolingual loss of tissue width with normal ridge height;
- Class II, apicocoronal loss of tissue height with normal ridge width; and
- Class III, combined buccolingual and apicocoronal loss of tissue resulting in loss of ridge height and width.

If a fixed partial denture is planned, connective tissue grafts can be used to restore the missing tissue volume. Slight to moderate Class I and slight Class II defects can usually be corrected in a single surgical procedure. Advanced Class I and most Class II and Class III defects will require multiple staged augmentations to re-establish normal ridge form. When multiple augmentations are necessary, a minimum of three months is required between procedures to allow for revascularization, shrinkage, and maturity of the previous graft. After three months, an assessment is made as to the need for more tissue prior to the final prosthesis. If no further surgery is required, the final

Figure 6A. Slight wide recession on Nos. 7 and 8 with moderately wide recession and a lack of attached gingiva on No. 9.

Figure 6B. Incisions and split thickness flap with papillary preservation.

Figure 6C. Connective tissue graft in place on Nos. 8 and 9. Coronally positioned flap planned for No. 7.

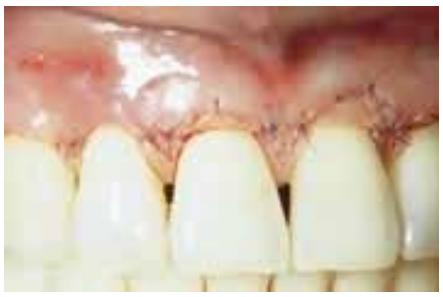

Figure 6D. Six-0, 7-0, and 9-0 microsutures used for flap closure and graft stability.

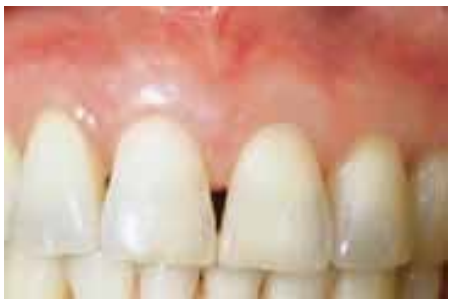

Figure 6E. One-year result with root coverage to the cementoenamel junction, increased dimensions of the gingiva, and inconspicuous blending of the grafted tissue into the site.

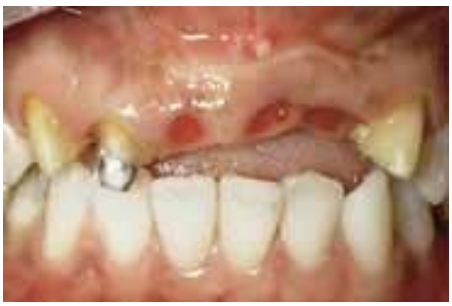

Figure 7A. Ridge defect. Seibert class: slight to moderate III. Note scaring from history of apical surgery and apical position of No. 11 relative to No. 6, which will limit vertical augmentation of the papilla.

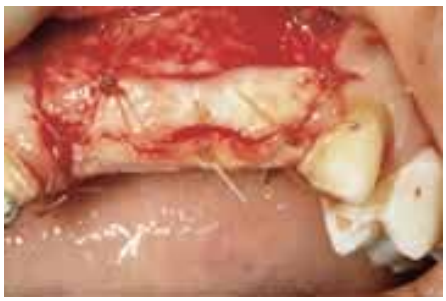

Figure 7B. Two connective tissue grafts laminated for vertical and buccal augmentation.

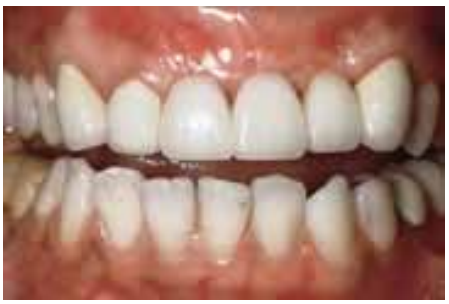

Figure 7C. Five-year result, fixed partial denture with modified ridge lap pontics, compare to ovate pontics in Figure 10C (Restoration by Dr. Bennett Dubiner).

fixed partial denture can be undertaken four months postoperatively.

Surgical preparation of the recipient site in small ridge defects is done with a single horizontal incision slightly palatal to the crest of the ridge extending to within 1 mm of the sulcus of the teeth immediately adjacent to the ridge. The incisions then parallel the sulcus as they swing to the facial terminating at the proximal line angles of the adjacent teeth. This type of incision will maintain the preoperative papillary height. If the papillary height or volume is deficient, the incisions are carried into the sulcus and an attempt is made to increase the height and volume of the papillae in conjunction with the ridge augmentation. A split thickness dissection on the facial is carried far enough apically to allow free movement of the superior flap. As the size of the defect increases, vertical releasing incisions into the mucobuccal fold become necessary, as does a split thickness palatal dissection.

For small defects, connective tissue grafts are harvested from the palate in the same way as a root coverage graft; however, the tissue taken needs to be thicker to restore the volume of the collapsed ridge. If necessary, based on the size of the defect, multiple connective tissue grafts can be laminated onto a deficient ridge during a single surgical

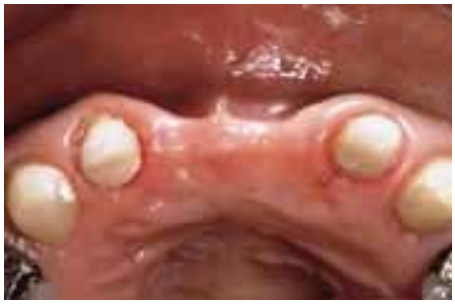

Figure 8a. Preoperative ridge defect. Seibert class: moderate III.

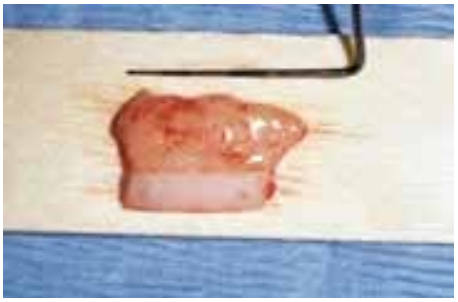

Figure 8b. Combined epithelium and connective tissue graft for ridge augmentation.

Figure 8c. Graft in place prior to flap closure.

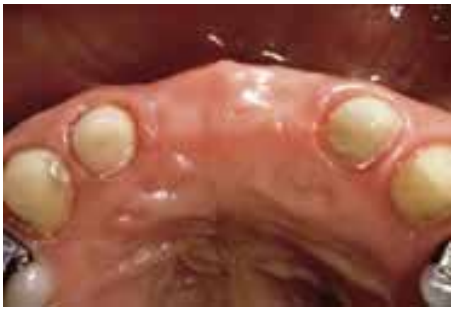

Figure 8d. Three-month result. Note restoration of tissue volume and blending of graft with the native tissues.

Figure 9a. Patient presented with buried implant in place No. 9. Note loss of tissue volume and lack of papilla on the mesial of No. 10.

Figure 9b. One-year result after combined epithelium and connective tissue graft followed by a tissue punch exposure of the implant (Restoration by Dr. Paul Hoyt).

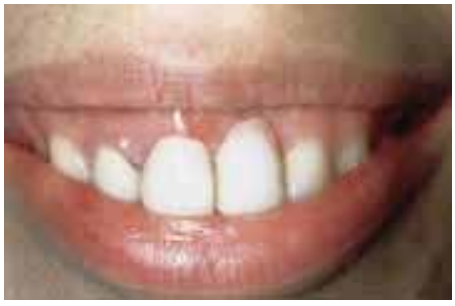

Figure 10a. Preoperative smile, Nos. 8 and 9 are pontics on a stayplate. Note excessive gingival display due to vertical maxillary excess; inconsistent gingival margin levels and lack of midline papilla due to traumatic loss of Nos. 8 and 9.

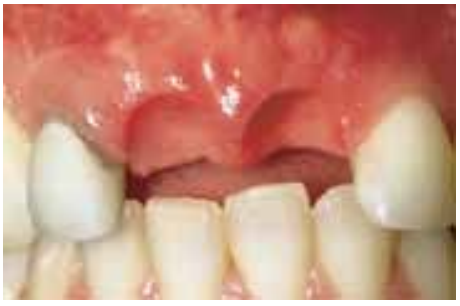

Figure 10b. Tissue appearance after crown lengthening on Nos. 6 through 11 and connective tissue grafting of the ridge followed by creation of ovate pontic recipient sites for Nos. 8 and 9. Note restoration of the papillae and the lack of inflammation in the tissues.

Figure 10c. One-year result, fixed partial denture with ovate pontics. Note symmetrical gingival margins and maintenance of papillary height and volume (Restoration by Dr. Tom Kuhn).

procedure (**Figure 7**). As the defect increases in size, combined connective tissue and epithelium grafts are used to further increase the bulk of the graft and to prevent a coronal shift of the mucogingival junction (**Figure 8**). Connective tissue grafts are sutured to the underlying periosteum to facilitate positioning and stabilization of the grafts. This technique can also be used to increase the volume of soft tissue around previously placed implants, and it can be helpful in the restoration of papillae adjacent to the ridge (**Figure 9**).

If implants are planned, the amount of bone in the site will determine the type and sequence of grafting. In early to moderate Class I defects, with adequate bone for implant stability, a connective tissue graft can be placed at the same time as the implant or during the uncovering procedure. In advanced Class I and most Class II and Class III defects, which lack adequate bone for implant stability in an esthetic position, the necessary bone volume should be restored first. This can be done with guided bone regeneration with autogenous particulate bone grafts, monocortical block grafts, or alveolar distraction osteogenesis. Once adequate

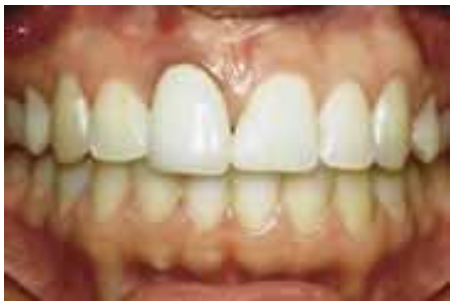

Figure 11a. Preoperative view, No. 8 will be removed. Note chronic erythema and apical position of gingival margin secondary to biologic width invasion, fistula near the apex, and surgical scar from prior apical procedure.

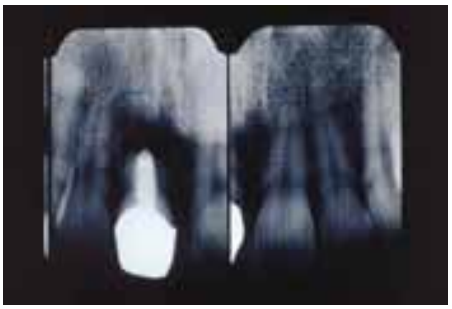

Figure 11b. Radiograph illustrating periapical radiolucency and evidence of prior bone graft near the apex.

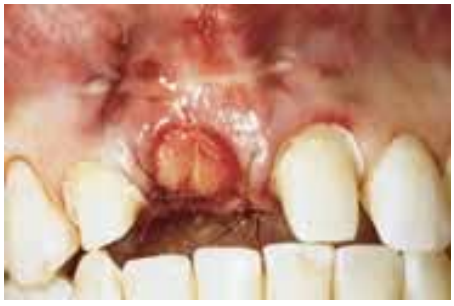

Figure 11c. Connective tissue graft in place after thorough degranulation of socket and placement of a bone graft to preserve the buccal plate.

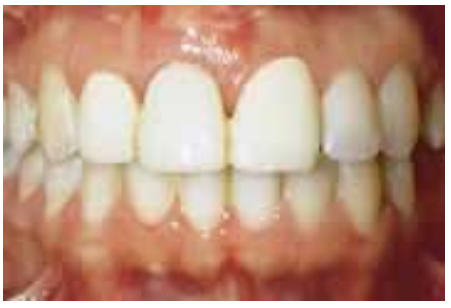

Figure 11d. Three-month healing with provisional in place. Note full buccal contour of alveolus and restoration of marginal symmetry. Apical fistula is sealed (Restoration by Dr. Rebecca Castaneda).

Figure 12a. Preoperative view. Teeth Nos. 8, 9, and 10 will be removed due to root resorption and ankylosis.

Figure 12b. Resorptive defects on roots.

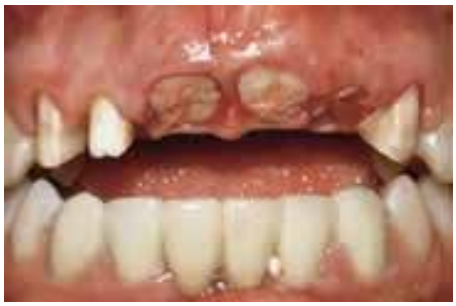

Figure 12c. Tissue plugs in place over bone grafts.

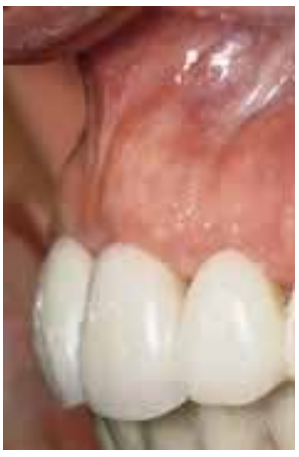

Figure 12d. Four-month healing illustrating preservation of the buccal plate.

bone volume is created for implant stability, the soft tissue is assessed for esthetic harmony and augmented as necessary.

Ovate Pontics

When a fixed partial denture will be used to replace missing teeth, an ovate (egg shaped) pontic will create the illusion that the pontic is emerging from the tissue. Esthetically, this is preferable to the display of the modified ridge-lap pontic that may appear as if it is sitting on top of the ridge. After grafting has restored the tissue volume, ovate-shaped pontic recipient sites are cut into the tissue to a depth of 3 mm using diamond burs and/or electrosurgery (**Figure 10**). The base of an ovate pontic needs to be at least 2 mm away from the alveolar crest to provide enough space for tissue health, which requires 1 mm of connective tissue and 1 mm of epithelium. The provisional restoration is modified into an ovoid shape at the tissue-bearing surface and is placed into intimate tissue contact with the receptor site. Slight positive pressure from the pontic will further form the tissue. In this way, a concave pontic zone is created that will present a contour on the labial that

Figure 12e. Five-year postoperative result with fixed partial denture in place (Restoration by Dr. Al Sze).

resembles the alveolar process, gingiva, and papillae of the adjacent teeth.[26] The convex tissue surface of the pontic is easily cleaned with dental floss.

Ridge Preservation

Ridge preservation procedures are combined soft tissue and hard tissue grafts of extraction sockets done in conjunction with the removal of teeth. The intent of these procedures is to prevent resorption of the alveolar bone and collapse of the soft tissues, thereby reducing the need for subsequent augmentation of a deficient ridge. Extraction should be done atraumatically, preserving as much of the supporting bone and gingival tissues as possible. Thorough debridement of the socket to remove all granulation tissue is followed by cortical perforations of the socket walls to enhance the supply of osteoprogenitor cells to the graft material. An osseous graft is then packed into the socket and covered with a connective tissue graft,[27] tissue plug,[28] or barrier membrane.[29]

Use of a connective tissue graft to seal the socket is indicated in situations where there has been a loss of soft tissue height at the free gingival margin or the papillae of the tooth to be extracted (**Figure 11**). After the tooth has been removed, horizontal incisions are made palatal to the col of the papillae. Split-thickness flaps are then elevated circumferentially around the extraction socket. These incisions extend into the palate far enough to allow ready elevation of the superior palatal flap and extend to the labial far enough to thoroughly mobilize the facial flap. Vertical releasing incisions are often necessary on the facial to provide an adequate degree of flap mobilization. A connective tissue graft at least 1.5 mm thick is placed to cover the osseous graft in the socket and is draped over the surrounding alveolar bone. The connective tissue graft should extend laterally far enough to cover the interproximal bone in order to coronally position and augment the papillae. The graft is placed at least 4 mm under the facial and palatal flaps to ensure adequate blood supply to the grafted tissue and to coronally position the marginal tissues. The connective tissue graft is sutured into position using periosteal sutures, the flaps are positioned to cover the graft, and the papillae are reapproximated over the graft. No attempt is made to cover the connective tissue over the orifice of the socket. The area over the socket is allowed to heal by lateral epithelial migration over the exposed connective tissue surface.

In cases where the alveolar crest is intact and the free gingival margin and papillae are in an esthetically acceptable position, the "tissue-plug" technique can be used (**Figure 12**). The teeth are removed as atraumatically as possible, and osseous grafts are placed in the extraction sockets. Then a tissue plug of epithelium and connective tissue is taken from the palate and placed in the orifice of the extraction socket. The tissue-plug graft must completely fill the opening of the socket and provide intimate contact with the gingival tissues to establish a blood supply to the graft. Horizontal mattress sutures are used to hold the plugs in position. These ridge preservation techniques can also be used in cases of immediate or delayed implant placement (**Figure 13**).

Ridge preservation procedures require that a provisional restoration be provided to the surgeon for the day of surgery. If a fixed partial denture is planned, there are three ways to approach fabrication of the provisional prosthesis. In the first method, the abutment teeth are prepared prior to surgery and individual provisional restorations are placed on these teeth. The restorative dentist also fabricates a provisional bridge prior to the surgery. This bridge is sent to the surgeon. The individual provisional units are removed during surgery, and the provisional bridge is placed at the conclusion of the procedure. The second method is to place a provisional splint on the affected teeth prior to the day of surgery. In this technique, the teeth slated for removal, as well as the abutment teeth, are prepared to receive provisional restorations; and a provisional splint is fabricated that covers all the prepared teeth. At the end of surgery, the provisional is back-filled with acrylic or composite to form pontics where the teeth have been removed. The third method is to cut off the crowns of the teeth slated for extraction flush with the gum line at the same time the anchor teeth are prepared for the provisional bridge. When fabricating the provisional bridge, a pontic is formed over the remaining root. The root is removed during surgery, and the provisional is modified as necessary prior to recementation.

If implants are planned, an interim partial denture is usually used as the provisional restoration. The interim partial denture must be fabricated in such a way to control pressure on the surgical site and to minimize movement during function.

Three months after the ridge preservation procedure is completed, the tissues are assessed. If there is no need for further augmentation, the final prosthesis is begun four months postoperatively.

Crown Lengthening

Patients may present with an excessive or aberrant display of gingival tissues. Periodontal surgery, orthodontics, or orthognathic surgery may be required to improve the esthetics of the smile. A

Figure 13a. Preoperative view. No. 9 will be removed due to a horizontal root fracture. Note slight apical position of gingival margin No. 9.

Figure 13b. Eight-month soft tissue result after tissue-plug ridge preservation followed by an implant. Professional restoration in place and ready to begin the final restoration (Restoration by Dr. Tom Kuhn).

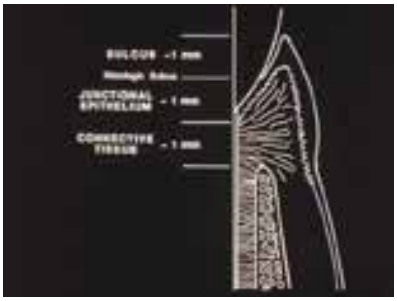

Figure 14. Diagrammatic representation of the dentogingival complex.

Figure 15a. Preoperative smile in a case of altered passive eruption. Note short squat teeth and excessive gingival display.

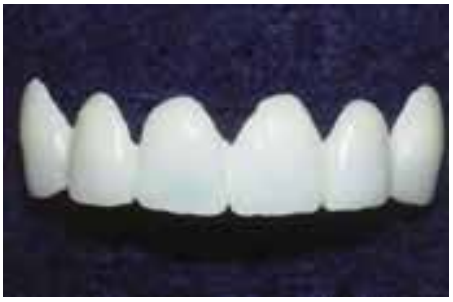

Figure 15b. Surgical guide stent.

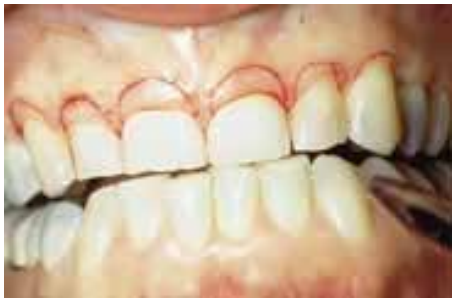

Figure 15c. Gingivectomy incisions placed with the aid of the stent. The goal is to increase the length of the teeth and develop a symmetrical gingival margin. Note that a wide band of gingiva will remain after the gingivectomy.

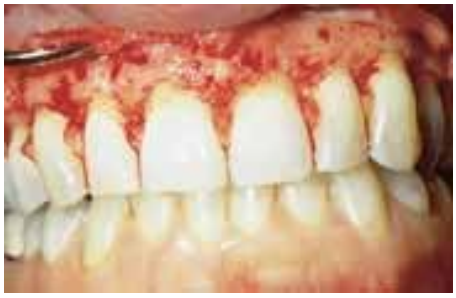

Figure 15d. Flap elevation leaving the papillae in place pre-osseous surgery.

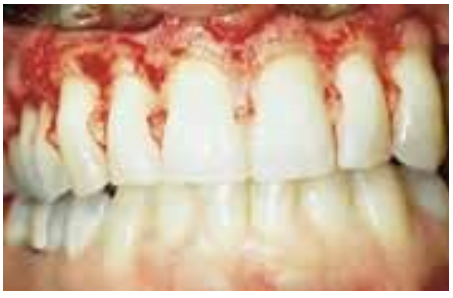

Figure 15e. Post-osseous surgery to establish room for the dentogingival complex and position the gingival margins.

Figure 15f. One-year postoperative view illustrating the characteristics of the ideal smile created by the combination of periodontal and restorative therapy (Restoration by Dr. Michael Hack).

Figure 16. The intracrevicular restorative margin. When the margins of restorations are placed apical to the free gingival margin, they should never invaded the biologic width of attachment but should reside in the sulcus. The circumferential architecture of the soft tissues dictates proper margin placement (JE -- junctional epithelium; CTA – connective tissue attachment).

thorough understanding of the patient's desires as well as proper diagnosis of the case is essential for developing the correct treatment plan. Often a multidisciplinary approach including several of these techniques is the best way to treat a given clinical situation.

Periodontal crown lengthening procedures can modify the supporting apparatus of the teeth through the judicious surgical removal and reshaping of the soft tissues and/or bone. The desired result is an increase in the length of the clinical crown and a concomitant reduction of gingival exposure. This will effect an improvement in esthetics by altering the ratio of the clinical crown to the marginal tissue in favor of the teeth. Crown lengthening in the esthetic zone may be necessary in cases of altered passive eruption, vertical maxillary excess, biologic width invasion, and inconsistent

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free gingival margin positions.

Successful crown lengthening requires an understanding of the biologic width of attachment and the relationship among the alveolar crest, the position of the free gingival margin, and the tip of the papilla. On the facial of a tooth, the biologic width of the attachment between the soft tissues and the root of the tooth has been shown to average 1 mm of connective tissue attachment coronal to the alveolar crest followed by 1 mm of epithelial attachment and then a 1 mm histologic sulcus.[30] (This combination of connective tissue attachment, epithelial attachment, and sulcus is known as the dentogingival complex). Therefore, there is a total of 3 mm from the alveolar crest to the free gingival margin on the labial of a tooth (**Figure 14**). At the midfacial, the crowns of maxillary central incisors and cuspids are 11 to 13 mm long.[31] Taking the dentogingival complex into account, there is 14 to 16 mm from the occlusal plane to the alveolar crest on these teeth. Between the teeth, the position of the interdental contact point, root proximity, and the height of the alveolar crest have been shown to affect the conformation of the interproximal tissues.[32,33] Clinically, one can expect 3 to 5 mm from the alveolar crest to the tip of the papilla. The goal of crown lengthening surgery is to reposition the dentogingival complex to a location on the tooth that is esthetically and structurally more favorable while maintaining the health of the tissues.

Periodontal crown lengthening can be accomplished in several ways: gingivectomy, apically positioned flaps, osseous surgery, or a combination of these techniques. A gingivectomy is appropriate when a wide band of gingiva is present (enough to leave at least 4 mm of gingiva after the gingivectomy) and when the bony crest is at least 3 mm apical to the desired position of the free gingival margin. This 3 mm is necessary so there will be adequate room on the root for the re-formation of the biologic width of attachment and a sulcus. The apically positioned flap is used when there is at least 3 mm between the alveolar crest and the desired position of the free gingival margin, but the entire band of gingiva must be preserved and moved apically. Osseous surgery is used with a gingivectomy or apically positioned flap when it is necessary to remove bone to establish 3 mm between the alveolar crest and the desired position of the free gingival margin.

In a case with adequate gingiva, the first step in surgical crown lengthening is a gingivectomy to establish the proper free gingival margin relationship (**Figure 15**). Once the free gingival margin position has been established, the operator can sound to the alveolar crest to determine if there is adequate distance (3 mm) to the osseous crest for the dentogingival complex. If there is less than 3 mm, then flaps need to be elevated and bone must be removed to re-establish the 3 mm distance between the bone and the free gingival margin. This will ensure long-term stability of the surgical results. In a case that lacks adequate gingiva, the gingiva must be augmented prior to, or in conjunction with, the crown lengthening surgery.

Less than ideal results will be produced if a gingivectomy is used as the sole method of crown lengthening in a situation that requires the removal of bone to establish 3 mm between the desired position of the free gingival margin and the alveolar crest. If bone is not removed, the soft tissue will rebound postoperatively to re-establish the proper dimension for the dentogingival complex. Therefore, the results of this inappropriately done crown lengthening will be short-lived, and the final free gingival margin position will be too far coronal on the tooth.

The restorative dentist can fabricate an acrylic or composite surgical guide stent that clips over the teeth and partially covers the gingiva (**Figure 15B**). This stent will aid the surgeon with the placement of incisions and the removal of bone.[34] The guide stent can also act as a preview device, giving the patient and doctors an opportunity to assess the proposed length of the teeth prior to beginning therapy. Before surgery, the stent is modified as necessary to satisfy the patient's esthetic desires. The stent is used by the surgeon as a template to locate precisely the position of the free gingival margin.

When crown lengthening is necessary only on the labial, a facial full thickness flap is reflected, leaving the interproximal soft tissues and the full height of the papillae in place. Then, ostectomy is performed to create the space for the dentogingival complex. The flap is then positioned and sutured in place. When circumferential crown lengthening is necessary, the flaps need to preserve the height and volume of the interproximal papillae and still provide 360-degree access to the supporting bone for ostectomy. The papillae are incised and elevated intact as part of the facial flap,35 the ostectomy is performed, and then the papillae are repositioned and sutured.

If an intracrevicular margin is planned, the restorative dentist should wait until maturation of the attachment and stability of the gingival crevice prior to the final restoration of the case. The time to full tissue maturity varies among procedures and patients. Postoperative tissue stability can only be ensured by two consistent measurements of sulcus depth and free gingival margin position over time. The interval between these measurements

should be at least six weeks.

Adequate sulcus depth for intracrevicular restorative dentistry may not develop for six months or longer after surgery[36] (**Figure 16**).

Summary

As the demand for esthetic dental procedures has increased, the dental field has responded with improved techniques and materials to address this demand. Periodontal plastic surgery can support the efforts of the restorative dentist by providing a healthy and esthetic dentogingival complex. The scope of periodontal plastic surgery procedures has been outlined to aid the dental team in the proper diagnosis and multidisciplinary treatment of the esthetic dental case.

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Routine Prophylactic Antibiotic Use in Diabetic Dental Patients

Roger E. Alexander, DDS

ABSTRACT There is no scientific evidence in the literature to support the premise that well-controlled, or even moderately well-controlled, nonketotic diabetic patients are prone to infection when undergoing uncomplicated dentoalveolar surgery. Routine administration of prophylactic antibiotics should be considered only in situations where prophylactic antimicrobials would be used for a nondiabetic patient. Poorly controlled diabetics (whether Type I or II), with fasting glucose levels above 250 mg/dL, should be referred for improved control of their blood sugar before nonemergency surgery is performed. If emergency surgery is needed for a poorly controlled patient, then prophylactic antibiotics are prudent, using the accepted principles of such use. Infections in diabetic patients, regardless of their control levels, should be managed aggressively, including possible early referral to oral and maxillofacial surgeons.

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Unproven myths abound in the fields of dentoalveolar surgery and surgical pharmacology. A number of these myths were recently explored and shown to be scientifically unproved or illogical.[1] Another, similar, area of confused clinical guidance relates to the prophylactic use of antibiotics following surgery in diabetic patients. For many practitioners, the guidance provided in the past has been a very vague and generalized axiom that seemingly has evolved over the years. It can be paraphrased like this: "All diabetics, as a group, are more prone to infection and therefore should have prophylactic antibiotics routinely prescribed for all dental surgery." Zoeller and Kadis present this viewpoint as their interpretation of the consensus of the current literature.[2] It is the purpose of this paper to examine the validity of this basic premise.

Diabetes and the Immune System

It is estimated that there are 15 to 20 million Americans (2 percent to 4 percent of the population) who have one form of diabetes mellitus or another.[3] The prevalence has increased significantly during the past 40 years. One of the least common forms is the so-called Type I diabetes, also referred to as insulin-dependent diabetes or "juvenile-onset" diabetes, which affects only 5 percent of the diabetic population. The vast majority of cases are the so-called Type II form, also referred to as "adult-onset" diabetes or non-insulin-dependent diabetes mellitus (even if the patient is using insulin). There are other variants of the disease; and the

reader is referred to current textbooks, such as Little and colleagues,[3] for further information on this family of diseases.

Does diabetes mellitus, indeed, consistently disrupt immune system performance? There are numerous studies and anecdotal reports in the literature on the subject of susceptibility of diabetic patients to infection, but the common denominators in many of those articles suggest the problem is confined to a fairly narrow set of circumstances:

- The patients are largely Type I diabetics who are in poor control and physiologically unstable.
- The infections often involved one or more extremities (usually lower) with notably poor vascular supply.
- The infections studied were pre-existing, requiring therapeutic management (not prophylaxis); few articles address the aspect of giving antibiotics to prevent infections (other than periodontal disease).
- Very few of these reported cases involved oral infections, other than periodontal disease, or involved head and neck infections in well-controlled diabetic patients.
- Many articles are filled with assumptions and contradictions, making them difficult to interpret.4

It is also unclear in many cases whether poor metabolic control was a contributing cause of the infection or actually resulted from the infection.

Overview

The defining of altered host responses in diabetes is hampered by the complexity of the immune system and diabetes itself. In vivo, the various "arms" of the immune system are highly dynamic and interdependent.5 It is overly simplistic to evaluate any single element of the system in isolation and equally difficult to compare studies.5 The literature is replete with contradictory findings, inconclusive results, and disagreement about the ability of the immune system to function normally in well-controlled diabetics. This is a reflection of the heterogenous nature of diabetes and the fact that the inclusion criteria for participants in many studies are often not well-defined. The literature more consistently suggests multiple immune system compromises in a small subset of unstable diabetic patients who exist in poor metabolic states with poor glucose control. Even in those patients, however, the clinical significance of impaired neutrophil function has not been fully determined.[4] Other studies have shown that granulocyte adherence, chemotaxis, phagocytosis, and microbicidal function in patients whose blood sugar levels are aggressively controlled are improved.[6] Unquestionably, glycemic control is important for the enhancement of leukocyte function.

A Medline search of the literature over the past 25 years has failed to uncover any valid studies in which the susceptibility of relatively well-vascularized, oral wounds (such as extraction sites) to acute infections was examined in well-controlled diabetic patients as opposed to nondiabetic patients, except for studies relating to periodontal disease.

Blood Supply

Maintenance of normal oxygenation and nutrition to the tissues and continuous delivery of humoral and cellular components of the immune system to the site(s) are dependent on an adequate blood supply to those tissues.[4] Diabetes is known for its cumulative damage to the microvasculature. Patients with infections in areas of poor vascular supply will not be able to respond to them with the same intensity as a noncompromised patient. In a poorly vascularized extremity, this is a clinically significant problem. With the relatively ample vascularity of the head, neck, and oral cavity, however, vascular compromise has not been shown to be a relevant factor.

Poor blood supply has also been shown to alter cellular components, increase local acidosis, and increase vascular permeability.[4] Interestingly, compromised blood supply to the tissues can also inhibit delivery of an antibiotic to tissue sites, an issue that is rarely addressed by those advocating routine use of prophylactic antibiotics.

Humoral Immunity

Although some earlier studies suggested otherwise, more recent studies show that diabetic patients respond as well as control patients to vaccinations.[4] No correlations between antibody response and patient age, glucose levels, or duration of disease have generally been shown, although one study shows that elderly patients do not respond as well to pneumococcal vaccines and that their antibody levels decline more rapidly.[4] The majority of studies of serum complement in diabetics have found normal or elevated levels.[4] Levels of antibodies against specific microorganisms (such as Pneumococcus) are no different in diabetics than in nondiabetics.[7] Complement deficiency is common in diabetics, but the clinical implications are unclear.[7]

Phagocytic Function

The inconsistent data in the literature are testimony to the difficulty in evaluating phagocytosis, since there are so many different steps in the process. Antigens must be sensitized by antibodies (opsonization), and phagocytes must be able to migrate to the area of infection (chemotaxis) and penetrate the endothelium of capillary walls (diapedesis), and then operate in the acidic environment of the infected tissue(s).

In vitro studies have suggested delayed chemotaxis in both Type I and Type II diabetic patients. Other studies suggest that diabetics may have abnormal chemotactic responses as part of the genetic makeup of their polymorphonuclear leukocytes.[5] Decreased phagocytosis is especially notable when fasting blood glucose levels are greater than 250 mg/dL.[5] On

the other hand, one computer-enhanced chemotaxis study has shown that a diabetic's chemotactic cells move at normal rates.[8]

Defective engulfment and intracellular killing by phagocytes in diabetic patients has been reported in several studies, but in studies of diabetic patients in which Staphylococcus aureus was used, ingestion of microorganisms was only found to be abnormal in patients who were uncontrolled and in ketoacidosis. Other studies show that a phagocytic cell's ability to mount an oxidative attack is reduced in the presence of high glucose levels.[7] Sentochnik and Eliopoulos cite a study that demonstrates a defect in phagocytosis of S. aureus in patients with Type II diabetes but provided no correlation with the adequacy of glycemic control.[5]

Lymphocytic Action

Again, the literature is contradictory. The lymphocyte response to Candida antigen is reported to be normal in diabetic subjects in one study, but another study's results do not support that finding in patients who are in poor control.[4] This impairment normalizes with the reinstitution of good metabolic control. In a study in the 1970s, Gilbert and associates found that diabetics exhibited basal levels of lymphocytes comparable to nondiabetics and that patients initial response to administration of an endotoxin was identical in both groups, but diabetic patients had reduced levels of circulating humoral elements after the third day.[9] The authors stress that the physiologic significance of those findings was undetermined, however.

Periodontal Disease

It has been shown in numerous studies that periodontal disease seems to be more common and more severe in diabetic patients than in nondiabetics.[4,10-12] In one animal study, 48 percent of the diabetic animals were shown to have impaired leukotaxis in the gingival crevices, and several had increased numbers of anaerobic microorganisms.[4] Nevertheless, numerous international studies on the relationship of diabetes and periodontitis have reported varying results.[12] Studies in the United States and other countries consistently fail to find overall differences in the prevalence of periodontal pockets, alveolar bone loss, or tooth loss in diabetic patients when compared to age-matched nondiabetics.[12] These conflicts are noted to be likely due to variations in the types of diabetes, severity, control, duration, and differences in oral conditions among patients. There has been at least one case report of a severe deep neck infection originating from a periodontal abscess. This appears to be an uncommon sequela, however; and it, too, occurred in an uncontrolled, ketotic diabetic.

Patients with controlled diabetes generally respond as well to periodontal treatment as nondiabetics.[12] It has also been shown that Type I patients whose disease is under strict metabolic and clinical control have periodontal complications at a frequency comparable to nondiabetic patients.[13] It further remains unclear and unproved whether a patient's susceptibility to chronic periodontal disease has any validity or relationship to a patient's susceptibility to acute infection following the performance of other dentoalveolar surgery, including extractions, in well-controlled and moderately well-controlled, Type I and Type II diabetic patients.

Response to Infections

Several studies have demonstrated that diabetics cope poorly with staphylococcal and Candida infections of the skin, and one study demonstrates that abscesses persist for longer periods in diabetic mice.[4] Indeed, staphylococcal infections of the skin are twice as common in diabetic patients as in nondiabetic patients with other disabling diseases.[4] Many extremity studies are also complicated by the frequent presence of osteomyelitis of the contiguous bones. The consensus of the literature is that studies of extremity infections in diabetic patients support the concept that compromised peripheral oxygen supply in the presence of an impaired peripheral vascular system and neuropathy contributes more to delayed healing and onset and establishment of infections than any other factors.[4] Again, there are few data that this is also a concern in the relatively well-vascularized oral environment.

Diabetics are thought to have an increased incidence of oral candidiasis,[14] but Fisher and associates found no correlation between level of sugar control and yeast colonization.[15] A study of diabetics and nondiabetics with dentures fails to reveal any statistically significant difference in the incidence of denture stomatitis.[16] Other studies, however, did find more mucosal colonization in diabetic patients, but some studies were flawed in their design and many originated in overseas countries where conditions may not equate to those in the United States.[5]

Postsurgical Wound Infections

One article has estimated that approximately 50 percent of the general population that has diabetes mellitus will require at least one operation during their lifetimes, and approximately two-thirds of those patients will experience infectious complications.[17] There is no reliable data, however, on how many diabetics undergo dental surgery and experience clinically significant odontogenic infections during their lifetime.

A large-scale study was carried out on 23,649 postoperative general surgery patients (both diabetic and nondiabetic), and it was found that the clean-wound infection rate in diabetics was more than five times greater than in the general population (10.7 percent as opposed to 1.8 percent).[18] Babineau and Bothe note that the study was criticized because it did not take other risk factors into consideration, such as advanced age,

nutritional status, levels of control, and co-morbid diseases.[17] In another study of 100 patients undergoing elective general surgery, diabetic patients with elevated glucose levels on postoperative day 1 had a 2.7 times greater infection rate than diabetic patients with more normal glucose levels.[6] In general, however, it is well-accepted that the incidences of infections and wound healing are similar between well-controlled diabetic and nondiabetic patients, except for extremity procedures.[7] In a retrospective study of 9,000 general surgery patients, the primary risk factor for postoperative infection was the presence of cardiac failure or valvular heart disease, which hampered patient mobility.[19]

The literature, therefore, appears to support the concept that when infections occur, they may be more severe and protracted in poorly controlled diabetics, perhaps due to impaired leukocyte function in later days of the infection process and/or compromised blood supply.[9]

It is known that severe surgical stress is accompanied by a marked increase in plasma glucagon, epinephrine, and cortisol. Counterregulatory hormones then increase hepatic glucose release and decrease glucose cellular intake, resulting in a relative hyperglycemic state. This effect appears to be more exaggerated in the diabetic patient than in the nondiabetic.6 It is further known that certain bacteria thrive in a hyperglycemic state. Studies cited by McMurry suggest that gram-positive bacteria (including staphylococcus) thrive in hyperglycemic serum while gram-negative bacteria grow less well, which may partially explain some of the observations that diabetic patients are prone to infections.[20] So, the question is this: How much stress is caused by uncomplicated outpatient dentoalveolar surgery, and is the severity of that surgically induced stress sufficient to trigger that physiologic state? This question has not been adequately addressed in the literature.

Wound Healing

Despite difficulties in interpreting data in the literature, the consensus is that poorly controlled diabetics do have poor wound healing, but adequate control with insulin usually resolves the problem.[20] Data also suggest that increased age and obesity make wounds increasingly prone to infection; but age and obesity are also factors in the development and advancement of the diabetic disease process itself, so it is difficult to separate the effects of one from the other. McMurry cites a study that indicates the percentage of wound infections in diabetic patients is more than double that of nondiabetic patients, but when the data are adjusted for age, the incidences of wound infection are nearly identical.[20] Studies have shown delayed wound healing, poor collagen formation, and poor tensile wound strength in diabetic animals; but these are corrected with restoration of adequate control (i.e., insulin administration).[20]

Optimal Glucose Control

It appears that an optimal blood glucose level for patients undergoing surgery is between a level where the surgical patient is not at risk for hypoglycemic emergencies and a level where wound healing and granulocytic function are not impaired. This level has not been precisely defined, but various authors suggest that the patient should have a serum glucose level at or slightly below 180 to 250 mg/dL.[7,10,20,21]

Discussion

It is widely believed that patients with diabetes mellitus are more prone to infection and other postsurgical complications, and all such patients require routine antibiotic prophylaxis for dental procedures. The professional literature fails to support this premise.

This myth of increased susceptibility to infection in diabetic patients following dentoalveolar surgery may have evolved from misapplication of information in the medical literature that documents severe extremity infections in diabetic patients. Impaired vascularity often predisposes the peripheral tissues in extremities to infection. In the presence of ongoing tissue hypoxia and impaired perfusion in poorly controlled diabetics, any pre-existing immune deficiencies are exaggerated by the presence of compromising neuropathy, which can lead to lack of patient attention to extremity skin wounds, malnutrition, co-morbid disease processes, and difficulty getting systemic antibiotics to the peripheral infection site. There is no reliable evidence, however, that relatively well-vascularized oral wounds are equally susceptible, nor is there any evidence that well-controlled Type I and Type II diabetic patients are at increased risk for postsurgical oral infections (other than chronic periodontal disease).

Many older studies, upon which this myth may rest, derived data from autopsy investigations following infections of the urinary tract, respiratory tree, and extremities; and controls were typically lacking.[5] More recent studies have attributed the complications and increased mortality to cardiovascular disease rather than uncontrolled infection.[5]

Babineau and Bothe note that recent well-designed clinical studies have shown that well-controlled diabetes (Type I or II) is no longer a risk by itself for postoperative surgical complications.16 In retrospective studies, diabetes was not proved to be an independent risk factor for complications from vascular, abdominal, or hip surgery, whether the patient was insulin -- or orally -- controlled.[19,22]

Nevertheless, surgical intervention still creates an increased sense of anxiety in both the doctor and the patient. Diabetic patients, especially Type I, continue to represent a subset of patients that are characterized as high risk. Authors emphasize that it is essential that the patient's blood sugar levels be

optimized prior to surgery. Although these diabetic oral surgery patients are not necessarily more susceptible to wound infections, infections in that population can be more severe and prolonged than in nondiabetic patients.[8,17,23] The question is, therefore, this: If we accept the position that well-controlled (Type I or II) diabetics are not more susceptible to postoperative infections, does the impairment of immune response when infection do occur justify the routine prophylactic administration of an antibiotic in well-controlled or moderately well-controlled diabetic patients following all types of dentoalveolar surgery? Generally, the answer is no.

Dentoalveolar Surgery

Pedersen and, more recently, Alling and colleagues have forwarded the position that well-controlled diabetic patients do not require prophylactic antibiotic therapy for routine oral surgical procedures, and delayed wound healing should not be anticipated in the rich vascular environment of the oral cavity.[24,25] Diabetics who are poorly controlled, however, may be at increased risk and therefore suitable candidates for administration of prophylactic antibiotics. If used, prophylactic antibiotics should be administered prior to the surgical procedure(s) and for a short duration, in accordance with currently recommended usage principles. It is beyond the scope of this paper to review the appropriate application of antibiotic principles, and the reader is referred to other articles for guidance.[26,27] Generally, antibiotics would be used in a protocol similar to that of the American Heart Association for prevention of infective endocarditis.[28] Starting antibiotic regimens after the surgery is completed is not considered an appropriate methodology for prophylaxis.[26,27]

More importantly, diabetic patients should be brought under proper control prior to elective surgery. That step by itself will reduce their risk for infection. It is not necessary to make the patient normoglycemic; as noted, glucose levels at or below 250 mg/dL appear to be acceptable. Levels below 100 mg/dL may incur a risk for hypoglycemic emergencies and therefore should be avoided.[20] In the absence of adequate glycemic control, initiation of prophylactic antibiotic coverage is prudent before the elective surgical intervention, using recognized protocols.

With this in mind, it is very useful for every dental office to have a state-of-the-art fingerstick glucometer to allow accurate chairside glucose testing. These now-automated devices are simple to use, reasonably priced, readily available in any pharmacy, and provide highly accurate readings in 30 to 60 seconds. This is a valuable tool that helps clarify a patient's status and facilitates prudent clinical decisions.

The use of 0.2 percent chlorhexidine gluconate may have some benefit as a presurgical rinse. One study demonstrated that exposure to chlorhexidine gluconate for one minute reduced colonies of Candida in the buccal mucosae,[29] but it is unknown whether preoperative chlorhexidine gluconate use in diabetic patients has any significant impact on reducing the incidence of post-surgical infections. Given the lack of any significant adverse effects, chlorhexidine gluconate use might be worthy of consideration, despite the lack of evidence of clinical efficacy.

Acute Infection Management

When orofacial infection is diagnosed in a diabetic patient, whether well-controlled or not, it should then be treated aggressively. Management of acute odontogenic infections is beyond the scope of this manuscript, but guidance can be found in numerous textbooks and articles in the contemporary literature.[24,27] Management typically consists of:

- Administration of appropriate antibiotics;
- Early surgical drainage of pus;
- Adequate hydration and nutrition; and
- Referral to an oral and maxillofacial surgeon.

In the diabetic patient, the additional aspect of frequent glucose testing and aggressive glycemic control would be essential. Close coordination with the patient's internist or endocrinologist is imperative when dealing with severely infected diabetic patients, so adequate control of the patient's metabolic state can be maintained.[30] Frequently, such patients require admission to a hospital and inpatient management. Antibiotic selection would be guided by the same factors affecting the choices for any other patient. Further elaboration on antibiotic selection is beyond the scope of this paper, and the reader is referred to other references for more information.[24,26,27,31]

Conclusions

There is no scientific evidence in the literature that well-controlled, nonketotic, diabetic patient are more prone to infection than nondiabetic patients when undergoing surgery. Once infected, however, they may have a more severe and prolonged clinical course. Routine administration of prophylactic antibiotics is not necessary in situations where antibiotics would not be considered for a nondiabetic patient, however. Poorly controlled diabetics, with fasting glucose levels consistently above 250 mg/dL, should be referred for improved control of their blood sugar before nonemergency surgery is considered. If surgery is essential in a poorly controlled patient, then prophylactic antibiotics are indicated, following accepted principles of such use. Infections in diabetic patients, regardless of their control levels, should be managed aggressively, including possible early referral to oral and maxillofacial surgeons.

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Two Approaches to the Diagnosis of Lesions of the Oral Mucosa

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ABSTRACT This article describes two approaches to the classification of oral mucosal lesions. One is based on the etiopathogenesis of the lesion and the second on the clinical appearance. These two approaches are compared and contrasted, and their integration is described. Combining these two classification schemas allows an excellent understanding of the various lesions so than an expeditious and correct diagnosis can result. Appropriate management and treatment can then follow.

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The establishment of a differential diagnosis for lesions of the oral mucosa is often problematic. This problem relates to the large number of lesions that may affect a patient and the fact that many occur only rarely. A systematic approach to nosology is crucial. Classically, oral pathology has been taught following the etiopathogenic approach. This approach, as most commonly used in general pathology, considers the basic disease processes or mechanism and the body's response, along with the etiologic factors involved. To approach the classification of disease from this viewpoint is efficacious and allows for effective management decisions. Once the etiology is understood, the treatment can be instituted.

General pathology texts are usually divided into chapters on inflammation and immunology, neoplasia, genetic and developmental disorders, and diseases of the various organ systems (e.g., cardiovascular, gastrointestinal, and liver).1-3 Oral pathology texts traditionally follow this same approach and include other categories specifically relating to oral lesions, such as odontogenic cysts and tumors, and salivary glands.[4-9]

The general etiopathogenic categories can be condensed into four major areas that can be best remembered by the acronym MIND (M = metabolic, I = inflammation, N = neoplastic, D = developmental diseases). This mnemonic reminds the student and practitioner to use his or her MIND to arrive at a correct diagnosis. This approach is condensed

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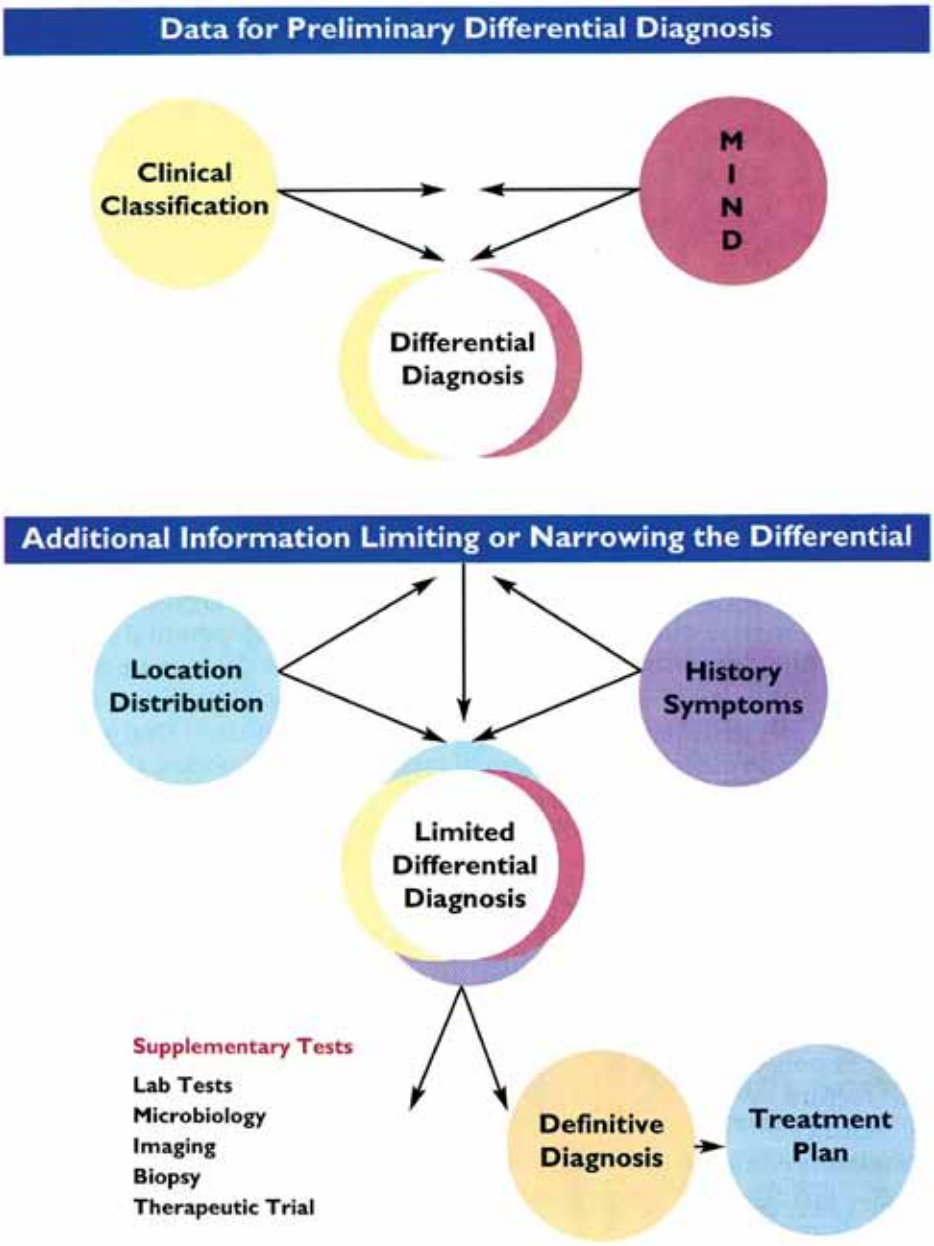

FIGURE 1. The clinical and etiopathogenic classification schemas can be combined to lead to a limited differential or working diagnosis.

and simplistic but provides a good starting point for cerebration and further amplification of this classification.10 The MIND classification system can then be expanded (**TABLE 1**).

Etiopathogenic Classification

The "MIND" Paradigm

- **Metabolic** is a group of oral lesions occurring as a result of various systemic diseases. These diseases may be of either a hormonal or a nutritional nature. The oral cavity may be affected directly, as occurs in Addison's disease, which leads to changes in oral pigmentations of the tongue secondary to hypovitaminosis B complex.
- **Inflammatory** lesions are the most common type and have many subcategories. Classically, these lesions may manifest the cardinal signs of inflammation: redness, swelling, heat, and pain. The subcategories include trauma, reactive, infectious diseases (viral, bacterial, fungal), and the immunologic lesions (allergic reactions, autoimmune and immunodeficiency diseases).
- **Neoplastic** lesions may represent a benign, premalignant or malignant process and therefore cover a large group of both epithelial and mesenchymal tissues that are growing uncontrollably.
- **Developmental** may be of a genetic (heritable) or acquired nature. Either of these may be of a congenital nature (present at birth) or exhibit an oral manifestation as the individual matures and develops. These maldevelopments may manifest as a number of clinical presentations, e.g., clefts and cysts.

Although most but not all areas of etiopathogenesis are included here, this simplified system will allow a quick and

TABLE 1.

The MIND Classification System
Metabolic (systemic)
A. Hormonal
B. Nutritional
Inflammatory
A. Trauma
B. Reactive
C. Infectious
1. Bacterial
2. Fungal
3. Viral
D. Immunologic
1. Hypersensitivity
a. Endogenous allergen (autoimmune)
b. Exogenous allergen
2. Immunodeficiency
Neoplasia
A. Benign
B. Premalignant
C. Malignant
Developmental
A. Acquired
B. Genetic (heritable)

easy review by the practitioner as the four major areas are considered. This system lends itself well to cognitive retention because one learns mechanisms of disease that are mentally imprinted as pictures, that occur as links. Learning the underlying basis of disease this way involves cellular processes in conceptional learning and is far more cognitively retentive than memorizing long lists.

Procurement of Data

The practitioner must then be able to recall the signs and symptoms of the various categories of disease as a pertinent medical history and physical examination are performed. Age, sex, race, and gender may be important factors in data collection. The medical history would include questions regarding the chief complaint, history of present illness (lesion), past medical history, social history, and family history. The lesional history should include duration, pain, periodicity, treatment, and location. As this data is collected and tabulated, various diseases will be considered and deductive reasoning employed.

Physical examination of the head, neck, oral cavity, and particularly of the lesion is then carried out, and several aspects of the lesion must be taken into consideration. The visual assessment and palpation of the lesion of the oral mucosa would include an evaluation of any surface changes in the normal color or texture, along with any alterations in the normal morphology, including swellings, blisters, and/or surface ulcerations. These clinical categories are seen in **Table 2**. Several oral pathology textbooks now include a clinical outline section.[11-15]

Clinical Classification

Setting aside the etiopathogenesis approach to disease classification for a moment, one must consider more practical clinical classification schemes. The categories that follow represent the various tissue alterations or lesions that clinicians observe.

- **White lesions** of the oral mucosa appear so because they represent 1) a pseudomembrane (intrinsic or extrinsic); 2) a thickening of one or more thickened layers of the epithelium (stratum corneum or spinosum); 3) subepithelial inflammatory cell infiltrate; or 4) dense fibrosis. White lesions frequently occur as a result of trauma that can cause either an ulceration or a hyperkeratosis depending on the chronicity of the process. A good clinical test is to determine the wipeability of the white area. Other important factors include pain, distribution, and duration of the lesion. Social habits, including tobacco and alcohol use, should also be ascertained.
- **Red lesions** may represent erythema (increased vascularity) or a thinning of the layers of the epithelium (atrophy). Diascopy or blanching of the lesions may help to differentiate intravascular from extravascular blood. These lesions may also represent inflammation (vasodilation) but may be the earliest sign of an epithelial premalignant lesion (dysplasia).
- **Pigmentation** that presents as black, brown, or blue may represent intrinsic or extrinsic pigments. The common oral mucosal pigmentations are extrinsic pigments due to amalgam filling material or root canal sealers. These generally are gray to black. Intrinsic pigments are melanin and blood products (hemoglobin and hemosiderin). Melanin is usually brown but may occasionally appear blue or black. Hemoglobin is found in red blood cells and is usually blue to purple. Diascopy may also be helpful with these lesions.
- **Ulcerations** occur as a result of a loss of the epithelium and may represent a primary lesion or occur secondary to rupture of a pre-existing lesion (vesiculobullous lesion). Other important distinguishing characteristics of ulcerations are whether they are focal or multifocal, the recurrence pattern, and the location.
- **Vesiculobullous** lesions begin as blisters of varying sizes. Vesicles are less than 5 mm in diameter and are usually of a viral or allergic nature. Viral diseases are associated with a fever in the primary infection and patients are afebrile during the recurrent episodes.

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TABLE 2.

Lesions of the Oral Mucosa (Clinical Classification)
1. White
2. Red
3. Pigmented
A. Brown
B. Blue
C. Black
4. Ulcerative
5. Vesiculobullous
6. Swellings
A. Smooth surface
B. Papillary, papular and multiple polypoid

Bullae are larger than 5 mm and usually represent one of the mucocutaneous diseases that are of an allergic or autoimmune nature. Intact blisters of oral mucosa are rarely seen. Most oral bullae appear as diffuse or multifocal desquamations.

- Swellings are the final group of lesions and range from a smooth to roughened surface (papillary, verrucous, papular, or polypoid nature). The roughened surface lesions usually represent proliferations of the surface epithelium and are frequently of viral origin. The smooth surface lesions are due to a submucosal enlargement. Important parameters are the location, consistency, and presence or absence of pain. Certain swellings have a propensity for a particular certain anatomic site and all of the etiopathogenic factors from the MIND classification may present as swellings in this category. Examples of these are:
- Metabolic – amyloidosis
- Inflammatory – parulis (gum boil)
- Neoplastic – adenoma
- Developmental – exostosis (torus)

The clinical classification approach to differential diagnosis of oral soft tissue lesions can be correlated with histopathologic findings.[15] As discussed for each clinical group of lesions, this correlation will allow the clinician to visualize and understand what is occurring at the microscopic level. When the clinician integrates the clinical and etiopathogenic classification schemas, the various lesions become mentally manageable. Therefore a combination of these two approaches, if performed correctly, will lead to a limited differential or working diagnosis (**Figure 1**). In general, the first step is to place the disease in one of the clinical appearance or lesional categories and then entertain thoughts as to which diseases present with such an appearance, while subcategorizing them as metabolic, inflammatory, neoplastic, or developmental. It may be necessary at this time to perform a supplementary diagnostic test to better delineate the definitive diagnosis. This test may be microbiological, serological, biochemical, imaging modalities, or therapeutic trial or may include a tissue sampling procedure (biopsy). Often a biopsy is necessary to establish the final diagnosis. However, occasionally a biopsy is unnecessary such as for a positive radiographic finding in an amalgam tattoo, a positive candidal microbiological test, or a positive serologic test for syphilis in a mucous patch. Once the definitive diagnosis has been established, the clinician can then administer the proper treatment. The final consideration is follow-up and re-assessment. This is a very important step that allows for the re-establishment of normalcy and ensures that a correct diagnosis was achieved and proper treatment rendered.

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Using Risk Assessment to Customize Periodontal Treatment

Thomas G. Wilson, Jr., DDS

ABSTRACT In recent years, understanding of the multifactorial nature of periodontal disease has taken great strides. Periodontal disease is initiated and sustained by the presence of bacteria, but disease progression is significantly modified by the body's response to the bacteria. This article highlights the emerging evidence regarding which risk factors are predominant in influencing the disease process and how the incorporation of prognostic risk factors in overall diagnosis can help facilitate treatment planning. These factors appear to be smoking, genetic susceptibility, compliance, and diabetes. The first three factors mentioned are the focus of this article. Each is discussed with regard to their role in amplifying the disease process and how this information can be used in clinical practice. By acknowledging the importance of these factors, dentists can consider their patients' risk to allow for more cost-effective planning and treatment. The opportunity to identify high-risk patients and treat them more proactively is significant; the challenge rests with dentists' willingness and ability to embrace the change before them.

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One of the most difficult challenges dentists face is how they can identify, treat, motivate, and sustain the oral health of a wide variety of patients in an increasingly complex practice environment. Dentists are faced with the need to develop appropriate treatment and maintenance plans based on what is often an uncertain prognosis and increasing pressure to deliver predictable, cost-effective outcomes. The explosion of knowledge in the past few years about periodontal disease in relation to other systemic disorders has added to the challenge of making these recommendations. As the mouth becomes increasingly important in connection to the overall systemic health of patients, the identification and treatment of periodontal disease early becomes even more critical.

With the emerging evidence of systemic interactions with the periodontal tissues comes a responsibility for dentists to provide better diagnoses and treatment outcomes for the whole patient. This requires improved techniques for assessing the presence or absence of periodontal disease and better classifications for the types of disease. It also demands more effective ways of identifying risk factors and understanding their impact on disease progression and on the treatments or procedures dentists recommend.

Research in this area has escalated in the past few years. This new scientific information has refined dentists' understanding of periodontal disease.

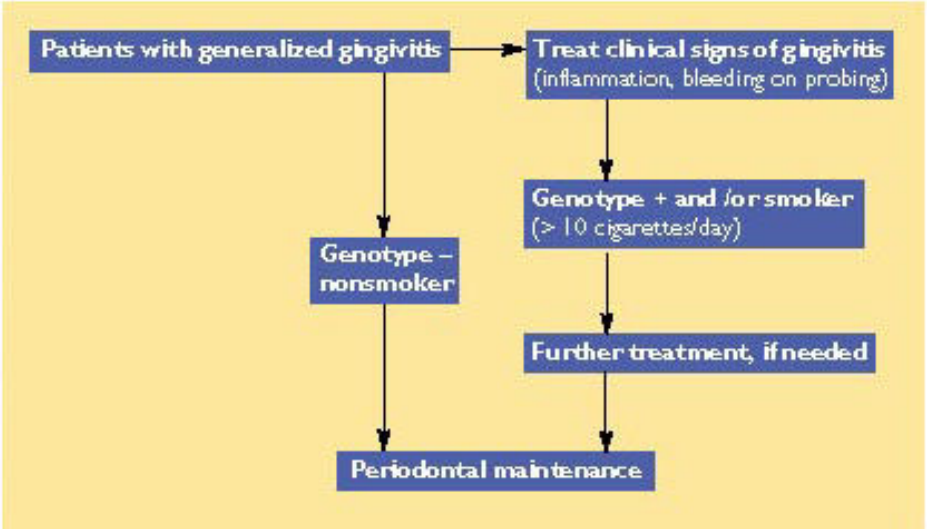

Figure 1. Decision making for high-risk patients with chronic gingivitis: Traditionally patients are placed into periodontal maintenance programs based only on the degree of anatomical destruction. With risk assessment, patients with generalized gingivitis may also enter periodontal maintenance if they are at high risk for periodontitis.

Prominent bacteria associated with generalized, "garden variety" adult periodontitis have been identified in disease initiation and progression. These are Porphyromonas gingivalis, Bacteroides forsythus, and Actinobacillus actinomycetemcomitans.[1-3] At the same time, there is much confusion as to how the presence or absence of bacteria affects disease progression and severity, which can vary considerably from one individual to another. For example, a patient who does not have the suspected pathogens may present with periodontal lesions; or, conversely, the patient may have the suspected pathogens but present with no lesions. Clearly, there is more to the story than a simple infectious disease paradigm can explain.

Current evidence supports an interaction between the bacteria and the patient's systemic response to it. This interaction plays an essential role in the disease expression and progression.[4,5] Stated simply, bacteria are a necessary condition for initiation of disease but insufficient for predicting the progression of the disease or determining how severe the disease will become. Individual patient risk factors – systemic, genetic, and behavioral – play a critical role in the clinical manifestation and severity of the disease.[6,7] Knowing this makes assessing the risk of an individual patient an increasingly important step in the treatment planning process.

The primary reason to assess risk is to understand future disease progression so that appropriate treatment plans can be developed based on current disease status. Other diagnostic tools, such as radiographs or probes, are much more effective in looking backward at the damage that has occurred. Periodontal disease, like most common chronic diseases, is multifactorial. There are many pathways to severe disease. As such, risk assessment is not helpful in determining why patients have developed severe disease (looking backward). On the other hand, risk assessment can be very helpful in determining treatments that alter the future course of disease. Assessing risk is only of value "looking backward" if one wants to establish that a factor such as smoking or genotype may have contributed to the current disease status.

Risk factors do not explain the past, but they do help predict the future. For example, high cholesterol is well-established as an important risk factor for coronary artery disease. This understanding has directly and substantially affected clinical decision making by the physician and behavior changes by the patient; yet, not everyone with high cholesterol has coronary artery disease and not everyone who suffers from coronary artery disease has high cholesterol. The challenge for practitioners is to adopt the right mind-set when assessing risk and not to confuse risk assessment with the diagnostic phase. Both are important, but one looks back while the other looks forward.

This review will discuss the recent evidence regarding the factors influencing the individual patient's response to bacteria and its impact on the clinical severity of the disease. By being able to better diagnose the disease and understand how risk factors amplify its clinical manifestation, practicing clinicians can provide more proactive and targeted treatment. This should allow dentists to serve their patients better than they do when they adopt a reactive approach of "wait, see, react." Specific case examples will help to illustrate the value this approach can offer patients and clinicians. Dentists' challenge is to decide whether they will continue to treat the event only after the fact or manage the treatment process proactively.

The Importance of Bacteria

During the past 40 years, much of the research in periodontics has focused on refining the understanding of the role bacteria play in the onset and severity of disease. From this work, it has clearly been established that bacteria are essential in causing or initiating the disease process.[8,9] Plaque has been used to

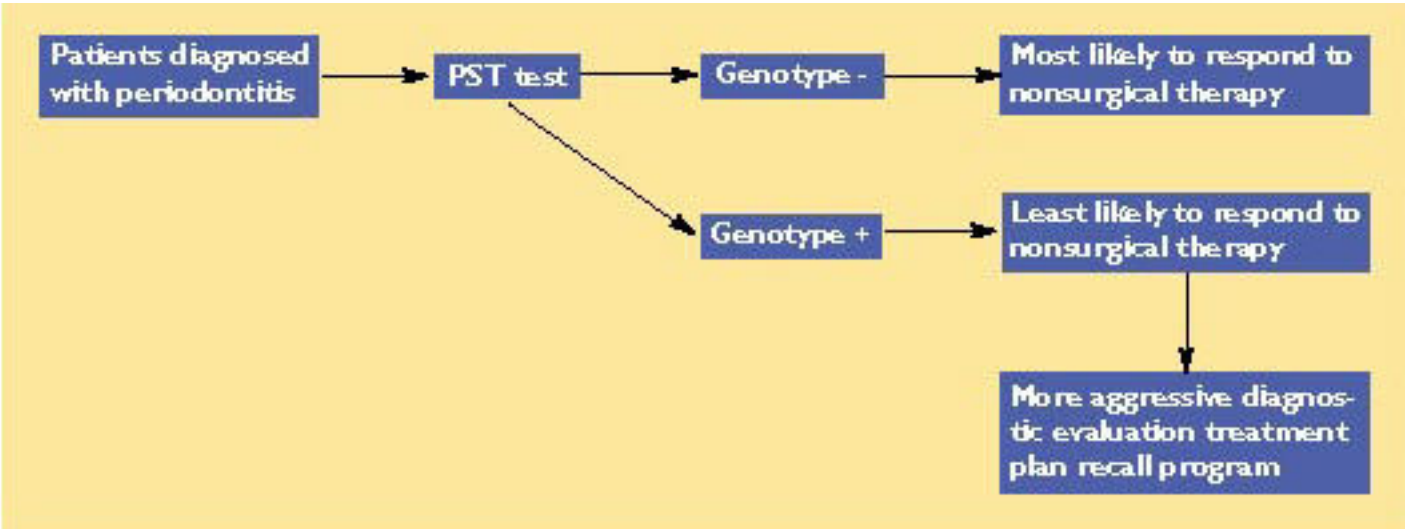

Figure 2. Decision making for high-risk patients with periodontitis: These patients will benefit from a more thorough diagnostic evaluation, aggressive treatment approach, and more stringent recall program.

describe the general collection of bacteria found on the teeth, but the evidence has clearly shown that specific bacteria can be found in individual patients and result in different forms of the disease. This, in turn, has led to specific treatment recommendations such as antibiotics targeted at the pathogens involved.

Attempts have been made to identify different forms of periodontal disease based on the specific bacteria/etiology identified.[3] In spite of these efforts, disease classification is not yet consistently applied. Obviously, the more unclear the diagnosis, the more generic the treatment plans tend to be, i.e., the shotgun approach as opposed to the rifle. As physicians who treat infectious diseases know, identifying the specific pathogens involved can be a key step in prescribing an appropriate antibiotic regimen or other treatment plan.

In general, however, knowing which bacteria are involved has not proved to be very helpful in predicting future clinical severity of the disease. As a result, leading researchers are now focused on understanding the role of systemic, genetic, and behavioral conditions on the progression of periodontitis.

Critical Risk Factors

During the past decade, periodontal research has focused on identifying risk factors for periodontal disease on a factor-by-factor basis. Eliminating the other variables from the analysis allows researchers to more fully examine the association of each factor with the disease process. From this work, strong evidence supports the negative impact of behavioral factors such as smoking and oral hygiene/compliance on the progression of disease.[10-12] Systemic conditions such as diabetes, HIV, and occupational or social stress have also emerged as strong contributors to disease progression.[13-19] In addition, one recent paper has identified local intraoral stress as a risk factor for periodontal breakdown. This author suggests that orthodontic trauma to the periodontium may increase the production of inflammatory mediators, such as IL-1, leading to periodontal breakdown. This suggestion may help explain why occlusal trauma leads to greater tissue destruction in some patients than in others.[20-22] And, most recently, the identification of a specific genetic marker has identified patients with a genetic predisposition to periodontal disease.[23]

Since in the practice environment one rarely treats patients with isolated variables, recent clinical research has focused more on the interaction between these factors (such as smoking and genetics) and the potential synergy that may exist, leading to an even greater negative impact on the severity of disease.[24] Only by bringing these factors together can dentists fully appreciate the impact they have on the health of patients in clinical practice and treat them in ways most appropriate for them as individuals with a different history of disease and risk for future progression.

This review will focus on the three risk factors that appear to contribute most predominantly to the progression of periodontal disease: smoking, oral hygiene/compliance, and genetics. Diabetes is also established as a strong risk factor; but, as it applies to only a limited subset of the patient population, it will not be fully addressed here.

Smoking

Amplification Effect

Smoking has been established as a major risk factor in modifying a patient's response to bacterial plaque. In a recent publication by Salvi and colleagues, the role and adverse effect of smoking in periodontal disease has been thoroughly reviewed.[4]

Also, numerous reports have been published that substantiate the negative impact smoking has on the progression and severity of periodontal disease, as well as on predicting treatment responses and outcomes. Representative findings include:

- Smoking has a more dominant effect on attachment and tooth loss than poor compliance and severe gingival inflammation.[25]
- Results from radiographic analysis of bone loss over 10 years in 350 patients showed that smokers lost bone at twice the rate of nonsmokers. Patients who quit smoking during the study fell in-between.[26]
- Less favorable probing depth reduction, less clinical attachment gain, and increased levels of bleeding on probing have been found in smokers, treated both surgically and nonsurgically, when compared to nonsmokers.[27] A similar treatment response has been seen with dental implants.[28]

The negative influence of smoking has been clearly documented even though the mechanisms of action are not yet well-understood. Most probably, the effects on vasculature, connective tissue, and immune cells compromise the repair and maintenance of the periodontium.

Clinical Risk Assessment

The negative effects of smoking appear to be directly related to the cumulative dosage over time, i.e., the number of cigarettes smoked per day and the number of years the individual has been a smoker. Evidence suggests that anything greater than 10 cigarettes per day substantially increases the risk for periodontal disease and will result in less favorable responses to treatment. Smoking cessation can help, as demonstrated by McDevitt and colleagues, whose findings indicated that risk for future disease was decreased by quitting smoking.[29] Of course, the effect is not clear-cut, since the cumulative effect of years of smoking can still impact bone loss and attachment levels. Studies related to other tobacco products, e.g., cigars and pipes, is virtually nonexistent; but it is reasonable to assume a similar effect.

Oral Hygiene/Compliance

Amplification Effect

As discussed previously, harmful bacterial plaque is the primary etiologic factor for initiating inflammation and periodontal disease. All plaque is not created equal. Unless routinely removed, the immature bacteria accumulate on the teeth; and the plaque continues to mature until it contains harmful pathogens contributing to the destruction of the tissue and supporting tooth structures. In a report recently presented by Socransky and colleagues, the authors concluded that patients with a positive genetic susceptibility present greater levels of harmful pathogens more frequently than those patients who do not have this genetic susceptibility.[30] This suggests a type of catch-22 effect for genetically susceptible patients, leading to increasingly severe periodontal involvement. This finding makes it all the more important to identify patients at genetic risk so dentists can better target their efforts at compliance modification and patient monitoring.

Oral hygiene and compliance are behavioral factors, which means that they can be controlled and/or modified. When niches around teeth are largely free of maturing bacteria and the plaque is removed on a regular basis, subsequent periodontal breakdown and tooth loss can be prevented. Research has clearly demonstrated that patients with periodontal disease and clinical breakdown who participate in a periodontal maintenance program as prescribed by their dentist present less attachment loss and tooth loss than patients who do not.[31,32] This is also true for patients who perform adequate oral hygiene home care as compared with those who do not.[33] So one of the opportunities to manage risk factors, and their amplifying effect on the progression of disease, is to place even greater emphasis on practicing regular home care and on professional supportive periodontal therapy.

Unfortunately, it has also been demonstrated that most patients do not comply with the suggestions provided by their dentist regarding the importance of these procedures. Dentists all recognize that the patient's compliance habits are well-established by the time clinical symptoms of the disease appear. Changing habits requires great and persistent motivation for the patient. Faced with this challenge, it has been reported that compliance can be improved; however, long-term behavioral changes do not usually occur. Most individuals find it difficult to modify their behavior when faced with a life-threatening disease; therefore, it is not surprising that for a non-life threatening disease, behavioral changes are that much more difficult to achieve.[32,34] However, this does not alleviate dentists

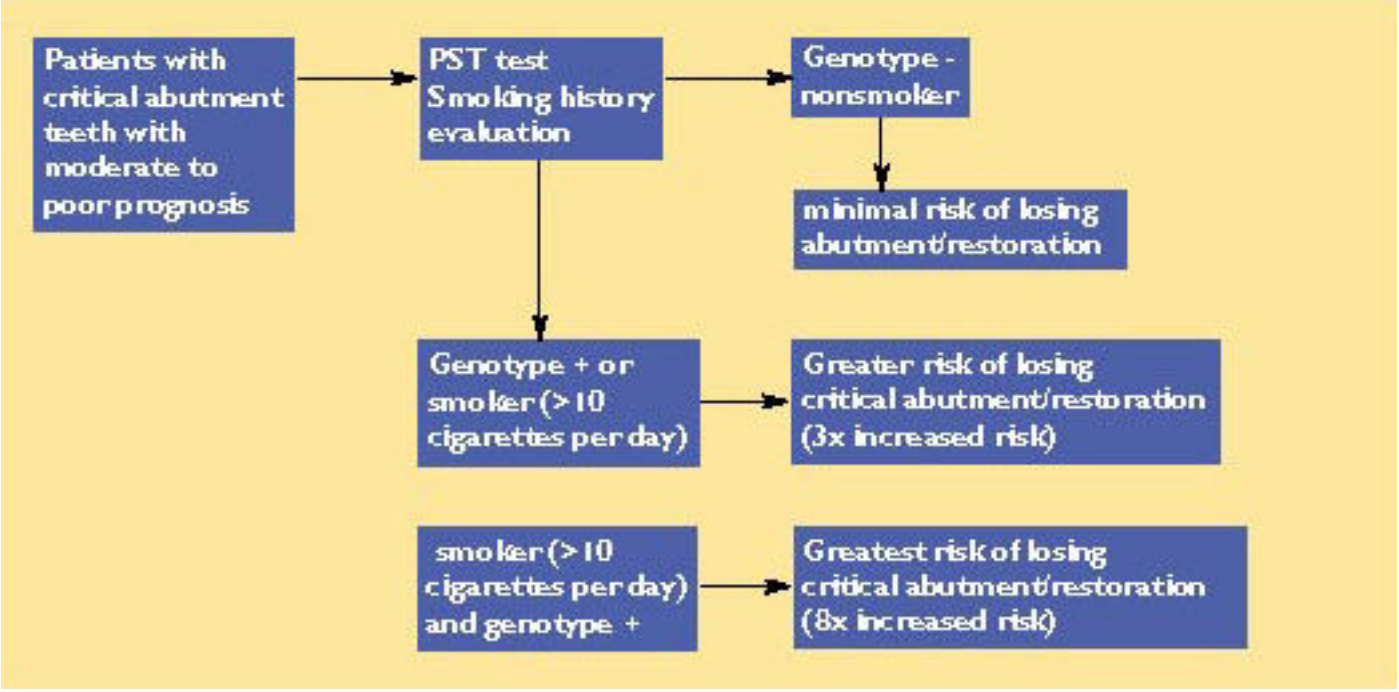

Figure 3. Decision making for high-risk patients with advanced periodontitis who are candidates for complex restorations: Patients at greater risk for losing critical abutments and costly restorations should be thoroughly evaluated diagnostically to assign a prognosis for critical teeth. This evaluation should be considered in conjunction with the predictability of the therapy options (e.g., periodontal or implant surgery) in recommending treatment. In high-risk cases, a more stringent recall regimen is warranted.

from the responsibility to inform the patient of their risk for disease and the importance of compliance in managing the risk. It also does not mean that dentists shouldn't continue to treat the destruction caused by the disease process itself, often with the aim of making oral hygiene easier for the patient, especially in those individuals with increased susceptibility for severe disease and subsequent tooth loss.

Clinical Risk Assessment

Inadequate control of bacterial plaque is the principal cause of periodontitis. Although all patients should comply with professional plaque control procedures and be instructed continuously in home care, patients who present with additional risk factors – e.g., smoking, genetic predisposition – may need to be seen more frequently for follow-up care. Even patients presenting with very early signs of disease may benefit from increased care through a periodontal maintenance program if they are smokers or genetically predisposed to periodontal disease.

Furthermore, adult patients who have not satisfactorily responded to periodontal therapy and who are smokers or genetically susceptible to severe disease may require more careful attention. This may include aggressive plaque control and bacterial culturing; increased supportive periodontal treatment frequency including oral hygiene adjuncts; additional surgical intervention to reduce or eliminate niches; or localized modifiers of host response. These options should be considered and encouraged in the treatment/maintenance plan of high-risk patients.

Understanding the patient's oral hygiene compliance history is important in suggesting the most appropriate treatment options given his or her individual risk for severe disease and tooth loss.

Genetics

Amplification Effect

It has long been suspected and speculated that a patient's genetic makeup plays a role in the progression of common, chronic inflammatory diseases. A series of specific studies on identical twins confirmed this suspicion in the case of periodontal disease.[35,36] With the advent of the human genome project and explosion of knowledge in the area of human genetics, dentists are now beginning to understand the specific genetic factors involved in susceptibility to periodontal disease and how these factors interact with the environment to amplify the disease process.

Genetic research has documented the existence of common but slight variations that occur in human genetic makeup. These genetic variations, called polymorphisms, usually occur in a large percentage of the population and

probably had some selective advantage in the past. This type of genetic variation often affects the host by regulating the body's response to environmental stimuli. This genetic effect is different than the genetic mutations that result in a more causal relationship with the onset and progression of disease. Examples of genetically inherited or genetically "caused" diseases include hemophilia and Huntington's Chorea. A person with the gene will manifest the trait, at least to some level of penetrance or extent. Polymorphisms do not cause the disease; they just influence the way the individual responds to a causative agent in the environment, often amplifying the response the individual with the polymorphism has to the trigger.

Genetic susceptibility to most common diseases, including periodontal disease, involves an interaction between the genetic response and the environmental stimuli necessary to manifest the condition. Examples include the role high cholesterol, poor diet, or lack of exercise play in heart disease in genetically susceptible individuals. Another example would be how lack of exercise, low calcium intake, and reduced hormonal secretion can lead to osteoporosis in some individuals, while having little to no effect in others. Periodontal disease appears also to involve an interaction between the genetic makeup of the host and the environment, with poor hygiene/compliance and smoking being more likely to lead to severe periodontal disease and tooth loss in genetically susceptible individuals than in those who are not.

Recent studies have shed new light on the genetics of periodontal disease and the functional significance of the variations. An important genetic discovery has identified the gene group (Interleukin 1" and Interleukin 1$) responsible for the production of inflammatory mediators. When these mediators are produced in high concentrations in response to a bacterial challenge, there is evidence that this leads to increased tissue destruction or more severe disease.[37,38] Some individuals have a common genetic variation or polymorphism that causes them to produce more Interleukin-1 than other individuals who do not have this polymorphism, even when faced with the same bacterial challenge.[39,40] This discovery has led to the development of a simple laboratory test that can identify those individuals who have the genotype ("positive") and are therefore at higher risk for periodontal disease.

Patients with this positive genotype produce substantially more (two to four times more) inflammatory mediators in response to the same bacterial challenge as patients who are negative for the genotype.[41] This exaggerated response leads to greater and more rapid tissue destruction. In the study that first identified this factor, it was found that 67 percent of the patients with severe disease were also genotype-positive for the gene marker and more than 80 percent of the severe periodontitis could be explained by the presence of two risk factors: IL-1 genotype or smoking.[23] In another recently published study, it was reported that there might be a synergistic effect between these two risk factors resulting in an even greater risk for disease and tooth loss. McGuire and colleagues reported that patients who were either genotype-positive or smokers were almost three times more likely to lose their teeth due to periodontal disease. They went on to report that when the two risk factors are combined, there is a multiplicative effect, resulting in a genotype-positive smoker having an almost eight times greater likelihood of losing teeth due to the periodontal disease than a negative nonsmoker.[24]

Emerging evidence is also identifying the role genetics may play in treatment response and maintaining treatment outcomes.[42-45] The challenge for dentists is to find meaningful ways to use risk factor assessment in their clinical practices for the benefit of patients and their long-term health.

Clinical Risk Assessment

The presence of the IL-1 genetic marker does not cause periodontal disease, it amplifies the response to bacterial stimuli resulting in more severe tissue destruction and an increased risk of tooth loss. The genetic test offers an alternative to the use of the unreliable self-reported family history by directly identifying the genetic predisposition for that individual. Performing the test is straightforward, requiring a fingerstick drop of blood to be collected and sent to a specific laboratory for analysis.

Using the test effectively, however, is a bigger obstacle. The key resides with clinicians' ability to connect genetic information to an overall risk assessment for each individual patient, and then to consider risk in specific decisions they make (such as when to refer and which restorative options to select) in the treatment planning process. Using genetic information in everyday practice will take time; but, just as in medicine, it is clearly an approach whose time has come and one that will be commonplace in the clinical practice of the future.

The PST Genetic Susceptibility Test (Medical Science Systems, Inc., San Antonio, Texas) is a targeted DNA test, identifying only the designated locations in the gene to evaluate the presence

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or absence of this polymorphism. This analysis is conducted through a specifically approved laboratory. The results are reported simply as "PST positive," indicating an increased risk for periodontitis, or "PST negative," indicating a normal risk for periodontitis. Because a person's genetic makeup doesn't change, the test is required only once and provides information that can be used in treating that patient for a lifetime.

Importance of Risk Factor Assessment

Periodontal risk factors may be assessed for all or selected patients. For example, risk assessment and genetic testing may be used with existing or new patients. Patients who already present with severe disease may want family members tested prior to the onset of clinical symptoms to allow for more preventive procedures or earlier intervention. Since risk is commonly assessed and considered in treatment planning for other multifactorial diseases such as coronary artery disease, to patients this may not seem as "new" as it may to dentists.

The identification of periodontal risk factors can provide many advantages and valuable information to clinicians in creating or modifying patients' treatment plans. If patients can be identified as "high risk" at an earlier stage of the disease process, the clinician may want to see the patient more frequently for periodontal maintenance rather than waiting for advanced symptoms to occur. For patients who do not respond favorably to treatments, it can be helpful to understand what risk factors may be interfering with the desired treatment outcome. If a patient is a smoker, it may be important to know if he or she is also genetically susceptible to periodontal disease prior to moving forward with periodontal therapy.

In addition, many patients with periodontal disease require complex and costly restorative work. Smoking and genotype have recently been identified as two important prognostic factors in providing a more accurate prognosis for periodontally involved teeth. If a patient presents with either or both of these risk factors, they should be considered in the treatment/restorative plan and in assigning a prognosis for the remaining teeth involved. In some cases, the restorative plan may need to call for additional abutments as a result, or possibly moving forward with implant therapy sooner where questionable teeth are involved and the patient is clearly high risk.

Minimizing the bacterial niches is just one consideration. In high-risk patients who have teeth with a poor prognosis, advanced periodontal or implant surgery is often clearly justified. In all cases, compliance to a maintenance program is critical to managing and sustaining the desired clinical outcomes.

Specific risk factors (smoking, compliance) have been associated with a less favorable postsurgical healing response.27,46 Although specific data on the surgical healing response in PST-positive patients is not yet available, it is expected that these patients will require extremely good plaque control prior to surgery and throughout the healing phase.

Following are suggestions regarding how risk assessment could be incorporated into the treatment planning process:

1. A traditional examination is performed.

2. A clinical diagnosis is made. There are three possible general designations: health (no signs of inflammation, no attachment loss); gingivitis (signs of inflammation, no attachment loss); or periodontitis (signs of inflammation, attachment loss).[47]

3. Risk factors are assessed.

a. Systemic, including genetic factors and diabetes.

b. Behavioral, including smoking habit and history, oral hygiene habits, and compliance history.

c. Local, including bacterial niches resulting from overhanging restorations or periodontal pockets, signs of occlusal trauma, and other factors.

4. Based on this information, the patient is provided with a suggested treatment plan. A few examples of how this may direct clinical decision making are provided.

Case Examples

Case 1

A patient with a diagnosis of chronic gingivitis who is risk-factor positive.

1. Inform the patient of the potential problems associated with being positive for the IL-1 gene and of the fact that he or she will need to be in good compliance with suggested oral hygiene and maintenance procedures.

2. Reduce or eliminate any additional risk factors, such as smoking.

3. Suggest testing family members.

4. Treat the patient using the traditional approaches of oral hygiene instruction and mechanical removal of bacteria and their products from the teeth.

5. Re-evaluate at 30 to 45 days. If the clinical signs of inflammation are absent, then place the patient in maintenance and see him or her in three months. The patient is re-probed at each maintenance visit.

6. If the clinical signs of disease are still present, then find the genesis of the

problem and eliminate it, if possible.

7. When the clinical signs of inflammation are eliminated, place the patient on a maintenance schedule (an average of three months) and do periodontal probing at each maintenance visit.

Case 2

A patient with a diagnosis of adult periodontitis who is risk-factor positive.

1. Inform the patient of the potential problems associated with being positive for the IL-1 gene and of the fact that he or she will need to be in good compliance with suggested oral hygiene and maintenance procedures.

2. Reduce or eliminate any additional risk factors, such as smoking, where possible.

3. Suggest testing family members.

4. Consider sampling bacteria and have antibiotic specificity testing done if the patient is compliant and is not responding to conventional periodontal therapy.

5. Such patients are much more likely to be candidates for periodontal surgery than those individuals with adult periodontitis who are genotype-negative.

6. Short maintenance intervals (every two months is often needed) after therapy has eliminated as many of the risk factors as possible. The patient is re-probed at each maintenance visit.

Case 3

A patient diagnosed with advanced adult periodontitis who is risk-factor positive.

1. Inform the patient of the potential problems associated with being positive for the IL-1 gene and of the fact that he or she will need to be in good compliance with suggested oral hygiene and maintenance procedures.

2. Reduce or eliminate any additional risk factors, such as smoking.

3. Suggest testing family members.

4. Consider sampling bacteria and have antibiotic specificity testing done, especially if the patient is compliant and is not responding to conventional periodontal therapy.

5. Teeth intended as critical abutments in a complex restoration with moderate to poor prognosis are likely candidates for periodontal surgery. Depending on the tooth location and defect severity, e.g., Class 2 or 3 furcations, where the periodontal procedure is unpredictable (for that clinician), dental implant therapy should be considered.[42]

Summary

While evidence continues to reinforce the role of bacteria in causing periodontal disease, recent studies support the importance of individual patient risk factors in the amplification and progression of the disease, leading to tooth loss. This information, when taken together, provides the dental professional with important direction on how to plan, treat, and maintain periodontal patients, especially by identifying and monitoring high-risk patients more frequently before advanced clinical symptoms appear. Smoking, compliance, diabetes, and genetic susceptibility have been identified as prominent factors substantially affecting the disease process. With the combination of targeted questions and the use of a new genetic test, dentists can now better predict a patient's response to bacteria, improve their ability to assign a prognosis, and intervene more confidently on an individual patient basis. Using combinations of new and previously tested methods, the dental professional can now come closer to the goal of maintaining his or her patients' oral health in comfort and function throughout their lives. As increasing links between periodontal disease and other chronic inflammatory medical diseases are confirmed, the dentist's role may serve an even greater purpose in supporting the overall health of patients.

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Collectible Dentition

Robert E. Horseman, DDS

A $17 million replica of Captain Cook's historical ship the Endeavour sailed into Newport Harbor this year. Had Capt. Cook been standing at the bow like that DiCaprio kid in the $250 million movie "Titanic," he would have been amazed at how much growth and commercialization has taken place there since 1778.

Or maybe not, since he had never seen it in the first place. Instead, he got into a hassle with some natives on the Big Island of Hawaii (formerly the Sandwich Islands, named after the Earl of McDonald) over the theft of a boat, so they killed him. So much for the Aloha hospitality. He'd have been better off dealing with the natives of Newport Bay and might have ended up buying Balboa Island for a couple bucks worth of beads and getting in on the ground floor of the frozen banana concession.

The point is, the fabrication of replicas is Big Business. Whether it is the Endeavour, the Spirit of St. Louis, Dolly Parton's bra or Archie Bunker's chair, make an exact replica and the world will beat a path to your door and your coffers will runneth over. If you are unable to acquire any suitable coffers, the money can be deposited directly into your account.

From a historical point of view, what dental artifact would be most likely to lend itself to replication? The answer, of course, is George Washington's teeth. Information about the dentition of all succeeding presidents is sparse, historians preferring to delineate the boudoir proclivities of our leaders instead. An inquisitive reporter recently asked our current president about the state of his teeth, only to have him equivocate, stating, "Depends on your definition of teeth."

We have had well over 200 years to study Washington's teeth because their owner, feeling that things had to be better in the Great Beyond, gladly left them behind. There are only four sets of Washington's dentures known to exist; one of which resides in a classy glass cube at the Samuel D. Harris National Museum of Dentistry in Baltimore. The whereabouts of the other three sets is questionable. Perhaps John Greenwood, Washington's dentist, sent them out to the lab for a reline and they've not returned yet.

George had only one remaining natural tooth when he was elected president. It was not thought fitting for the Father of Our Country to deliver the State of the Union address looking like Ollie from the "Kukla, Fran and Ollie" show popular at the time. All the other heads of state around the world, many of whom had as many as four or five teeth of their own, would have poked fun at George. Potentates and kings can be so cruel.

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John Greenwood was commissioned to make full upper and lower dentures with Delta picking up 50 percent of the fee after a six-month qualification period and the meeting of the deductible. Delta wanted a radiograph of Washington's one remaining tooth, but the X-ray hadn't been invented yet, so the tooth was posted to them in a little green box with an image of the Tooth Fairy engraved on the cover, along with suitable documentation and a request for an estimate of benefits. George's portion, after deductible, came to $3.79.

With that background, you will understand why we decided to make a replica of the Washington dentures and maybe go into the museum business ourselves.

Fortunately, we have an ideal patient, one Filbert Fischbyne. We have made Mr. Fischbyne at least six sets of teeth, none of which have been satisfactory, but he liked the notion of being part of history when we explained our plan. After taking the necessary impressions in alginate because we didn't have any beeswax, or whatever was in vogue in 1778, the models were sent off to the lab with detailed instructions. Shortly after, the phone rings.

Lab: "Doc, couple questions on this Fischbyne case."

Us: "Shoot."

Lab: "Lessee (reading from lab slip), you're asking for cast gold base, hippopotamus bone, elephant ivory, eight assorted human teeth and a couple springs, right?"

Us: "No, the base is swaged, whatever that is, and the teeth are to be attached with little wooden pegs."

Lab: "Attached to what, Doc? The hippo bone or the elephant ivory? And how come only eight teeth? What about the other 20?"

Us: "We'll get back to you."

This is going to be tougher than we thought. A study of pictures of Washington's teeth reveals little, except that the anterior teeth are square, like Chiclets, and it's hard to tell whether they are composed of real enamel, hippo bone or ivory. We can see the springs pretty clearly, but the mechanics of their use is puzzling. Would they stretch upon opening the jaws, creating a tension, which would then cause the dentures to snap together whether George was ready to close or not?

We have never seen a picture of President Washington with his mouth open, a presidential condition not noted since the departure of Calvin Coolidge in 1929.

Us (to lab): "How you coming with the Fischbyne case? It's been six weeks."

Lab: "Had a little trouble with the springs, Doc. We could only find garage door and screen door springs. So we cut down the screen door springs a bit, and if this Fischbyne guy has enough Fixodent he can probably get his teeth open about a quarter inch. Also, elephant ivory is a prohibited import, so we cut up some pool balls. You may have a little shade match problem, but the numbers won't show."

The Fischbyne/Washington case is in. When Filbert dons the powdered wig, there's a remarkable resemblance. The nose isn't quite right, but he's got the grim look down pat. He says it's because the "dang things don't fit," but we think it's because Delta denied payment based on the fact that he's had a half dozen other dentures inside their five-year limit. Also they said our $9,745 fee falls outside the 90th percentile range for our area.

If you are interested in obtaining an exact replica of the famous George Washington teeth (with a spare set of springs), please contact Mr. Fischbyne or this office.