CDA

Crown Lengthening
Osseous Surgery
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Current Views on PERIODONTAL THERAPY

David F. Levine, DDS

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Doing the Right Things

Jack F. Conley, DDS

The December 1998 "Views" column of Journal of the American Dental Association Editor Lawrence Meskin brought forth some very positive news. Dr. Meskin noted that "After years of flat or even decreasing take-home income (for dentists), substantial increases recently have been noted." He reported increases of 31 percent for general practitioners and 40.9 percent for specialists since 1986. Further, he noted that these increases exceed income growth in other health professions, with the average real income of dental GPs surpassing that of physician family practitioners! Meskin's data also suggests that the smaller, traditional dental office demonstrates economic advantages not seen in the larger multidentist practices. The economies of a larger scale operation apparently are more difficult to achieve in dentistry than in other fields.

This positive news will undoubtedly disappoint the gloom and doomsayers we encounter from time to time who allege that dentistry has retreated from the position of prestige and income expectation it once enjoyed. The point to be made here is that dentistry and many colleagues individually and collectively must be doing the right things! As examples, we list education of the public (the dental patient) about the importance and value of the service dentistry provides, fluoridation, and the continuing efforts of the organized profession to support a strong code of professional ethics and develop appropriate standards of care. Without these strengths, the private practice of dentistry would certainly not be sought after by increasing numbers of dental school applicants, nor would the statistics discussed by Dr. Meskin be possible.

Another one of the "right things" the profession must ultimately come to an agreement on if dentistry is to continue to experience the status of income and respect that it has earned, is the concept of continuing competency assessment. For more than four years, leadership and a dedicated committee of the California Dental Association have actively debated this issue and its many ramifications. These deliberations resulted in a voluntary assessment program (QUIL3) and, most recently, a CDA position paper titled "Concepts of Continued Competency," which was approved by the 1998 CDA House of Delegates.

It is doubtful this position paper will slow public interests outside of dentistry and other health professions from seeking active mechanisms to periodically evaluate the competency of health care practitioners. As an example of efforts on behalf of the public interest, in November 1998, the Pew Health Professions Commission released a proposal that, if carried forward, would require a written examination and require state regulators to conduct in-office inspection of treatment procedures and patient records at least every six years for physicians and "other health workers." The chairman of this independent commission, which has no power beyond persuasion, is George Mitchell, a former U.S. Senate majority leader. He stated that "Once you are in the club, you are in forever. We became convinced there is today, a public system which isn't protecting the public."

Whether or not the initiatives of "persuasion" from this commission are successful in achieving the stated goal, this issue will continue to confront the dental profession. The position statement recently approved by the CDA House was the latest

compromise affecting association policy on this controversial issue. It takes a position that must be more visible than secluded residence in the CDA Policy Manual! It does acknowledge that activities such as the aforementioned Pew Commission report will be occurring and must be monitored: "Given present trends related to post-licensure competency assessment, the association will best serve its members by closely monitoring regulatory and legislative bodies and managed care organizations which may seek to impose mandatory continued competency."

Many CDA resources were expended in developing standards and methodologies that could serve as a model. While QUIL3 resulted from these efforts, even the recent position statement had to survive numerous revisions over a two-year period before gaining approval.

Given the high level of dissatisfaction in dental circles with any discussion of "continuing competency" (yes, some colleagues even sought to adopt alternative terminology to use for "continuing competency," deeming its very mention unacceptable), the final sentence in the position statement is the most important and far-reaching: "California Dental Association should make every effort possible to have a proactive and participatory role in the process of researching, defining, and developing such programs [continuing competency assessment]."

If the good news discussed by JADA Editor Meskin is to be continued into the future, the profession will need to reject the complacent approach of the position paper and instead seek to carry out the proactive intent of its final passage.

To date, the proactive approach to this issue has been controversial within the profession. However, we believe it to be the "right thing" in shaping a successful future for the dental profession.

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Children Reap Benefits From One Man's Desire to Help

By David G. Jones

Perhaps it is fate, destiny putting the right person in the right place at the right time.

More likely, it is the result of thorough training, hard work and a passionate approach to providing care for those who seem to need it most.

Regardless of the confluence of forces making it a reality, Jerry L. Lanier, DDS, is making a difference in the lives of thousands of children. Through his two clinics in the Los Angeles area and his far-reaching program to provide care for underprivileged children, Lanier is fulfilling his long-held desire to offer the possibility of better oral health to children who otherwise might not have it.

Lanier, 43, was one of 11 children born to uneducated parents in a backwoods North Carolina town. He attended Meharry Dental School in Nashville, Tenn., on a scholarship and originally planned to specialize in oral surgery following his 1983 graduation. But four years of working on youngsters at a children's dental clinic changed his mind.

"While working at a clinic in a New Orleans housing project, I saw so many kids who needed treatment and couldn't afford to go elsewhere," says Lanier, a member of the Los Angeles Dental Society. "I realized there was a lot of suffering going on, and it made me realize this career was something special."

After working for the Public Health Service in New Orleans, Lanier moved to Los Angeles in 1991 and worked in a variety of dental offices. Finally, he struck out on his own.

"I was riding around and saw an abandoned dental office for lease in a low-income neighborhood," Lanier says. "I couldn't afford a lot, this place was affordable, and I saw kids up and down the streets, so I didn't need to do any demographic study. I just looked for the strollers."

Lanier wanted to give his new clinic a catchy name, one that clearly communicated a high level of care and commitment to his young patients.

"I wanted to give the clinic a name that explained our entire mission," Lanier says. "With a three-word name, Kids Dental Kare, I wanted people to know we are there to treat kids, and the care we provide was something special, just for kids."

Soon after the clinic opened, it was February, Children's Dental Health Month. Lanier decided to make a concerted effort to establish relations with the mostly Hispanic and Armenian community surrounding the clinic.

"I wanted within the month to go to every school in the area," Lanier says. "I gave away toothbrushes in every classroom and spent almost every morning going from one school to the next to meet with school nurses. They are on the front line and see the kids with toothaches and mouths in terrible condition."

Lanier's registered dental assistant developed a skit to present to the schoolchildren. Now, according to Lanier, many schools are requesting presentations.

"She goes to the school in her RDA uniform and uses a hand puppet to demonstrate proper brushing technique while singing a song in Spanish to the children," he says. "Sometimes I take my guitar along and play, and the kids love being involved."

After five years of work at the clinic, Lanier and his associates have treated more than 12,000 children. A year ago, while gearing up for a new office opening, Lanier wanted to make his young patients feel more at ease. Today, the 40 patients he and his associates treat daily have a lot of entertainment choices.

"The new office has a movie room with surround sound, a video room with game stations, and quite a large waiting area with lots of play toys," he said. "We even

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have a TV in each of the 17 operatories with intraoral cameras."

Lanier also operates a mobile screening van that stops at schools, health fairs, and anywhere in the area where there is a group of 20 or more children. He and his associates -- general dentist Michael Rice, DDS; and pediatric dentists, Michael Vert, DDS, and Scott Fishman, DDS -- are busy: They do at least 50 screening events a year, for a total of about 10,000 screenings annually. At the screenings, they give away toothbrushes, balloons and dental education kits. He accepts no donations from outside sources to support his efforts.

For Children's Dental Health Month 1999, Lanier plans to participate in four health fairs with area physicians, the Red Cross and other agencies, and to visit up to two schools a day to do screenings, for a total of about 50 screenings and presentations during the month.

"We'll also give out up to 50,000 toothbrushes and work to educate a lot of parents during that time," he said. "I believe one of the things lacking most in children's dentistry is education. Most of the time parents don't know anything about caring for primary teeth, so we want to give them a little education. Our focus during the month is to educate parents and raise awareness."

Discipline is the Key to Investment Highs and Lows

Following a disciplined approach is one of the first rules of successful investing, particularly with stocks. Two time-tested investment strategies, based on stocks in the Dow Jones Industrial Average, have shown that discipline can pay off over time. These strategies are known as Top 10 and Low Five.

Under the Top 10 strategy, an investor buys the 10 highest-yielding common stocks in the Dow Jones and holds them for 12 months. After 12 months, any stocks that are no longer among the Top 10 are sold, and any that are new to the list are bought.

With the Low Five approach, an investor purchases the five lowest-priced of the 10 highest-yielding Dow Jones common stocks and holds them for 12 months, after which time the investor makes readjustments so that he or she continues to hold the five lowest-priced of the 10 highest-yielding Dow Jones stocks.

Here are four reasons that these strategies appeal to investors:

1. The 30 companies in the Dow Jones are large, well-known and the "blue chip" names.

2. When buying the highest-yielding stocks, investors are in effect purchasing issues that may be out of favor. The high yield can mean the share prices are depressed and have potential for appreciation.

3. The yields may provide support for the stock prices in down markets because stocks offering moderate dividend yields may perform better during weak markets than stocks that do not pay dividends.

4. The strategies may be carried out either by individual investors or, for sometimes as little as $1,000, through professionally selected fixed portfolios of securities offered by major financial firms.

(Please note that the Dow Jones Industrial Average and Dow Jones are the property of Dow Jones & Company, Inc., which is unaffiliated with and has not participated in any way in the creation of the Top 10 and Low Five strategies or any products based on these.)

Marios Gregoriou is associate vice president and financial adviser with Morgan Stanley Dean Witter in Sacramento. He can be reached at (800) 755-8041. Information in this article was obtained from sources considered to be reliable. This article does not constitute investment advice. Consult an investment adviser before making investment decisions.

Singin' the Managed Care Blues

Just as managed care has come to dominate medicine and is making greater inroads in dentistry, HMOs' biggest musical critic has segued into the dental arena with his latest tune, "Mastoid Sally."

Dr. Sam Bierstock and his band of "preferred music providers" have added a dental ditty to their repertoire of managed care blues tunes. They've recently recorded the CD single "Mastoid Sally." The parody is a follow-up to the band's CD, "Minimal Service CPT 99211," which also bemoans the confusion and red tape often associated with HMOs.

"Mastoid Sally" is a foot-tapping, teeth-grinding tribute to dentists sung to the tune of "Mustang Sally." The band's keyboard player, Dr. Jimmy Pantel, is a dentist.

"We wanted to do a song specifically for our colleagues in dentistry and otolaryngology," Bierstock says. "Not everyone realizes that dentistry has many concerns about managed care just as the rest of the health care industry does."

With managed care issues dominating headlines and emerging as a top concern for millions of Americans, Bierstock, a former eye surgeon, says he recorded the song after it was repeatedly requested at performances and on his web site. He says people enjoy the humorous twist to the song, which describes Sally's ongoing battle with bruxism and TMD. In the song, the dentist admonishes:

Mastoid Sally, guess you better slow that chewing down.

Mastoid Sally, guess you better slow that chewing down.

You been grinding in your sleep girl.

Guess I better do a bonding or a crown.

Bierstock's satire has not escaped

the attention of politicians who are hotly debating health care issues on Capitol Hill. Rep. Greg Ganske, R-Iowa, who co-authored the Patient's Bill of Rights, distributed a copy of Bierstock's "Minimal Service" CD to every member of Congress, noting in an attached letter that "a little levity always helps when you are discussing the subject of health care."

Bierstock agrees and has used this premise to embark on a national tour that has taken him and his band across the country to medical conventions and corporate events to spread his message.

"I realized the medical profession was in serious need of a good laugh," Bierstock says. "But we're also trying to help people understand how managed care really works -- or perhaps why

it doesn't."

The "Mastoid Sally" CD single costs $4.25 plus shipping and handling and can be ordered by calling (888) 426-7529 or from Bierstock's web site at www.managedmusic.com.

Original songs from Dr. Sam and the Managed Care Blues Band include:

- "You Picked a Fine Time to Leave Me Blue Shield"
- "You're One Hip Mama ('Cause They Won't Pay for Two)"
- "I'd Love to Kiss You Baby, but I Just Came Across Your Medical Records on the Internet"
- "If You Won't Refuse Treatment We'll Find Someone Who Will"
- "What Now My Glove" (a digital recording)

Managed Care Profits Expected to Rebound in 1999

Higher premiums will return the managed care industry to modest profitability after four tough years, a new study finds, but the market "will ruthlessly weed out under-performing organizations, both not-for-profit and for-profit."

"Beyond HMOs: the Outlook for Managed Care in 2001," published by Corporate Research Group, Inc., forecasts a six-fold increase in industry profits in 1999, to more than $2 billion.

With premiums expected to rise 8 percent to 9 percent in 1999, the report says, the managed care industry can expect revenue growth of 15 percent to reach a projected $173 billion in 1999 revenues. The three largest for-profit managed care companies are United HealthCare, with projected 1998 revenues of about $17 billion; Aetna U.S. Healthcare, projected revenues of $14 billion; and Cigna Life & Health, projected revenues of $12 billion. Kaiser, with revenues of $14.6 billion, is the largest not-for-profit.

Corporate Research Group projects that the managed care industry will finish 1998 with 765 million members, up 8 percent from 1997. Explaining the industry's recent woes, authors Carl Mercurio and Efrem Sigel note, "With HMOs enrolling more elderly, poor and sick members, utilization soared, as did medical costs. ... Profit margins shrank. ... Mega-mergers aimed at improving financial performance often had the opposite effect."

Over the next three years, large managed care companies will continue to prune operations, such as money-losing Medicare or Medicaid plans. "Successful companies will have to meet escalating demands for quality" while controlling costs, and will have to satisfy "multiple constituencies of members, employers, providers, government regulators and investors and creditors," the report says. It predicts that dozens of HMOs will go out of business by 2001.

Intervention Helps Teen Athletes Quit Spit

High school baseball players are nearly twice as likely to stop using spit tobacco when dentists or dental hygienists, as well as their teammates, actively intervene than when they don't, a new University of California at San Francisco study has found.

The study, reported at the International Association for Dental Research meeting, found that 27 percent of spit tobacco users stopped using the potentially cancer-causing substance for at least one year when dental health professionals, with the help of teammates, intervened. About 14 percent of the athletes who received no intervention quit using spit tobacco, which includes chewing tobacco and snuff.

The study tracked baseball players at 44 high schools throughout rural California. Dental health professionals intervened at 22 of those schools. There was no intervention at the other 22.

"High school baseball players who participated in a peer-led team discussion of the negative health effects of spit tobacco use, and who received an oral cancer screening exam by a dentist or dental hygienist who pointed out to players sores in their mouths related to spit tobacco use and advised them to stop their tobacco use, were twice as likely to stop using than those players who received nothing," says Margaret Walsh, EdD, UCSF professor of dental public health and the study's principal investigator.

The study's results, Walsh said, show that oral health experts must become more aggressively involved in teaching youths the risk of using spit tobacco.

The study was funded by California's tobacco tax.

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Pack Your Bags and Make Your CPA Smile

Taking your spouse to Paris for a big dental meeting is one way of writing off your anniversary trip, but it's not the only option.

"Mixing business with pleasure is one of the wisest tax moves you can make," according to Ken Rubin, CPA. "It's fun and easy to structure your vacations so they'll be tax deductible."

The most obvious way to do that is to attend dental continuing education seminars and conventions in places such as Hawaii and Aspen, Rubin says in Facets, August/September 1998. A less obvious method is visiting dental offices at the vacation destination. According to Rubin, if you can establish that the primary purpose of your trip was to visit dental offices, the trip is tax-deductible. Some reasons for visiting the offices could include observations and discussions with dentists about the following items:

- Office design;
- Employment or partnership opportunities;
- Marketing methods;
- Specific dental procedures; and
- Practice management issues.

Rubin strongly recommends getting follow-up letters from the dentists you meet, detailing what was discussed. According to Rubin, you are required to spend at least four hours per day on business-related matters for the entire day to qualify as a business day. If your spouse is employed by your practice, his or her expenses can also be deducted.

Because tax rules related to travel are complicated, Rubin urges dentists to consult their CPAs to help with the tax planning aspects of vacations.

The Mighty Mouse of Marketing

Ten reasons why your business card is your best promotional device:

- It's your first (and sometimes only) piece of promotional material, usually printed as soon as the business starts.
- It is your cheapest advertisement -- a boxful goes a long way.
- It has a wide targeted distribution -- it's mostly handed out face-to-face.
- It sets the business' style and format, which is then echoed on stationery and products.
- It is a basic sales tool, uncomplicated and flexible.
- It is the most frequently used marketing tool for small businesses.
- For many businesses, it generates more patients and referrals than any other form of advertising.
- It is versatile. It is easy, quick and inexpensive to tailor for different markets or purposes.
- It is expected. Business cards are an established business practice that can also serve as an appointment card.
- It creates name recognition, personalizes you, and builds credibility.

The Price Also Rises

New dentists who graduate from dental school and prepare to establish their own practices are on average $81,688 in debt, according to the 1997 Survey of Dental School Seniors produced by the Chicago-based American Association of Dental Schools.

That figure, which includes students from private, public and private/state-related schools, represents a 7.8 percent increase from 1996. In contrast, graduating debt in 1980 was $18,500.

First-year tuition and fees have increased an average of 6 percent each year since the 1989-90 academic year. Average first-year tuition costs for the 1996-97 academic year were $12,771 for residents and $20,709 for students who live out-of-state.

"In order to finance their education, many students are borrowing $20,000 per year or more just to cover the costs of tuition and expenses," says David J. Fulton, DDS, a general dentist in Waukegan, Ill., and president of the Chicago Dental Society. "That makes it incredibly tough to make payments on loans while trying to open an office and establish a dental practice. Some new dentists just don't make it. I would hope that dental schools would step up their efforts to tackle the problem of student debt."

Following are other statistics from the 1997 Survey of Dental School Seniors:

- 13.3 reported no debt.
- 6.8 percent reported debt of $0 to $30,000.
- 10.6 percent reported debt of $30,000 to $50,000.
- 23.6 percent reported debt of $50,000 to $80,000.
- 17.5 percent reported debt of $80,000 to $100,000.
- 18.7 percent reported debt of $100,000 to $150,000.
- 9.5 percent reported debt of more than $150,000.

When the 13.3 percent of students who report no debt are omitted from statistics, the average graduating debt in 1997 rises to $94,182.

Link Between Chlamydia and TMJ is Found

The bacterium Chlamydia trachomatis, which is the leading cause of pelvic inflammatory disease and its resulting infertility, may also cause TMJ dysfunction. This condition affects 10 million Americans, the vast majority of whom are women, according to the National Institutes of Health.

A research team led by oral and maxillofacial surgeon Dr. Charles Henry is the first to identify the presence of Chlamydia

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trachomatis in human temporomandibular joint tissue, finding the bacterium significantly more prevalent in patients with TMJ dysfunction than in the general population. Henry, assistant professor at the Goldman School of Dental Medicine at Boston University, presented these results at the American Association of Oral and Maxillofacial Surgeons annual meeting in September.

Chlamydia is the most common sexually transmitted bacterium in the United States with an estimated 5 million cases per year. If recognized early, it can be effectively treated with a simple course of antibiotics.

"Our study indicates that chlamydia-induced arthritis may cause TMJ dysfunction and pain in many patients," Henry says.

Since chlamydia is frequently associated with sexually acquired reactive arthritis, its presence in the TMJ tissue suggests that TMJ dysfunction may be a previously unrecognized form of reactive arthritis.

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Current Views on Periodontal Therapy

David F. Levine, DDS

The more things change, the more they remain the same. Although periodontal therapy has undergone many changes in the past decade, the basics of periodontal therapy remain the same. Periodontal disease is primarily a bacterial infection. No matter what therapeutic modality is used, the main goal of therapy must be to reduce and then maintain the bacterial load at a level that the host (patient) can successfully defend. It may, in fact, be true that the most important factor in successful periodontal therapy is not the therapeutic modality, but the post-treatment maintenance program.

During the past decade, the research on and treatment of periodontal disease has been heavily weighted toward regeneration. Periodontists were quick to jump on the bandwagon of regeneration as the "new and improved" treatment of periodontitis. At previous national periodontal meetings, a significant number of the lectures were about using periodontal membranes to regenerate lost periodontal structures. In the evening, companies gave lavish parties to promote their regeneration products.

At the most recent national periodontal meeting, very few of the lectures were about regeneration using periodontal membranes. No lavish parties were given, and one company was even giving away their membranes. Their profits were down, and they were no longer planning on selling membranes for regeneration.

The point is not to denigrate the use of periodontal membranes. The use of membranes for regeneration does have its place in the armamentarium used to treat periodontal disease. However, time has shown that periodontal regeneration with membranes is not the panacea we had all hoped for. Time has again proven that the standard treatment modalities for periodontal therapy may still be the most effective. This is not to say that future therapeutic modalities will not bring about significant changes in the way we treat periodontal disease. However, at the present time, the standard therapeutic modalities are the only methods that have stood the test of time. Therefore, this issue of the *Journal of the California Dental Association* is dedicated to reviewing some of the standard therapeutic modalities used in the treatment of early to moderate periodontitis.

As a review of the current views about periodontal regeneration, Dr. William Becker discusses regeneration of lost periodontal structures using different materials. Dr. Becker has been involved with much of the research regarding periodontal regeneration. He reviews current literature regarding regeneration and discusses whether regeneration is really possible.

Dr. Greg Filippelli and I discuss osseous resective surgery as a surgical treatment option to treat early to moderate periodontitis. While the concept of pocket reduction is a fundamental objective of periodontal therapy, traditionally, there has been discussion and controversy associated with the different treatment methods utilized. Dr. Filippelli and I address this controversy, as well as discuss surgical pocket therapy directed toward pocket reduction through recontouring the underlying bone.

Dr. Perry Klokkevold discusses the new approaches to the diagnosis and treatment of periodontal disease in light of dentistry's improved understanding of the pathogenesis and appreciation for the influence of host factors.

Finally, Dr. Handelsman, Dr. Ravon, and I discuss surgical crown lengthening. Surgical crown lengthening is one of the most important periodontal surgical therapies, yet seems to be one of the least performed. Dr. Handelsman, Dr. Ravon and I discuss the indications for crown lengthening, as well as several of the benefits of completing it prior to final preparation of the permanent restoration.

It is hoped that these articles will help bring the reader up to date on some of the current views on these topics. It is important to be aware that some of these topics were included in this journal because it was believed that they would bring about controversy. It is hoped that through discussion of the controversies, we may all expand our knowledge of the treatment of periodontal disease. Whether you strongly agree or disagree with the position of the articles in this journal, if something is learned, my goal in putting this journal together has been reached.

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Periodontal Regeneration: Myth or Reality

William Becker, DDS, MSD

ABSTRACT One of the goals of periodontal therapy is regeneration. During the past 20 years, several materials and techniques have been developed and tested for enhancing periodontal regeneration. This paper evaluates flap debridement, allogenic and alloplastic grafting, and the use of nonresorbable and resorbable barrier membranes as regenerative techniques. One of the most predictable regenerative therapies is treatment of the three-walled intrabony defect. This defect can be repaired with 2 to 2.5 mm of bone fill and results in significant gains in clinical probing attachment and decreases in probing depths. There is a slightly greater improvement in periodontal measures with barrier membranes. Commercial preparations of allogenic bone and alloplastic fillers have a long, safe history of use and are primarily osteoconductive. They decrease probing depths and provide short-term gains in clinical attachment levels. Barrier membranes provide short-term evidence of improving Class II furcation invasions, however there is insufficient evidence that these improvements are sustained long-term. Class III furcations are not predictably treated by regenerative therapies. To date, there is an absence of clinical evidence that regenerative therapy increases the long-term life span of teeth.

AUTHOR

William Becker, DDS, MSD, is a clinical associate professor in periodontics at the University of Southern California School of Dentistry and a clinical professor in periodontics at the University of Texas at Houston. He also maintains a private practice, limited to periodontics, in Tucson, Ariz.

Regeneration is one of the primary goals of periodontal therapy. During the past 20 years, there has been an explosion of techniques and materials designed to increase the predictability of periodontal regeneration. Regeneration is defined as restoration of lost parts.[1] Periodontal regeneration requires the restitution of cementum with inserting fibers and bone. Repair implies healing by long junctional epithelium. Unfortunately, regeneration can only be ascertained by histologic evaluation, hence most of the procedures designed for regeneration probably result in repair. Studies using filler materials have demonstrated improved periodontal measures. To date, none of these studies has provided evidence that filler materials increase the life span of the treated teeth.

The purpose of this paper is to evaluate periodontal regenerative procedures and determine their patient benefits.

Defect Anatomy

Prichard described the classic intrabony defect as having three bony walls, with the root forming the fourth wall. The defect has definite limits and does not extend into the furcation.[2] Saari[3] and Tal[4] examined dry skulls and described the frequency and location of intrabony defects. These defects were frequently found distal to mandibular second molars. Alveolar bone in this location has thick cortical plates with varying quantities of cancellous bone. In the presence of inflammation, cancellous bone resorbs, leaving a bony crypt surrounded by a varying number of bony walls.

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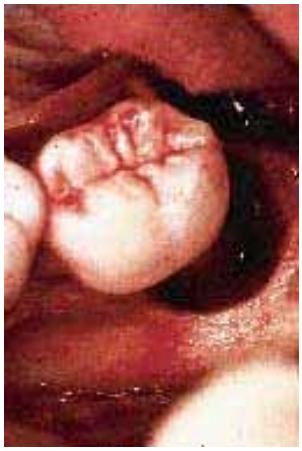

Figure 1a. Deep three wall intrabony defect distal to the mandibular second molar. The defect is contained, and furcation is not involved.

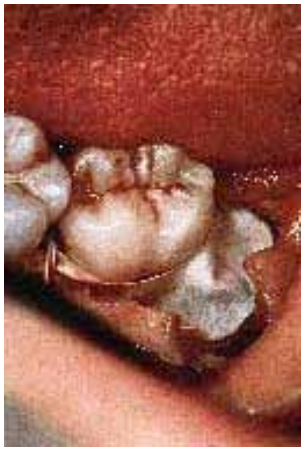

Figure 1b. An ePTFE barrier has been fitted to cover the defect.

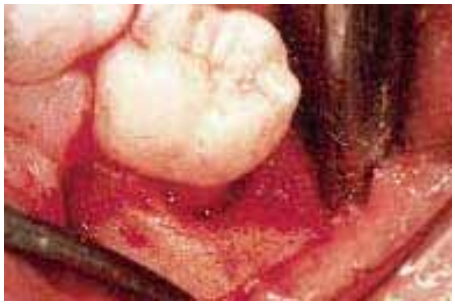

Figure 1c. The defect was re-entered 11 months after treatment to reduce a residual defect. Note defect bone fill.

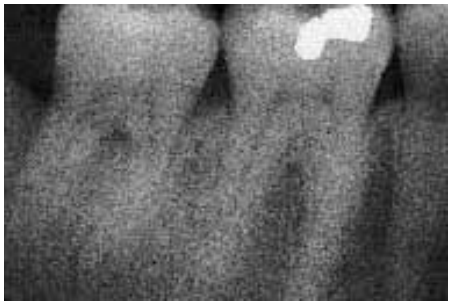

Figure 1d. The initial defect appears wide and extends close to the root apex.

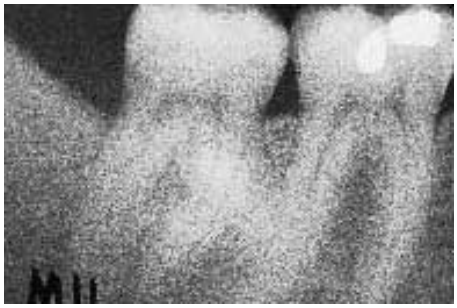

Figure 1e. Eleven-month follow-up X-ray. Note slight residual defect and extensive amount of bone fill.

Open Flap Debridement

Carranza[5] and Prichard2 are credited with presenting successful treatment of classic three-wall intrabony defects. These defects are surrounded by bone on three sides, with the root forming the fourth wall. Prichard followed successfully treated patients for more than 30 years. Examination of case reports demonstrated clinical evidence of bone fill. These defects appeared to be deep and were primarily located distal to mandibular second molars. Polson and Heijl6 reported an average gain of 2.5 mm of bone after treatment of deep two- and two-to-three-wall intrabony defects. Becker and colleagues[7,8] reported clinical findings after treatment of 36 consecutive three-wall defects. These defects were deeper than 5 mm and were wide. The average gain of bone fill was 2.5 mm, with significant decreases in clinical probing depth.

Treatment of these defects by flap debridement requires an understanding of anatomy, a thorough clinical examination, and identification of etiologic factors. The diagnosis and treatment depends upon evaluation of probing depths, clinical attachment levels, and good periapical radiographs. Defect depth can frequently be estimated from radiographs. In defects greater than 5 mm, the presence of subgingival calculus and positive tooth vitality are indications that treatment will result in a favorable outcome.

Surgical treatment consists of elevation of full-thickness mucoperiosteal flaps and thorough defect and root surface debridement. This is performed with a combination of rotary and ultrasonic instruments. The defect is probed using the lowest wall as reference. If the defect is greater than 5 mm, it is a good candidate for repair by flap debridement. Ochsenbein9 described the relationship of the defect to the surrounding bony walls. He reported that bony walls of the same height never surround three-walled intrabony defects. He recommended that the highest wall be reduced to the level of the adjacent walls. Adjusting the bony walls should allow for complete repair of the defect. The clot is then allowed to stabilize after which the flap margins are sutured with interrupted sutures. Postoperatively, patients can be placed on antimicrobial rinses, and the sutures are removed in a week. Oral hygiene is reinstituted and the area is evaluated by probing and radiographic examination after nine months of healing. These defects likely repair with a long junctional epithelium.

Allografts and Alloplasts

During the 1970s, the use of alloplastic filler and allogenic bone were introduced for treatment of periodontal defects.[10-17] Freeze-dried bone became a popular filling material, since early reports indicated this material was osteoinductive. The principle of osteoinduction means that bone morphogenetic proteins or other growth factors or proteins can affect undifferentiated mesenchymal cells to differentiate into cartilage and subsequently bone.

Urist, in a series of rodent studies, first described bone formation in ectopic sites (muscle tissue) by induction.[18-20] Urist isolated a complex series of inductive proteins known as bone morphogenetic proteins.[20, 21] The indications, expectations, and contraindications of when and where to use allogenic bone became confusing. Numerous clinical studies have demonstrated that placement of allografts into periodontal defects will result in decreased probing depths, bone fill, and gains in clinical attachment levels.[13,14,22]

Other investigators have presented histologic evidence of periodontal regeneration following implantation of demineralized bone matrix.[15,23] When interproximal defects were treated with either barrier membranes alone or with allogenic bone, the results were similar.[24] Unfortunately, clinical studies do not demonstrate evidence of bone induction by the implanted material. Recently bone induction with commercially available allogenic bone has been questioned.[25-27] There is sufficient scientific evidence that commercially available bone matrix has varying amounts of bone-inductive activity. The capacity of the bone implants to initiate bone induction diminishes with age and varies from batch to batch. The quantity of bone matrix proteins or other growth factors or proteins necessary to induce clinically relevant amounts of bone is unknown. Commercially available bone allografts can be considered osteocondutive. When the allogenic bone particles are in contact with host bone over time, new bone will surround the bone matrix particles. Allogenic bone away from native bone will remain unresorbed for long periods. There is insufficient evidence to indicate that graft particles will ever be totally replaced by host bone. There is also insufficient evidence to indicate that allografts will be resorbed by osteoclasts, hence replacement by substitution is highly variable. Healing of defects treated with allogenic bone implants likely occurs by repair. This may be sufficient to prolong the life span of the tooth.

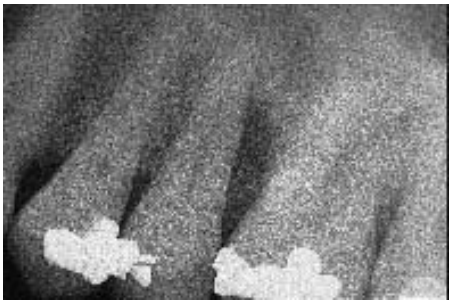

Figure 2a. Radiograph demonstrates deep, intrabony defect distal to the maillary right first bicuspid.

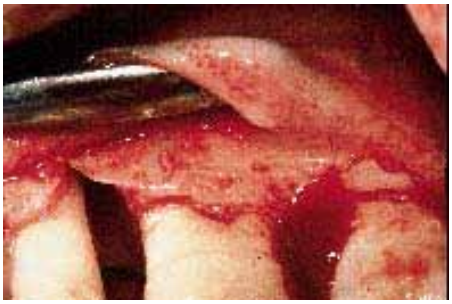

Figure 2b. Buccal view, showing inconsistent bony margin.

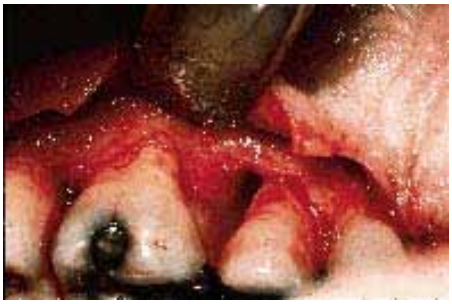

Figure 2c. Palatal view demonstrates contained, three-walled intrabony defect.

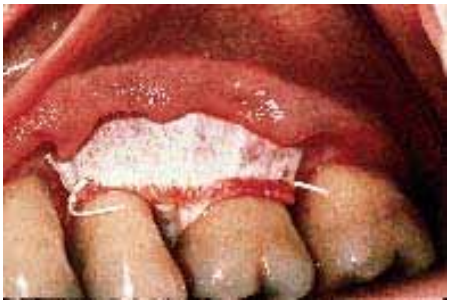

Figure 2d. An ePTFE barrier membrane has been used to isolate the defect.

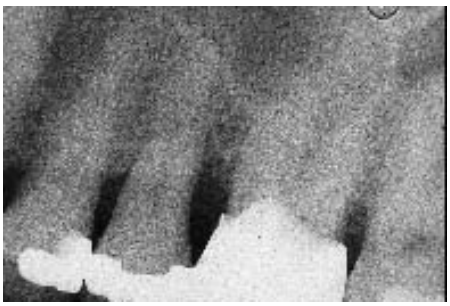

Figure 2e. Three-year follow-up radiograph demonstrating bone fill of original defect. Compare with Figure 2a.

Investigators created furcation defects in baboons.[28] They implanted bone morphogenetic proteins extracted from bovine demineralized bone utilizing the demineralized matrix as the carrier for test sites. Collagen matrix alone was used for control defects.

Histologic evaluation of treated defects indicated significant periodontal regeneration within the test furcations; however, large quantities of unresorbed demineralized matrix carrier was present within the surrounding connective tissue.

Alloplastic materials are synthetic bone substitutes. These materials are biocompatible, osteoconductive, and either resorbable or nonresorbable. They are of questionable value as regenerative materials. They can effectively be used for ridge augmentation, providing that dental implants do not significantly contact these fillers. Treatment of periodontal defects with filler materials results in decreased probing depths, gains in clinical attachment levels, and radiographic evidence of "bone like" material within the defects. The defects heal by repair. Long-term follow-up studies of teeth treated with these materials are not available, and the patient benefits are questionable.

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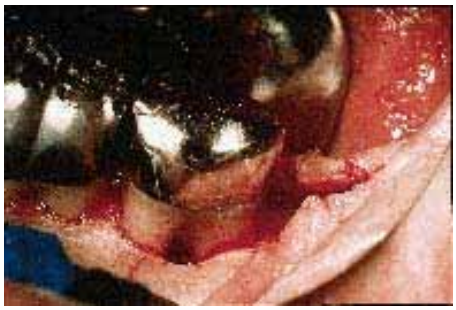

Figure 3a. A Class II furcation with a narrow buccal furcation defect after debridement

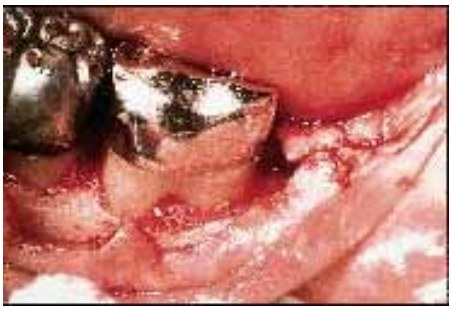

Figure 3b. Defect filled with demineralized freeze-dried bone.

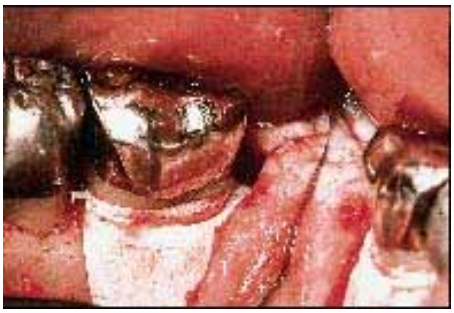

Figure 3c. A barrier membrane was fitted over bone-filled defect.

Guided Tissue Regeneration

Scandinavian investigators introduced the principle of guided tissue regeneration.[29-35] The concept was based on isolating periodontal defects with barrier membranes.

The purpose of the barriers was to exclude epithelial down-growth and to allow periodontal ligament cells to repopulate the previously diseased root. The first commercial barrier membranes were made of expanded polytetrafluoroethylene (ePTFE) (WL Gore and Associates, Flagstaff, Ariz.) and were cell-occlusive. There was an open microstructure collar that was fitted around the tooth at or near the cementoenamel junction. The biologic rationale for using these barriers was based on scientific and clinical studies. These barriers have been used to treat intrabony and furcation defects.[32,33,35-44] Results after treatment of multiwalled defects with debridement and barrier membranes demonstrated improved probing depths and gains in clinical attachment levels with varying amounts of reported bone fill (**Figures 1 and 2**). Several studies provided evidence of sustained gains in clinical measures when compared with the short-term reports.

Barrier membrane treatment of furcation defects is technique-sensitive. There is minimal evidence of complete furcation closure with or without allogenic bone alone or with barrier membranes.[40,42] There are several reports in which barrier membranes have been used with allogenic bone.[45-47] The primary reason for adding the bone implants was to support the barrier membranes. Barrier membranes with allografts have primarily been used to treat furcation defects. Studies that compared debridement alone with barriers plus grafts generally demonstrated similar results.[48,49]

Figure 3 demonstrate a furcation defect that was treated with a demineralized freeze-dried bone and an ePTFE barrier. Probing depth had decreased by the nine-month evaluation. The patient was maintained for six years with progressive loss of the initial soft tissue gains in clinical probing attachment and a return to preoperative probing depth. Failure to predictably close Class II furcations and the tendency for initial probing depth

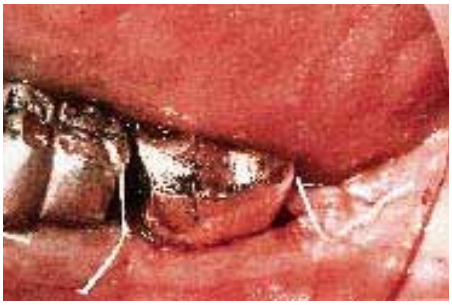

Figure 3d. Flaps sutured in an attempt to completely cover the barrier membrane.

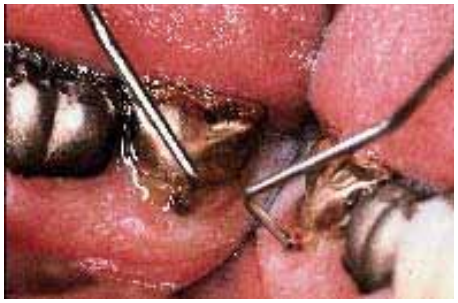

Figure 3e. Patient was maintained at four-month intervals for six years. Defect probed during a maintenance visit. Note proble penetration into furcation. Probing depth reduction originally recorded postoperatively has been lost. The defect probes to the original depth.

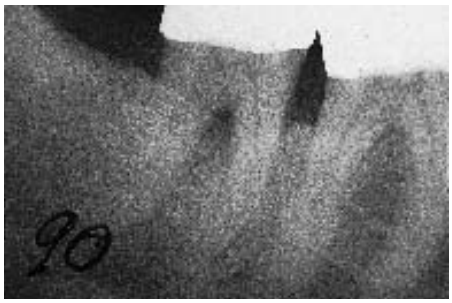

Figure 3f. Preoperative radiograph taken in 1990

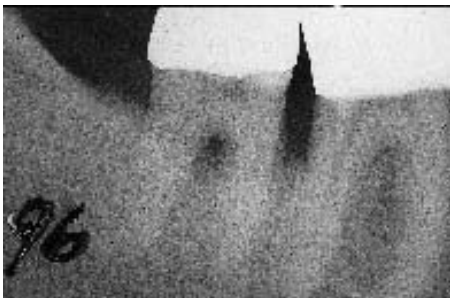

Figure 3g. Radiograph taken at follow-up visit in 1996. Compare with tradiographi taken in 1990. There was no apparent change in the furcation status.

decreases to regress to pretreatment depths has brought into question the long-term patient benefits of treating furcation defects with barrier membranes alone or in combination with allografts. Moderately involved Class II defects can be effectively treated with open flap debridement, however long-term patient outcomes are unknown. There are no predictable methods for treating Class III furcations with regenerative procedures.

Bioabsorbable Barriers

Nonresorbable barriers required a second minor surgery for removal. These membranes frequently became exposed and plaque-infected. When this occurred, there was less improvement in clinical measures when compared with sites where the membranes remained unexposed.[50-52] As a consequence of these problems, a new generation of barrier membranes was developed. These barriers resorb and are manufactured from either polyglycolic and polylactic acids, collagen, or various combinations of these. Resorbable barriers have been used to treat intrabony as well as furcation defects. Results from studies comparing nonresorbable to resorbable membranes demonstrate comparable results.[53] Intrabony defects were treated with resorbable barriers and compared with flap debridement controls.54 Findings indicated improved clinical measures for probing depth reduction and clinical attachment level gains. These improvements were not significantly enhanced with guided tissue regeneration therapy.

Conclusion

Technological and surgical techniques have been implemented to enhance periodontal regeneration. Results from these advances were met with enthusiasm because they provided the possibility for improving results from periodontal regenerative procedures. When these procedures are critically evaluated, they appear to have slightly better clinical outcomes than flap debridement procedures. These slight improvements may not provide cost or patient benefits in terms of improved periodontal health and may not increase the health or longevity of the treated teeth.

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A Review of Osseous Resective Surgery

DAVID F. LEVINE, DDS, AND GREG FILIPPELLI, DDS

ABSTRACT The treatment of periodontal diseases associated with attachment loss has involved a variety of approaches. While the goal of periodontal surgical treatments is to access the root surfaces for proper debridement, the decision to remove or reshape the supporting bone has been controversial. This paper will address the controversy as well as discuss surgical pocket therapy directed toward pocket reduction through recontouring the underlying bone.

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Over the years, the treatment of periodontal diseases associated with attachment loss has involved numerous surgical and nonsurgical approaches. Although the concept of pocket reduction is a fundamental objective of periodontal therapy, discussion and controversy have been associated with the different treatment methods used. Several surgical treatment modalities have been proposed to treat the soft tissue lesions associated with periodontitis as well as to gain access to the root and supporting bone. These include the apically positioned flap with and without osseous resection, modified Widman flap surgery, open flap curettage, and several other repositioned-flap procedures. While most of these periodontal surgical treatments have as their main goal to access the root surfaces for proper debridement, it is the decision to remove or reshape the supporting bone that has produced the most controversy. This paper will address this controversy, as well as discuss surgical pocket therapy directed toward pocket reduction through recontouring the underlying bone.

Schluger is often credited with first describing osseous resective therapy. In 1949,[1] he attempted to describe and identify the rationale for and technique of osseous resective surgery for pocket elimination. He stated that total pocket elimination could be maintained only with the removal of the bony component of the pocket. He further advocated the need to reshape the bone to a physiologic form resembling the pattern of horizontal

atrophy. Otherwise, the gingiva would not adapt adequately; and pockets, especially interproximally, would re-establish. In 1935, Carranza[2] advocated a mucoperiosteal flap approach and listed specific indications where bone should be surgically remodeled to stimulate the rebuilding of healthy support around the teeth. Since then, several people have talked about and defined osseous resective therapy. The World Workshop in Periodontics defines osseous surgery as "that aspect of periodontal surgery which deals with the modification of the bony support of the teeth."[3] Sims and Carranza define osseous surgery as the procedure by which changes in the alveolar bone can be accomplished to rid it of deformities induced by the periodontal disease process or other related factors, such as exostosis and tooth supraeruption.[4] Friedman described osseous surgery as surgical removal of the gingiva and reshaping of the bone to eliminate the pocket and correct unphysiologic architecture.[5] What all these definitions have in common is that the goal of osseous surgery is to produce osseous contours that are consistent with the shape and form of the healthy gingival tissues. By creating a bony architecture that mimics the final shape of the gingiva, it is thought that pocket reduction cannot only be obtained, but also maintained.

Gingival tissue is elastic, that is, it tends to return to its original architectural contours. Even when deep pockets exist, the gingiva will retain a scalloped form that follows the shape of the cementoenamel junction. This form is independent of the underlying contours of the osseous crest. When the gingiva has its normal architecture and the underlying bone has a similar architecture, there is a shallow probing depth. When there is a discrepancy between the gingival tissue and the underlying bone, the difference is expressed in pocket depth. If the gingival tissues are contoured to the form of the diseased osseous crest, the irregularities created in the gingiva would not be maintained. In time, the soft tissue will round out and revert to its original scalloped pattern.

This soft tissue proliferation results in pocket depth. Residual increased pocket depths are more likely to break down, need constant and meticulous treatment in the office and at home, and present a potential nidus for reinfection. To be truly successful in reducing pocket depths, the reshaping of bone must be done with curves and slopes that mimic the contours of the healthy gingiva. The tendency of gingiva to assume a pre-existing form dictates the architecture of the bone that must be created to achieve a stable result. Therefore, the eradication of the periodontal pocket is dependent upon the correction of the underlying bony deformity. Surgical elimination of the pocket with resultant minimal probing depths allows the patient access for proper plaque control and facilitates maintenance by the therapist. This is the basis of osseous resective surgery.

Procedures used to correct osseous defects have been classified into two groups. In 1955, Friedman defined these two procedures as osteoplasty and osteoectomy.[5] Friedman defined osteoplasty as a plastic procedure in which the periodontal pocket is eliminated and the bone reshaped to achieve physiologic contour of the bone and the gingiva overlying it. In this operation, the bone that is reshaped is not part of the attachment apparatus, thus no bony support of the tooth or teeth is lost. Friedman defines osteoectomy (also referred to as ostectomy), as an operative procedure in which bone that is part of the attachment apparatus, is removed to eliminate a periodontal pocket and establish gingival contours that will be maintained. Ostectomy requires the loss of some bony support of the tooth or teeth, and the amount involved will be an important criterion for its use.

The terms positive architecture and negative architecture are also widely used when discussing osseous surgery. These terms describe the position of the interdental bone in relation to the radicular bone. Positive architecture refers to a situation in which the osseous crest follows the shape of the cementoenamel junctions, that is, the interdental bone is more coronal than the radicular bone. Negative architecture is used to describe a situation in which the osseous crest does not follow the shape of the cementoenamel junctions, that is, the interdental bone is more apical than the radicular bone. The discrepancies between the shape and position of the osseous crest and the gingival margin result in pocket depths.

Indications for osseous surgery as described by Carranza and Carranza Jr.[6] are to recontour bone that forms part of the outer wall of the pocket, to prevent recurrence of the pocket, and to reshape the alveolar crest, establishing a normal fiber arrangement. Indications for osteoplasty according to the World Workshop in Periodontics[3] are buccal or lingual bony ledges, tori, etc.; intrabony defects associated with tilted molars; shallow buccal or lingual intrabony defects; flat interproximal areas; the elimination of deep interproximal defects to achieve physiological contour; incipient furcation involvements; and for improvement of alveolar contours for flap adaptation. Ostectomy has been indicated for the elimination of interdental craters, intrabony pockets not amenable to

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reattachment procedures, horizontal alveolar bone loss with irregular marginal bone height, and moderate and advanced furcation involvement.[3]

The most common indication for osseous resective surgery is to treat the shallow two-wall osseous crater. Osseous craters are concavities in the crest of the interdental bone that are confined within the facial and lingual walls of the alveolus. Practically all craters have a slope from buccal and lingual walls to the base. These slopes represent more than two-thirds of the crater.

The site of the initial periodontal lesion is usually the interproximal area, with the two-wall osseous crater as the most common type of osseous defect. The osseous crater has been found to make up about one-third of all defects and about two-thirds of all mandibular osseous defects.[7,8] They are twice as common in posterior segments as in anterior.[7,8] The greater frequency in the posterior areas is probably due to the thickened alveolar housing in the posterior, as well as the wider interproximal contact area between adjacent teeth. In the anterior region, the slender cone of the interproximal bone is gradually blunted by progressive bone resorption. Crater formation in the anterior occurs only after extensive bone loss. The bone between the posterior teeth presents a flat occlusal surface in health. Bone destruction rapidly creates an intrabony crater. To a great extent, the thickness of bone and the pattern of inflammation determine the pattern of bone loss on the lateral and medial surfaces of the teeth. Thin plates of bone resorb in an apical direction without crater formation. Thicker ledges of bone undergo incomplete resorption, resulting in the formation of a crater or well adjacent to the tooth.[7-9] Reasons for the high frequency of interdental craters overall include the facts that the interdental areas are more difficult to clean, and only a small percentage of people floss regularly. Other possible explanations include the lack of keratinization of the gingival col area and vascular patterns from the gingiva to the center of the crest that may provide a pathway for inflammation.[7-9]

Contraindications for osseous resective surgery include deep osseous craters, three-wall osseous defects, moderate to deep circumferential defects, and bony defects situated on the buccal aspect of terminal mandibular molars associated with the external oblique ridge. It is important to remember that osseous surgery is best-suited to treating early and moderate periodontal defects. Advanced periodontal lesions or isolated deep craters may require some bone contouring, but not for the express purpose of eliminating the defect. Selective extraction, grafting procedures, and/or root amputations are often necessary to manage such areas. Three-wall bony defects should be managed by regenerative techniques since removal of supporting bone would often jeopardize the future of the affected tooth, as well as the adjacent teeth. Other precautions must also be taken into consideration before one decides to use osseous surgery. If too much supporting bone must be sacrificed on sound teeth to retain a neighboring affected tooth, it may be better to sacrifice the involved tooth or leave a residual bony defect. If a furcation will be exposed because of an extensive sacrifice of bone, it may be better to accept a deep gingival crevice. It should also be noted that osseous surgery should not be done in areas that have pocket depths of less than 5 mm. Shallow pockets treated with osseous surgery result in a net loss of attachment.[10-14]

Other contraindications for surgery are inadequate plaque control by the patient, noncompliance with supportive periodontal therapy, and certain medical and anatomic conditions.

Although the concept of osseous surgery was introduced into the United States in the late 1940s, a critical description of surgical guidelines, tenets, and limitations was essentially unavailable for decades. The lack of standardized guidelines and the variability in surgical technique among clinicians made it difficult to scientifically compare the effectiveness of the surgical treatment modalities. Clinicians differed in opinion concerning the initial incisions, amount and location of bone removal, degree of flap elevation, and methods of suturing. The variability in postoperative maintenance regimes further complicated the transition of these procedures from research to clinical practice. This disparity fueled a healthy exchange of ideas that has improved the modern design of clinical research and helped define the periodontal surgical procedures now provided for patients on a daily basis. However, this disparity also made for great difficulty in scientifically determining the superiority of one procedure over another. Thus was born the controversy that is still debated.

Part of the controversy surrounding osseous surgery has involved the extent of bone removal necessary for the creation of a positive osseous architecture. Ochsenbein said, "The primary objective of osseous surgery is to remove the minimal amount of bone that will meet the needs of an adequate architectural form."[15] A study by Selipsky[16] found that during osseous surgery, the average height of supporting bone removed per tooth was only 0.6 mm. Loss of interproximal bone support was negligible, except where

severe angular defects were present. He also found that the majority of ostectomy performed was midbuccal, midlingual, or palatal adjacent to interproximal defects. Even on these surfaces, only about 1 mm of supporting bone was removed. Selipsky also noted that removal of buccal or lingual bone seems to be less important in terms of tooth support than the removal of interproximal bone. This is because roots are generally irregular in shape, and the buccal or lingual surfaces have a smaller surface area than the flattened root form extending buccolingually. The result is that interproximal bone gives more support in terms of surface area than does buccal or lingual bone, especially in the posterior.[16]

It was also once believed that removal of supporting bone might increase tooth mobility. However there is no scientific evidence to support this claim. Selipsky found that teeth initially loosened postsurgically, but returned to pre-operative mobility levels within one year. This response occurred without any form of splinting. Only time and a healthy environment were needed to obtain the reduction in mobility to presurgical levels.[16] Smith and colleagues, in a study comparing osseous resection with flap curettage, also confirmed these results. They found no net change in tooth mobility at six months postsurgery.[14]

The most complete treatise on osseous surgery to date is Ochsenbein's "Primer for Osseous Surgery."[17] In this work, he describes in great detail the classification of molar osseous craters and the variations of molar root morphology that affect surgical decision-making. These relationships are critical to understanding the disease process, and they aid in proper surgical management of the posterior regions. The dimensions of the osseous crater and the size of the molar root

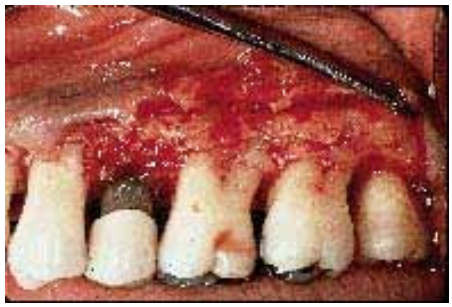


Figure 1a. Preoperative view of the maxillary left buccal.

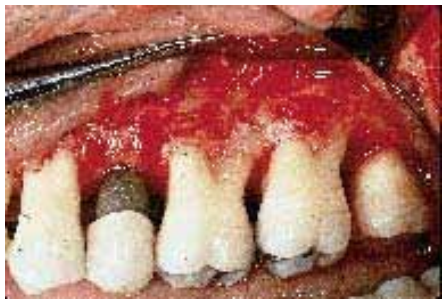


Figure 1b. View of the maxillary left buccal after osseous recontouring.

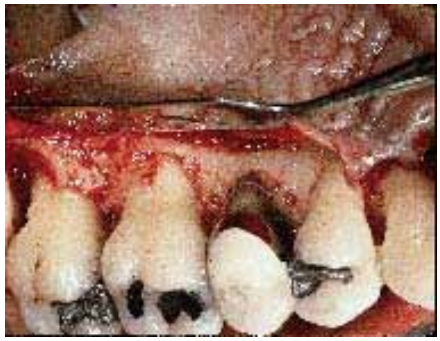


Figure 1c. Preoperative view of the maxillary left palatal.

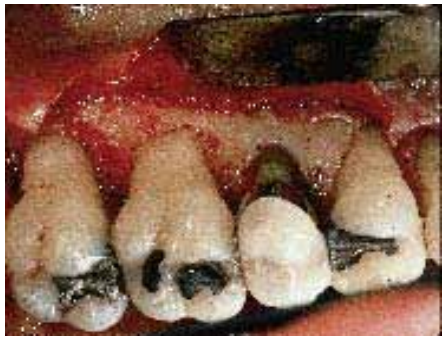


Figure 1d. View of the maxillary left palatal after osseous recontouring.

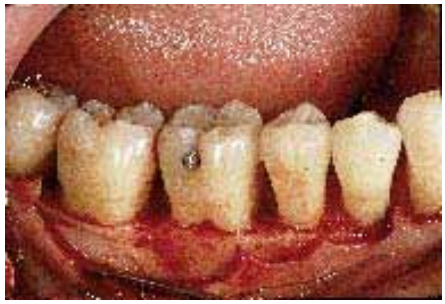


Figure 2a. Preoperative view of the mandibular right buccal.

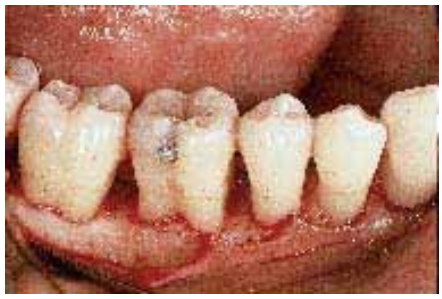


Figure 2b. View of the mandibular right buccal after osseous recontouring.

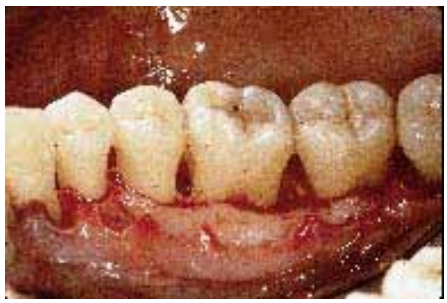


Figure 2c. Preoperative view of the mandibular right lingual.

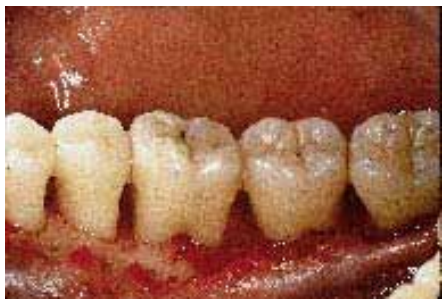


Figure 2d. View of the mandibular right lingual after osseous recontouring.

trunk give an indication as to how much bone is present coronal to the level of the furcation. This information dictates the limits of osteoplasty and ostectomy performed in every periodontal osseous surgery. A maxillary first molar with a short root trunk, for example, may have only 1 mm of radicular bone coronal to the buccal furcation. Anatomic considerations such as this call for judicious management of the osseous crest when performing recontouring procedures so as not to invade the furcation unnecessarily.

The palatal and lingual approach to osseous surgery is advocated in the posterior regions due to the location of the buccal furcations, the level of the osseous crest, inclination of the mandibular molars, and the position of infrabony defects.[15] This approach conserves alveolar bone on the buccal aspect, thereby sparing the furcations. While this technique is more time-consuming than others, it helps to prevent the overzealous removal of bone on the buccal aspect and the creation of a negative osseous architecture (**Figures 1 and 2**).

Becker and colleagues11 compared scaling, osseous surgery, and modified Widman surgery in 16 patients over one year. Each technique was carried out by a periodontist who is a proponent of that technique and is experienced in its application. All patients were diagnosed as having moderate to advanced adult periodontitis. Prior to surgery, each patient had two one-hour sessions of oral hygiene instructions with scaling and root planing. All patients had each of the three procedures performed in different, randomly selected quadrants. After one year, the results showed that scaling, osseous surgery, and the modified Widman procedure were effective in treating moderate to advanced adult periodontitis. However, the greatest increase in 1-3 mm probing depths was found in the osseous surgery group. Sites treated by osseous surgery also had the fewest sites in the 4-6 mm and 7 mm or greater range at the one-year evaluation period. They also found an increase in clinical attachment levels for the 4-6 mm pockets from postsurgery to one year. This increase occurred for all three of the treatment modalities. Kerry and colleagues reported on the results of this study after five years. The five-year results were very similar with regard to probing depths and clinical attachment levels.[12]

Kaldahl and colleagues[10] followed 82 patients over two years. Quadrants were randomly assigned to coronal scaling, root planing, Widman surgery, or osseous resection surgery. Initial therapy consisted of coronal scaling in the quadrant that received coronal scaling as the treatment modality. The quadrants designated for root planing or surgery received root planing. At the re-evaluation, additional root planing was completed as needed for the quadrant receiving root planing as the treatment. The areas assigned for surgery had surgery completed as planned. In the quadrants receiving osseous surgery, teeth were extracted and roots amputated to facilitate pocket elimination. Surgery was only performed where pocket depths of 5 mm or greater were present after initial therapy. Results of this study show that probing depth reduction was greater in deeper pockets and greatest for osseous surgery. In 5-6 mm sites, Widman and root planing resulted in a slightly greater gain in clinical attachment levels than osseous surgery, but these gains were similar in pockets greater than 6 mm.

Kahdahl and colleagues18 also reported on the seven-year follow-up of these patients. Of the original 82 patients, 51 were available for follow-up. As was the case with the earlier study, the quadrants treated with flap and osseous resection resulted in greater probing depth reduction in sites that originally demonstrated probing depths greater than 5 mm. All three modes of therapy produced significant and equal gains in clinical attachment levels in sites greater than or equal to 7 mm. However, shallower sites treated by root planing did show slightly better gains in clinical attachment levels than modified Widman and osseous resection.

In a companion study, Kaldahl and colleagues[19] evaluated the incidence of breakdown sites in the above group of patients. If a site lost 3 mm or more of clinical attachment level from three weeks postsurgically or post scaling and root planing, it was classified as a breakdown site. Over the course of the study, the incidence of sites breaking down was greater for deeper sites than for shallow sites. This was true for each of the treatment modalities. Sites treated by osseous surgery, however, did show the lowest incidence of breakdown when compared with the other treatments. This was true for pocket depths in all ranges, i.e., 1-4 mm, 5-6 mm, and greater than or equal to 7 mm.

In 1985, Townsend-Olsen and colleagues[13] published a follow-up study initiated by Smith and colleagues. They re-evaluated 12 patients with moderate periodontitis who underwent open flap curettage and osseous resection surgery in bilateral quadrants. Both the open flap curettage and osseous resection surgeries were completed by apically positioning the flaps at the osseous crest. Plaque control, root planing, and occlusal adjustment were performed prior to the surgery in all quadrants; and the patients were followed at six-month intervals for the first two years. After two years, the

patients were seen every three months until the completion of the study three years later. Probing depths, clinical attachment levels, and sounding to bone were used to measure differences between procedures at the various time points. Plaque index, gingival index, mobility, width of keratinized tissue, and level of the gingival margin were also measured. Results after five years showed that both procedures reduced probing depths initially. However, the osseous surgery quadrants maintained these decreased probing depths to a significantly greater extent than the flap curettage quadrants, especially in the interproximal areas. In addition, the quadrants treated with osseous surgery had significantly fewer sites that bled on probing. In areas that initially demonstrated attachment loss, there was no change from baseline in attachment levels at five years with either procedure.

Recently, Tuan and colleagues20 reported a study comparing the clinical and microbiological study of apically positioned flaps with and without osseous surgery. They evaluated 14 adult periodontitis patients with interproximal craters. In seven patients, osteoplasty and ostectomy were performed from the lingual/palatal aspect to eliminate interproximal osseous defects, and to mimic the original alveolar bony transition to the adjacent teeth. In another seven patients, the surgical flap was adapted to pre-existing osseous defects. Patients were instructed in oral hygiene and were seen on a three-month supportive periodontal therapy schedule. Three periodontal sites having initial depths from 4 to 8 mm were examined at baseline and one, three, and six months. Clinical measurements and subgingival microbiota were evaluated at each examination period. Results showed that pocket depths were reduced more in the group that received osseous recontouring. This was true for the one-, three- and six-month examinations. They also found that in those patients who received osseous recontouring, the levels of subgingival periodontal pathogens were also significantly lower at all examination periods.

Periodontal osseous surgery has been shown to effectively reduce mean probing depth, decrease clinical signs of inflammation, and contribute to the overall stabilization of the clinical attachment. According to the American Academy of Periodontics parameters of care,[21] "the goals of periodontal therapy are: to alter or eliminate the microbial etiology and contributing risk factors for periodontitis, thereby arresting the progression of the disease and preserving the dentition in a state of health, comfort, and function with appropriate esthetics, and to prevent the recurrence of periodontitis." Osseous surgery is effective in achieving many of these therapeutic goals. When properly performed, it achieves a physiologic architecture of the marginal alveolar bone that is conducive to gingival flap adaptation and minimal probing depth. The advantages of this surgical modality include the following:

- Produces immediate and predictable reduction in probing depth;
- Improves access for daily oral hygiene and periodic maintenance;
- Preserves gingival width via apically positioned flaps;
- Allows complete removal of granulomatous tissue;
- Allows visualization and access for definitive debridement of radicular surfaces;
- Permits recontouring of bone anomalies (e.g., tori, ledges);
- Allows appropriate access for root resection and hemisection when necessary;
- Permits access for correction of radicular anomalies (e.g., cervical enamel projections, enamel pearls, pin perforations, etc.);
- Facilitates recontouring of restorative overhangs; and
- Permits restorative crown lengthening where indicated.

The advent of osseous surgery for the treatment of early to moderate periodontal disease was spurred by the shortcomings of soft tissue procedures such as gingivectomy and gingivoplasty. These procedures were effective in the short-term reduction of pocket depth and improved access for root debridement, but did so at the expense of the gingival width. Apical positioning of the mucoperiosteal flap during surgery preserves keratinized tissue while achieving a minimal posttreatment probing depth. Bony projections and aberrations such as tori and ledges are often encountered during flap surgery and require osteoplasty to achieve proper adaptation of the gingival flap. Osseous surgery allows for the appropriate access to recontour these anomalies, and achieve minimal sulcus depth wherever possible (**Figure 3**). This improved access is also beneficial during root resection procedures to access complete removal of the root and allow the creation of a cleansable convex surface.

The difficulty in removing all causative agents from the radicular surface of a tooth has been extensively reported in the periodontal literature.[22-26] Although surgical access does not guarantee definitive instrumentation of the root surface, its effectiveness is greatly enhanced by visualization of the field during debridement. It is not unusual during osseous surgery to encounter a deep fluting or narrow furcation that is laden with calcareous deposits (**Figure**

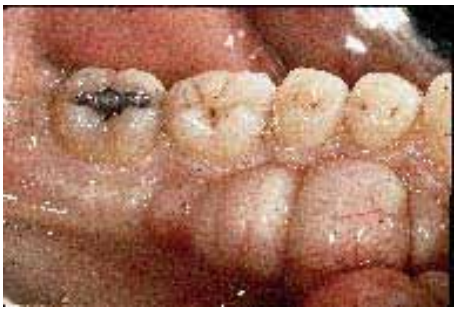


Figure 3a. Preoperative view of the mandibular left lingual showing large lingual tori.

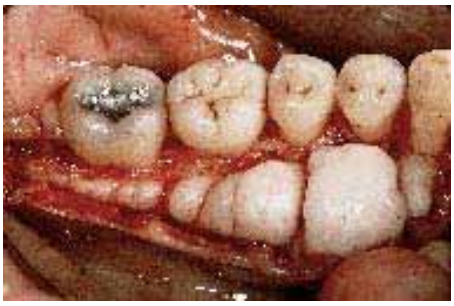


Figure 3b. View of mandibular left lingual with lingual flap elevated, showing large lingual tori.

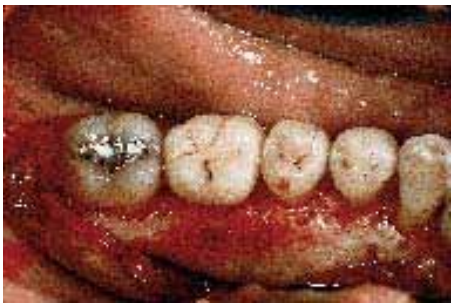
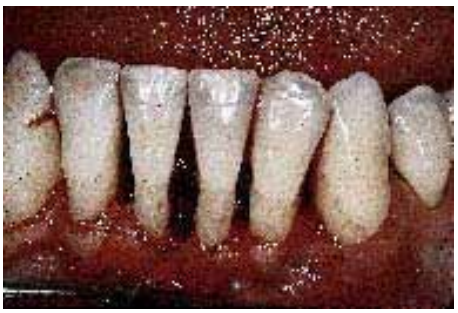
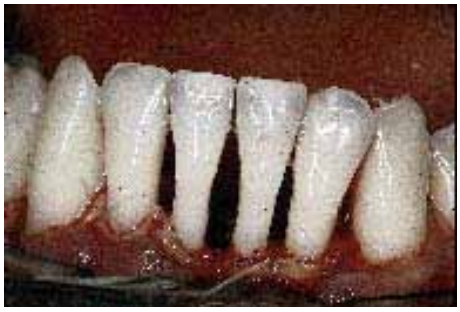


Figure 3c. View of the mandibular left lingual showing osseous recontouring and removal of tori.





Figures 4a and 4b. The main objective of any periodontal surgical procedure is to the gain access to the root surfaces for more effective removal of calculus and the associated subgingival microbiota.

4). Osseous surgery permits access to these root features and allows removal of deposits and anomalies such as enamel projections or enamel pearls that encroach upon the furcation and may exacerbate the progression of attachment loss.

The application of osseous surgery to restorative dentistry is also very significant. Today's esthetic procedures demand an even greater attention to the soft tissue margin adjacent to a restoration than ever before. Violation of the biologic width often detracts from the esthetics of a restoration and poses a problem in capturing the impression of the tooth preparation. Osseous surgery achieves a greater axial crown length that aids in retention and creates an appropriate sulcus depth to conceal the restorative margin. In the case of early to moderate periodontitis, the immediate and predictable reduction in probing depths following osseous surgery is reassuring to the restorative dentist, especially when it concerns strategic abutments for a fixed partial denture.

Perhaps the most notable advantage of osseous surgery is the improved access it provides postsurgically for daily oral hygiene and periodic maintenance. Shallower pockets are easier for both the patient and the therapist to maintain. Minimal pocket depths (less than 3 mm) have been shown to be one-fourth as likely as pocket depths greater than 3 mm to show subsequent attachment loss.[27] This improved accessibility to the root surface to remove etiologic factors and reduction in pocket depth are consistent with the goal of preventing the recurrence of periodontitis after surgical intervention.

Although endpoints to therapy may differ slightly among the periodontist, restorative dentist, and patient, the advantages of osseous surgery are significant to all. The treatment of periodontal disease often requires the application of several procedures to obtain the intended outcome. Determining factors for success reside in proper clinical diagnosis, appropriate choice and execution of therapies, and the realistic assessment of outcome. It is important to remember that osseous surgery is but one of the several surgical treatment modalities that may be used to treat periodontal disease. Other surgical modalities may be effective when executed properly in the appropriate situation. The European Workshop on Periodontology stated that periodontal therapy using different surgical modalities has been shown to be equally effective in reducing pocket probing depth, controlling the progression of chronic adult periodontitis, and achieving improved levels of probing attachment.[28] It may be that the most important factor in successful periodontal therapy is not the therapeutic modality, but the posttreatment maintenance program. However, osseous surgery does remain a viable, time-tested treatment modality that offers a predictable reduction in probing depths, a decrease in gingival inflammation, and a soft tissue form that is conducive to long-term maintenance.

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Periodontal Medicine: Assessment of Risk Factors for Disease

Perry R. Klokkevold, DDS, MS

ABSTRACT The approach to the diagnosis and treatment of periodontal disease is changing. The disease has not changed, but dentistry's understanding of the pathogenesis and appreciation for the influence of host factors has improved. As a result, the approach to the management of the disease is evolving. This paper reviews some of the host risk factors that have been linked to an increased severity of periodontal disease and briefly highlights some of the evidence that has led to the current belief that periodontal disease may be a risk factor for adverse systemic health conditions.

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During the past several decades, a great deal has been learned about the pathogenesis of periodontal disease and the bacterial pathogens that are responsible. The improved understanding of the pathogenesis has led to the development of many new diagnostic tools and therapies. Both diagnostic and therapeutic advances have been primarily directed at local factors (i.e., bacterial plaque). Specifically, they have focused on identifying the bacterial pathogens and decreasing or blocking the effects of their tissue-destructive enzymes. Although many new diagnostic tests have been developed, such as DNA probes for detecting known periodontal pathogens, they have not had the impact on diagnosis and therapy one might have hoped to achieve with such advances. More importantly, these advances in knowledge, understanding, and technology have not changed the routine periodontal examination nor the practitioner's ability to identify patients at increased risk of future disease.

Periodontics, like many other specialized areas of dentistry, is undergoing yet another change. The current era of change in periodontics is focusing on host factors. This evolution is being stimulated by new evidence that suggests a link between systemic factors and the severity of periodontal disease. At the World Workshop in Periodontics in 1996, a committee appointed by the American Academy of Periodontology to assess the current knowledge and summarize the status of periodontics concluded that

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assessment of risk was an integral part of diagnosing and treating periodontal disease. Page and Beck reported that evidence to support risk assessment in clinical decisions is substantial.[1] Evidence emerging from clinical research has shown that patients with systemic diseases (e.g., diabetes) as well as patients with other systemic factors (e.g., smoking) have an increased risk and severity of periodontal disease. As a result, these systemic "host" factors are now being recognized as significant contributors to development and progression of periodontal disease.

In addition to findings that support an increased risk relationship between systemic disease and periodontitis, evidence is also emerging to support the idea that periodontitis may have an adverse effect on systemic health.[2-8] Specifically, periodontal disease has been associated with an increased risk of coronary heart disease; poor glycemic control in diabetics; respiratory disease; and preterm, low-birth-weight babies. As a result of these new findings, much attention is being given to the interrelationship between periodontal disease and systemic health. Periodontal medicine is a term that has been used to describe this new era in periodontics. This paper reviews some of the host risk factors that have been linked to an increased severity of periodontal disease and briefly highlights some of the evidence that has led to the current belief that periodontal disease may be a risk factor for adverse systemic health conditions.

Local Risk Factors for Periodontal Disease

There is ample evidence to demonstrate a relationship between bacterial plaque and gingival inflammation.9 Poor compliance with oral hygiene and other local factors expose individuals (and specific periodontal sites) to increased bacterial plaque and the risk of periodontal disease. Anatomically difficult to clean or inaccessible areas such as deep periodontal pockets, calculus deposits, furcation defects, defective restorations, pontic spaces, and poor interproximal contact areas can serve as harbors for the undisturbed growth and maturation of bacterial plaque. These inaccessible areas (especially periodontal pockets of 5 mm or more) are more likely to harbor and promote the disproportionate growth of pathogenic microorganisms because they have decreased oxygen tension. As a result, they become sites for increased growth of anaerobic, gram-negative, putative pathogenic bacterial species. In an attempt to eliminate the pathogenic microorganisms, the host mounts an inflammatory response. Gingivitis results when the tissues become inflamed. Vascularity increases, which causes edema; and tissues become erythematous. The sulcular epithelium thins and bleeds easily. The inflammatory response includes the release of many cytokines, which act as chemoattractants for the recruitment of additional inflammatory cells and perpetuates inflammation. Some of these cytokines – most notably IL-1B, TNF-a, and PGE2 – directly contribute to gingival connective tissue and alveolar bone destruction. Periodontitis results when these pathogens and the inflammatory response begin to break down the periodontal attachment. Periodontal pocket depth increases and alveolar bone resorbs.

There is little debate about the fact that periodontal pathogens contribute to the pathogenesis of periodontal disease. However, the evidence to support a relationship between bacterial plaque and the progression of periodontitis is limited. Results from well-controlled clinical studies have found that the quantity of plaque was only weakly correlated with periodontitis.[10-12]

However, it appears that the progression of periodontitis can be controlled with meticulous oral hygiene and professional cleanings (i.e., elimination of plaque can halt the progression of periodontitis).[13] Studies using qualitative measures of plaque (i.e., specific periodontal pathogens) have offered mixed results. While periodontal pathogens are essential for periodontal disease destruction, these pathogenic microorganisms alone are not sufficient to explain the differences observed in periodontal disease severity. Recent studies on various factors that influence disease progression indicate that the putative bacteria associated with periodontal disease are only slightly significant. Factors in addition to bacterial plaque must also contribute to periodontal disease destruction. Periodontitis is now seen as resulting from complex interplay of bacterial infection and host response, often modified by behavioral factors[14] (**Figure 1**). Perhaps the most fundamental change in our understanding of periodontal diseases is that not all individuals are equally susceptible to severe disease. Some individuals are more at risk for periodontitis than others. Several studies have led to the current understanding that only about 5 percent to 20 percent of the population is vulnerable to severe periodontitis.[15]

One of the best predictors of future disease progression appears to be past disease.[15] An individual who has suffered from periodontitis previously is more likely to experience future disease destruction than an individual who has not had previous disease. On the one hand, an individual with increased pocket depth (past disease) is more likely to experience further disease destruction

because the increased pocket depth is more likely to harbor and promote the growth of pathogenic bacteria. However, the other possibility is that systemic or environmental factors that originally predisposed that individual to periodontitis may continue to predispose him or her to future disease. Hence, the existence of previous disease is a positive indicator for that individual's "risk" of future disease. This latter view suggests a significant role of the host in the development and progression of periodontitis. Host factors that may have more influence on disease progression than periodontal pathogens include diabetes, smoking, stress, and genetic predisposition. Thus, it has become essential to identify systemic "host" factors that increase the risk of periodontal disease destruction.

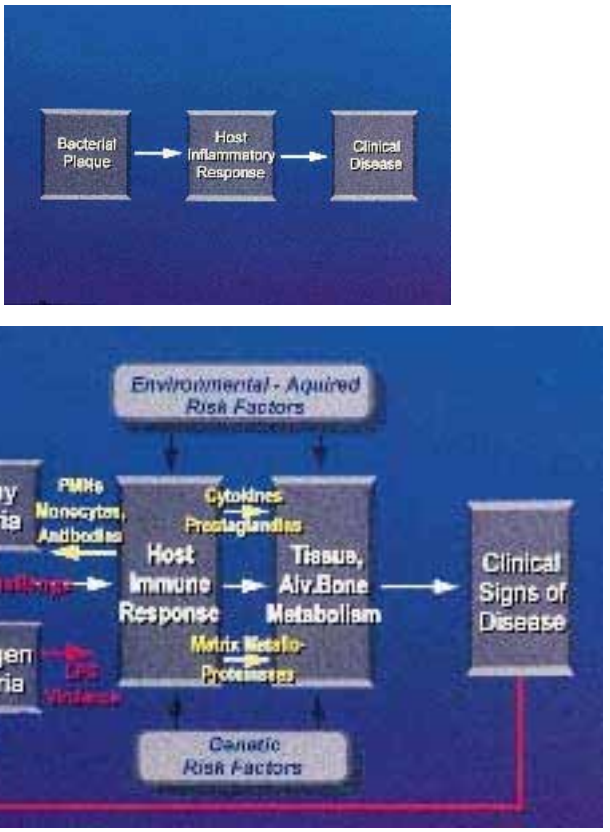

Figure 1. Over the past several decades, our understanding of periodontitis has evolved from a simplistic model to a more complex interplay between bacterial infection and host response. In the simple model (top) we assumed that pathogenic bacteria were responsible for periodontitis and all individuals were equially susceptible to disease. The current more complex model (bottom) maintains a bacterial pathogenic etiology by also takes into consideration difference in individual susceptibility as well as environmental and host response influences.

Systemic 'Host' Risk Factors

Diabetes Mellitus

Diabetes is a systemic condition that has long been associated with an increased risk and severity of periodontal disease. The reasons for this relationship are many and relate to the pathogenesis and control of diabetes. Diabetes mellitus is a disorder that results in poor metabolic control of glucose levels in the blood. The hyperglycemia that results from poor control secondarily creates systemic changes that are commonly associated with the disease. Specifically, poor glucose control in diabetics results in complications such as retinopathy (blindness), atherosclerosis (cardiovascular disease), poor wound healing, infections, and nephropathy. Both Type I (insulin-dependent) and Type II (noninsulin-dependent) diabetes are risk factors for periodontitis. Those with poorer control have greater periodontal attachment loss and bone loss and progress more rapidly than diabetics with better metabolic control of their disease.

The mechanism(s) responsible for increasing the risk in diabetics is unclear but is likely to be related to an increased susceptibility to infections, an impaired immune response, poor wound healing, or a combination of these factors. An altered periodontal microflora has also been suggested as a possible cause for the increased periodontal disease seen in diabetics. However, a study of the quantitative and qualitative aspects of the microflora (i.e., the periodontal pathogens) in both Type I and Type II diabetics revealed no significant trends nor differences between diabetics and nondiabetics.16 Some specific mechanisms that have been proposed to contribute to periodontitis in diabetics include polymorphonuclear neutrophil leukocyte dysfunction, abnormal collagen metabolism, and genetic predisposition.[17,18] While the mechanism that exacerbates periodontitis in diabetics is not well-understood, the increased risk has been well-documented; and periodontitis can be considered a complication of diabetes, especially in the poorly controlled. Page and Beck reported that diabetics experience about a 2.8 to 3.4 times greater risk of developing destructive periodontitis as compared to nondiabetics in an adult population.[1]

In many diabetic patients, especially those who are poorly controlled, the accumulation of glycosylated proteins and lipids increases as compared to well-controlled diabetics and nondiabetics. The glycosylation of proteins is a normal nonenzymatic occurence for all individuals, but it is significantly increased in those with hyperglycemia. The change permanently alters the structure of the protein and can be detected by laboratory assay. This is the basis for the latest and most accurate blood screening for diabetes – the glycosylated hemoglobin test. It is an accurate diagnostic test used to measure how well-controlled a diabetic patient has been during the previous two to three months, which is equivalent to the half-life of a red blood cell (hemoglobin molecule).

The accumulation of advanced glycosylated end products in the tissues of diabetics alters the integrity and function of the affected tissues. This may be part of the underlying mechanism responsible for some or all of the complications observed in diabetics. In poorly controlled diabetics, the vessel walls show increased basement membrane thickness due to an accumulation of advanced glycosylated end products. In the vessel wall, these changes narrow the lumen and interfere with transport across to the connective tissues. These advanced glycosylated end-product related changes on the vessel walls are responsible for the microvascular complications of diabetes such as retinopathy, atherosclerosis, poor wound healing, infections, and nephropathy. In turn, advanced glycosylated end products may be related to the increased incidence and severity of periodontal disease observed in diabetics.

The effect of advanced glycolsylated end products on periodontal disease associated with diabetes is being studied.[19] The accumulation of the end products in the periodontal tissues may contribute to periodontitis by changing the microvasculature, impairing membrane transport, and reducing the immune response as stated above. It has been suggested that advanced glycosylated end products induce an oxidant stress in the gingival vasculature that may be responsible for the accelerated injury and impaired healing seen in diabetics.[20] The accumulation of the end products may also make diabetics more suseptible to periodontitis by altering the collagen structure and function. In the case of collagen glycosylation, it increases cross-linking and results in diminished turnover.[21] The normal repair and replacement of collagen in the periodontal tissues becomes impaired and more vulnerable to damage.

Well-controlled diabetics with good oral hygiene do not show an increased risk of developing severe periodontitis. In fact, well-controlled diabetics have fewer systemic complications than poorly controlled diabetics and have been shown to respond well to periodontal therapy.[22] Perhaps more importantly with respect to systemic health, periodontal therapy has been shown to improve the ability of patients to metabolically control their diabetes.[23] They can more easily maintain normal blood sugar levels and, thus, require less insulin. There appears to be significant advantages for diabetic patients to be well-controlled and maintain good periodontal health. For this reason, awareness, improved diagnosis, and better communication between dentists and physicians have the potential to enhance the prognosis and therapy for diabetic patients. Coordinating the periodontal and medical management of diabetes benefits the patient by improving the control of hyperglycemia and enhancing the response to periodontal therapy.

Smoking and Tobacco

Smoking has long been associated with adverse systemic health effects such as respiratory disease and cancer. An analysis of data from the 1971-75 National Health and Nutritional Examination Survey in the United States showed a clear relationship between smoking and periodontitis.24 Many studies since then have provided strong evidence to support an increased risk relationship between smoking and periodontal disease severity. In a comprehensive review, Salvi and colleagues reported that the increased risk for periodontitis in smokers was 2.5 to seven times greater than that of nonsmokers.[25]

Proposed mechanisms include an altered immune response, decreased vascularity, impaired polymorphonuclear neutrophil leukocyte chemotaxis and phagocytosis, and decreased antibody production. Smoking also appears to decrease local oxygen levels. It has been suggested that the resulting decreased oxygen tension may encourage growth of anaerobic pathogens. However, experimental studies have shown that there is no difference between smokers and nonsmokers in the amount of plaque accumulation nor in the prevalence of periodontal pathogenic microorganisms.[26-28] On the other hand, Grossi and colleagues found that significantly fewer smokers became negative for periodontal pathogens (P. gingivalis and B. forsythus) than nonsmokers.[22] This is consistent with a report by Zambon and colleagues that smoking increases the risk for subgingival infection.[29] The periodontal pathogens are re-established in the periodontal pockets of smokers much more rapidly than in nonsmokers following periodontal therapy. This finding is also consistent with previous reports in that the periodontal pathogens are the same and may account

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for the poorer response to periodontal therapy seen in smokers.

Bacterial plaque (periodontal pathogens) may not be the major contributor to bone loss in the periodontal destruction seen in smokers. To appreciate the effect of smoking on alveolar bone, Bergstrom and Eliasson evaluated 235 dental hygienists (a group thought to have very good oral hygiene habits).[30] Seventy-two were smokers. Alveolar bone height was significantly reduced in smokers as compared to nonsmokers. The degree of bone loss increased with years smoking and amount smoked. Presumably in this dental-hygiene-educated population, bacterial plaque did not play a contributory role in bone loss. Only 2 percent had light calculus. The amount of smoking in pack years is important to assess when determining an individual's risk for periodontitis.

Refractory periodontitis is characterized by low levels of plaque and a poor healing response to periodontal therapy. MacFarlane and colleagues evaluated a group of 31 patients with refractory periodontitis and found that there were no chemotactic defects, but phagocytosis was significantly impaired.[31] Interestingly, they retrospectively discovered that a vast majority (28 of 31) of the refractory disease patients were smokers. The unusually high number of smokers (more than 90 percent) found in this group as compared to the percentage of smokers in Minnesota's general population (21 percent) appears to strongly implicate smoking as a major risk factor for refractory periodontal disease.

In some smokers, the appearance of the gingiva does not reflect the severity of the inflammation nor the degree of periodontal destruction. This lack of inflammation in the gingiva may be explained by decreased vascularity, increased vasoconstriction, an impaired immune response, or a combination of these factors. Smoking suppresses the vascular reaction normally observed with gingivitis and periodontitis.[32,33] This may be due to decreased vascularity (fewer new vessels and/or vasoconstriction) in the gingiva of smokers. There are mixed reports about whether nicotine causes vasoconstriction or vasodilation in gingival tissues. In central tissues such as heart muscle, nicotine causes vasodilation, whereas in peripheral tissues such as the skin, nicotine causes vasoconstriction. Some reports suggest that nicotine causes vasoconstriction of gingival tissues. However, Baab and Oberg, using a microdoppler (flux = velocity x number cells), found that nicotine caused an increase in gingival blood flow.[34] It is possible that an increase in heart rate may have contributed to this finding by increasing the velocity.

In addition to the vascular effects, nicotine causes a decreased immune response. Both oral and peripheral neutrophils are effected by nicotine. They have decreased chemotactic response to antigen and decreased phagocytic ability. The immune system in smokers is decreased via impairing polymorphonuclear neutrophil leukocyte phagocytosis and decreasing antibody levels. The antibody production, specifically IgG and IgA, is suppressed in smokers.[25]

Nicotine plays a role in periodontal destruction by up-regulating cytokine production. Payne and colleagues showed increased production of prostaglandin (PGE2) and cytokine (IL-1fl) in response to nicotine.[35] These immune regulatory mediators are known to increase periodontal destruction. The vasoconstrictive activity of nicotine along with the destructive effects of immune system up-regulation may explain the paradoxical findings in smokers, that is, less gingival inflammation and less gingival bleeding associated with more periodontal destruction as compared to observations in nonsmokers.

In addition to an increased prevalence and severity of periodontal disease, smokers have a decreased capacity to respond to surgical therapy. The healing response following periodontal therapy is decreased as compared to nonsmokers.[36] However, individuals who quit smoking recover with a healing response that is comparable to nonsmokers.[36] Smoking cessation has been shown to have beneficial effects on the periodontal tissues and the response to periodontal therapy.[37] The decreased capacity of smokers to respond well to surgical therapy appears to be true for implant therapy as well.[38] Although the findings suggest that a perioperative smoking cessation program would improve implant success rates, it is important to note that the number of patients and duration of study are limited. More studies are needed to evaluate the benefits of a short-term smoking cessation program.

Psychosocial Stress

Psychosocial stress has been associated with periodontal disease. In World War II, soldiers on the battlefield presented with trench mouth, a condition also known as acute necrotizing ulcerative gingivitis. This form of periodontal disease is known to be stress-related.[39] The mechanism of stress-induced periodontal disease destruction is not well-defined. However, it has been known for many years that increases in corticosteroids, whether exogenous or endogenous, decrease the immune response. Psychosocial stress may predispose a susceptible host by decreasing the immune response to pathogenic bacteria and altering wound healing.

Genco and colleagues are studying the various aspects of psychosocial stress such as type, duration, and the patient's ability to cope as it relates to periodontal disease.[40] They believe that psychosocial stress without the ability to cope may be more detrimental than similar stresses experienced by individuals with good coping mechanisms.

The role of stress in aggravating systemic conditions (e.g., cardiovascular disease) has been well-documented. However, the evidence to support a relationship between psychosocial stress and periodontal disease is limited. In a case-controlled analysis of psychosocial factors and adult periodontitis, Moss and colleagues showed that individuals testing positive for antibody to the periodontal pathogen B. forsythus and rating high on the depression scale were 5.3 times more likely to have periodontitis than individuals testing negative for B. forsythus antibody and depression.[41] Although these findings are suggestive, it is too early to make conclusive statements about the relationship of stress and periodontal disease destruction.

Genetic Predisposition

Genetics or familial inheritance of periodontal disease has long been suspected but difficult to prove. With their study of adult twins and periodontal disease, Michalowicz and colleagues were the first to demonstrate that, indeed, periodontal disease was linked to genetics.[42] Several studies have subsequently shown a similar relationship between juvenile periodontitis and genetic predisposition. In 1997, Kornman and colleagues published the first evidence of a specific genetic marker for susceptibility to severe chronic adult periodontitis.[43] The basis of that work was the discovery of a relationship between specific polymorphisms of the IL-1 genotype and the expressed phenotype of severe adult periodontitis. Subsequently, the first commercial genetic test for susceptibility to periodontitis became available (PST, Medical Science Systems, Flagstaff, Ariz.). The degree of increased risk of severe periodontitis for genotype-positive patients is estimated to be about 6.8 times greater as compared to genotype-negative individuals. It is estimated that approximately 30 percent of the population may be positive for this genetic marker. No previous studies have shown a genetic relationship for chronic severe adult periodontitis. It is interesting to note that this relationship was only appreciated when smokers were removed from the data analysis. The effect of smoking on periodontitis had such a strong negative impact that it outweighed the effects of genetic predisposition.

An important distinction between genotype-positive individuals and patients with existing periodontal disease is that individuals who are genotype-positive do not necessarily have the disease. It is possible that these individuals have a genetic predisposition for severe periodontitis, but they have not yet been challenged by periodontal pathogens and they do not have signs of periodontitis. Theoretically, if these genotype-positive patients are not challenged by periodontal pathogens, they may remain periodontally healthy. Once identified, these patients (as well as other patients with known risk factors) may be able to be placed into high-risk preventive programs to prevent or reduce the future incidence of disease, that is, if an individual is known to be susceptible (genotype-positive, diabetic, smoker, etc.), then every effort should be made to prevent the exposure to the periodontal pathogens (i.e., excellent oral hygiene and prevention via a frequent recall program).

Periodontal Examination

Traditionally, the complete periodontal examination consisted of evaluating and documenting several findings (signs and symptoms of disease). It typically includes an evaluation and charting of probing pocket depth, attachment loss, gingival margins, inflammation, bleeding on probing, sulcular exudate, missing teeth, contacts, and occlusion. These findings are used to detect or diagnose existing periodontal disease. In practice, re-evaluations are used to "monitor" patients and to alter the course of their treatment based on clinical findings. Unfortunately, these clinical findings do not provide any prognostic information. Even those findings thought to be predictive of future periodontal disease breakdown, such as bleeding on probing and poor oral hygiene, have failed to correlate with future disease activity.[44] Only purulence, a relatively rare finding, has been associated with periodontal disease progression.45 Conversely, lack of bleeding on probing and good oral hygiene are consistent with periodontal health.[13,46]

An example of the poor prognostic ability of traditional clinical parameters was reported by McGuire and Nunn.[47,48] They classified each tooth into prognostic groups based on clinical findings at baseline and five and eight years later. Following 12 years, patients were examined and the prognostic values were compared to actual tooth retention. Except for those teeth given a good prognosis, the predictive value of these findings (with an experienced periodontist) was poor. Teeth given a good prognosis were retained and continued to have a good prognosis. However, those teeth given a less than good (i.e., fair, poor, questionable, hopeless) prognosis were often incorrectly predicted. The accuracy of prognostic factors following five years was 43 percent. The accuracy fell to 35

percent following eight years. Based on this study, the ability to predict tooth retention using traditional clinical parameters was less than 50 percent. Recognizing that this is only one study and that teeth are sometimes extracted for reasons other than periodontal disease, the findings suggest that prognosis requires more that just an evaluation of traditional clinical parameters.

This method of examination and documentation remains an important part of identification, prevention, and treatment of patients with periodontitis. However, the problem with this method of diagnosing periodontal disease is that it is limited to detecting disease after it has occurred, and it assumes that all individuals are equally susceptible. It does not have any predictive value nor does it take into consideration differences among individual patients (i.e., host factors). To identify the patients early (ideally prior to the onset of severe periodontitis), practitioners must place an emphasis on determining risk factors for each individual via better medical history (diabetes), family history (genetic predisposition), and social history (smoking), as well as an evaluation of other environmental factors (stress). Furthermore, since patients may present without knowledge of their own medical condition (50 percent of diabetics are undiagnosed), dentists must consider referral to a physician when severe disease or poor response to therapy cannot be explained by known factors.

Periodontal Disease as a Risk Factor for Systemic Disease

One study analyzed data from the 1971-75 National Health and Nutritional Examination Survey and correlated it with cardiovascular data. The report concluded that people with periodontitis at baseline had a 25 percent greater risk of subsequent coronary heart disease than those without periodontitis.[7] The risk was especially high for men under age 50 (1.7 times greater). This analysis also found that periodontitis and poor oral hygiene were more strongly associated with total mortality th an with coronary heart disease, which could indicate that neglect of oral health is more an indicator of poor health habits than an etiologic factor.

The mechanism(s) for the relationship between periodontitis and the systemic effects it may have are beginning to be understood. As an example, Beck and colleagues suggest that periodontitis, once established, represents a biologic burden of endotoxin and inflammatory cytokines that serves to initiate and exacerbate atherosclerotic and thrombogenic events.[6] Herzberg and Meyers' study on the effects of oral flora on platelets supports this hypothesis.[2] They found that S. sauguis induced platelets to aggregate, and they propose a hypothesis that they may cause coronary thrombosis. Additional hypotheses are being studied to evaluate the effect of periodontitis on respiratory diseases and the incidence of preterm, low-birth-weight babies.[3,5] Interested readers are referred to a comprehensive report from the 1997 Sunstar-Chapel Hill Symposium on Periodontal Disease and Human Health published in the Annals of Periodontology.[8]

It is interesting to note and important to remember that these hypotheses are reminiscent of the focal infection theories that fell out of favor in the 1950s and '60s because of a lack of evidence. The link between periodontitis and systemic disease requires further study with well-controlled clinical trials.

Conclusion

Once again, the approach to the diagnosis and treatment of periodontal disease is changing. The disease has not changed, but dentistry's understanding of the pathogenesis and appreciation for the influence of host factors has improved. As a result, the approach to the management of the disease is evolving. This article has highlighted some of the emerging evidence that links periodontal disease and systemic health. There is a rapidly growing body of data that supports a periodontal medicine interrelationship. Current evidence suggests that systemic factors contribute to the severity of periodontitis, and periodontitis may be a risk factor for systemic diseases. As part of a comprehensive examination of patients for periodontal disease, dental practitioners must act more like physicians to evaluate systemic illnesses and other conditions that may contribute to the risk and severity of periodontal disease. Conversely, physicians must understand the role of periodontitis in the health of their patients and become aware of the signs of severe periodontitis. This is the beginning of a new era in periodontics and provides an opportunity for dentists to develop new relationships with physician colleagues. Dentists should be encouraged to communicate with physicians about the health of their patients, and physicians should be alerted to the possible risks of severe periodontitis.

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Crown Lengthening Surgery: A Restorative Driven Periodontal Procedure

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ABSTRACT Improper management of the periodontal tissues during restorative procedures is a common, but often overlooked, cause of failure. When a restoration is placed, the preservation of an intact, healthy periodontium is necessary to maintain the tooth or teeth being restored. Predictable long-term restorative success requires a combination of restorative principles with the correct management of the periodontal tissues.

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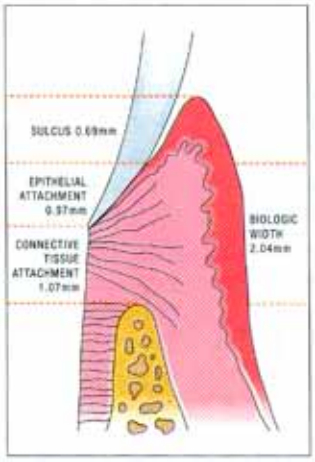
The replacement of form, function, and esthetics is the primary goal of restorative dentistry. Equally important is doing no harm when restorations are placed. Improper management of the periodontal tissues during restorative procedures is a common, but often overlooked, cause of failure. When a restoration is placed, the preservation of an intact, healthy periodontium is necessary to maintain the tooth or teeth being restored. The restorative dentist must attempt to eliminate all factors that could lead to the accumulation of bacterial plaque and its subsequent effects on the gingival tissues, root surfaces, and underlying alveolar bone.

Predictable long-term restorative success requires a combination of restorative principles with the correct management of the periodontal tissues. One factor that is of particular importance is the potentially damaging result to the periodontium when margins are placed below the gingival margin. To avoid these potential problems to the supporting structures of the teeth, clinical crown lengthening can provide adequate clinical tooth structure to enable the placement of margins either coronal to or at the gingival margin.

Anatomy of the Gingival Complex

Clinical doctrines and common myths regarding the gingival response to restorative materials and the limitations of intracrevicular tooth preparation make it difficult to place restorative margins subgingivally. Because tissue components



Figures 1a-1c. Comparison of gingival anatomy and biologic width relative to the cemento-enamel junction.

Figure 1a. Normal crest.

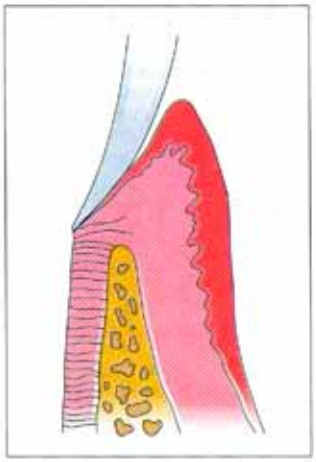


Figure 1b. High crest.

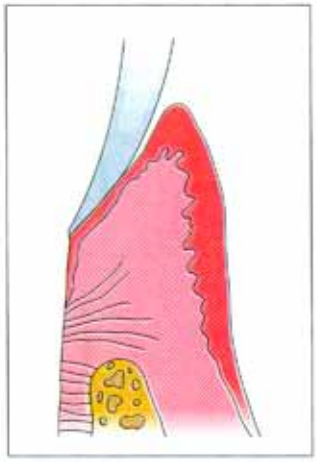


Figure 1c. Low crest.

cannot be visualized, the guidelines of sulcus depth are often misunderstood and clinically mismanaged. Therefore, before discussing crown lengthening, it is important to review the anatomy of the supra-alveolar tissues of the healthy periodontium and the relevance of these dimensions to the position of restoration margins.

The supra-alveolar tissues include the gingival sulcus and the gingival attachment. The gingival attachment joins the gingiva to the tooth and is made up of the connective tissue attachment and the epithelial attachment. The gingival attachment is made up of connective tissue fibers that are embedded in cementum. The epithelial attachment is an adhesion of epithelial cells through hemidesmosomes.

The concept of a minimum dimension of tissue from the alveolar crest to the bottom of the gingival sulcus is based on a study by Garguilo and colleagues.[1] They examined 30 cadavers with clinically healthy periodontia and reported on the average histologic dimensions of the connective tissue attachment, the epithelial attachment, and the gingival sulcus. They found that there appears to be a proportional relationship between the crest of the alveolar bone, the connective tissue attachment, and the epithelial attachment. The investigators found that the average histologic dimension of the connective tissue fibers was 1.07 mm, the average histologic dimension of the epithelial attachment was 0.97 mm, and the average histologic dimension of the sulcus was 0.69 mm. The combined dimension of the junctional epithelium and connective tissue attachments is referred to as the "biologic width"[2] Subsequent authors[3,4] have also shown that a definite dimensional relationship exists among the alveolar crest, the supra-alveolar connective tissue attachment, the junctional epithelium, and the base of the gingival sulcus.

Understanding and clinically managing the concept of biological width and the level of the osseous crest is key to maintaining periodontal health in the presence of dental restorations. The location of a restorative margin relative to the crest of the alveolar bone is more critical for preserving gingival health than its distance below the free gingival margin.[5] The restorative dentist must be able to determine the height of the osseous crest and width of the gingival attachment before placing intracrevicular margins. This is done to prevent impingement of the soft tissue attachment, otherwise referred to as violation of the biologic width.

Violations of Biologic Width

Several studies have shown that the position of the crown margin in relation to the gingiva can significantly affect the gingival index, as well as the pocket depth and the position of the epithelial attachment.[6-10] It was shown that crown margins positioned subgingivally were associated with the most gingival inflammation, whereas supragingivally located crown margins were associated with the least gingival inflammation.[6,9-10] Valderhaug[7,8] showed that loss of periodontal attachment was significantly higher around teeth with subgingivally located crown margins than around similar teeth with crown margins located supragingivally. Furthermore, subgingivally prepared teeth exhibited deeper pockets than teeth prepared with the margin at the height of the gingiva or supragingivally. The same study also showed that after five years, 30 percent of the subgingivally located crown margins were associated with gingival recession.

There are also reports in the periodontal literature that claim that in the presence of inflammation, restorations that impinge upon the gingival attachment or biologic width will trap bacterial plaque, induce inflammation, and increase the severity of periodontal breakdown[3-5,11-14] (**Figure 2**). Maynard and Wilson[3] reported that

violation of the biologic width will lead to a progressive inflammation with downgrowth of the epithelial attachment and loss of connective tissue attachment. Allen[13] reports that wherever the biologic width is violated, there is a reaction by the periodontium. Alveolar bone will resorb inconsistently in an attempt to provide space for a new connective tissue attachment, which will result in an increase in probing depth. Re-establishment of the periodontal attachment at a more apical position and a deepened sulcus, combined with a deep subgingival restorative margin, frequently lead to chronic inflammation and localized periodontal breakdown. This is especially applicable in periodontally susceptible patients.

It is possible that the gingival inflammation associated with restorations that impinge on the gingival attachment is not from a physical insult, but from a bacterial insult. Crown margins are inherently imperfect and will eventually collect bacterial plaque. The average marginal fit of gold restorations is 57 m and ceramic restorations is 48 m.[15] Christensen[16] has stated that all cast restorations have cement lines and that most studies show these lines to be at least 20 to 40 m thick. Since the size of a typical microorganism is only about 1 m thick, it can be assumed that most crown margins will eventually harbor bacterial plaque. Several studies have shown that subgingival crown margins interfere with gingival health.[6-10]

Waerhaug[17] claimed that the inflammatory lesion radiates 1 to 2 mm from the plaque front. Therefore, it is probable that a 1 to 2 mm zone of inflammation is going to be contiguous with a subgingival restorative margin. Waerhaug[18] also stated that the loss of connective tissue attachment rarely occurred when the plaque front was less than 1.2 mm from the apical border of the junctional epithelium and that there was no loss of bone when the plaque front was greater than 2.7 mm coronal to the bone. It has been claimed that the rationale for obtaining 3 mm of distance from the expected restorative margin to the alveolar bone should not be to allow for the gingival attachment and sulcus, but instead to position the restorative margin, with its anticipated plaque deposit, beyond the 2.7 mm danger zone from the bone.[19]

Regardless of the etiology of the inflammation and bone loss, to ensure periodontal health in the presence of subgingival restorations, there must be a minimum amount of sound tooth structure coronal to the alveolar crest. Most authors[3-5,12] recommend that when supragingival margins are not feasible, restorative margins should be placed at least 3 mm from the alveolar crest. This dimension allows a distance of 1 mm for each part of the gingival attachment (connective tissue attachment and epithelial attachment), for a total of 2 mm. The additional 1 mm is for a healthy gingival sulcus. Other authors have recommended 4 mm or even 5 mm for the working dimension of biologic width.[20,21] However, these authors take into consideration an additional 1 to 2 mm of tooth structure for a restorative margin.

It is also important to remember that the above guidelines are averages. Each clinical situation varies and should be examined prior to margin placement.

Another factor to take into consideration is the relationship of the position of the biologic width relative to the cementoenamel junction. In the normal crestal to soft tissue relationship, the junction of the connective tissue fibers and the epithelial attachment is located at the cementoenamel junction. In what is termed a "high crest," the alveolar crest is at the cementoenamel junction, resulting in a minimal connective tissue attachment and what is often called "delayed passive eruption." A "low crest" is when the osseous crest is more than 2 to 3 mm apical to the cementoenamel junction. This most often results in a long junctional epithelial attachment, with the junction of the epithelial attachment and connective tissue fibers on cementum.

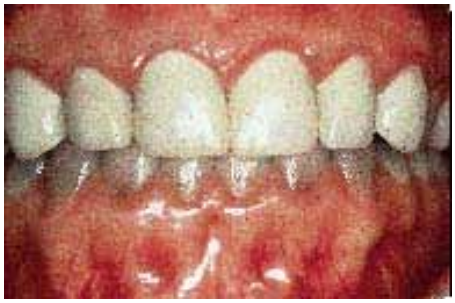


Figure 2a. Chronic gingival inflammation around anterior crowns.

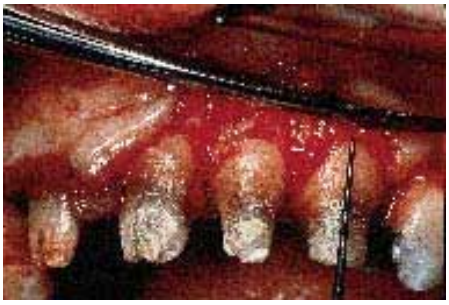


Figure 2b. Elevation of a flap shows crown margins in close proximity to alveolar crest and violation of gingival attachment or "biologic width".

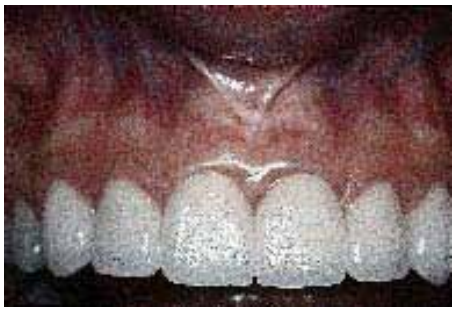


Figure 2c. New restorations after surgery to re-establish room for gingival attachment.

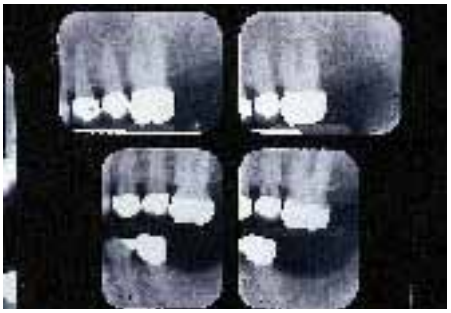


Figure 3a. Radiographs of maillary left showing decay under existing restorations Nos. 12 and 13 and supra-eruption No. 14.

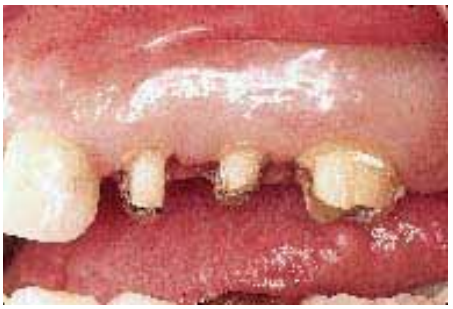


Figure 3b. Buccal view of maxillary left showing subgingival margins and soft tissue cratering.

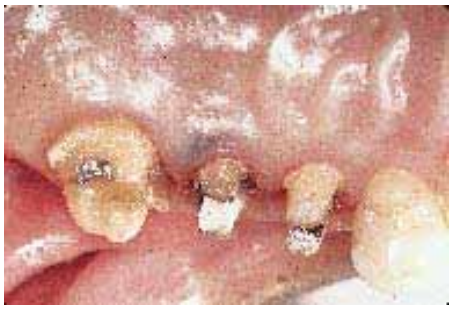


Figure 3c. Palatal view of maxillary left showing short clinical crowns and extent of subgingival decay removal.

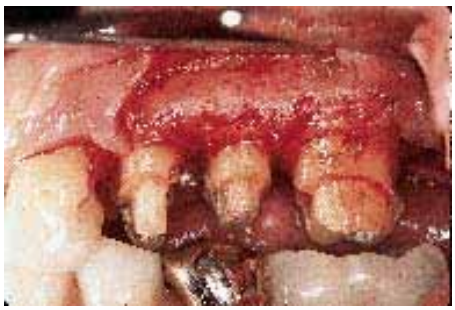


Figure 3d. Buccal view of maxillary left pre-osseous resection.

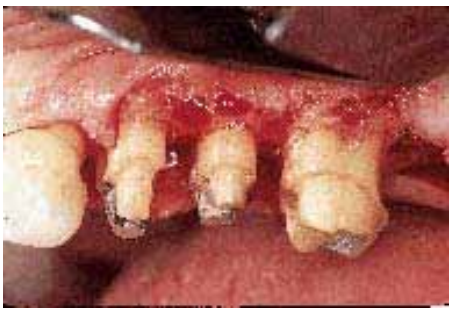


Figure 3e. Buccal view of maxillary left post-osseous resection.

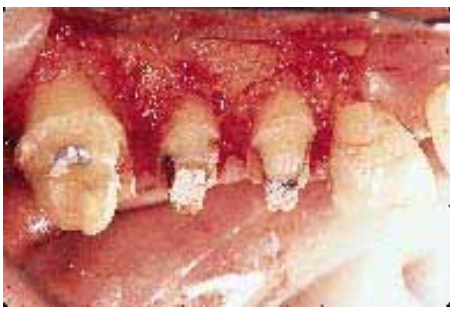


Figure 3f. Palatal view of maxillary left pre-osseous resection.

Ochsenbein and Ross[22] further defined tissue types as either flat or scalloped. A scalloped architecture will have a low crest and is usually found with a thin periodontium, causing a tendency for soft tissue recession. A flat architecture is usually found in patients with thicker tissue. Only by sounding to bone under local anesthesia can these relationships be determined.

Measurements should be made from the free gingival margin to the osseous crest with a periodontal probe. It is also important to remember that the straight and interproximal surfaces have different requirements. In health, the facial aspect has approximately a 3 mm depth from the gingival margin to the osseous crest. The interproximal surfaces have a gingival margin to osseous crest depth ranging from 3 to 4.5 mm. The interproximal depth varies depending on the amount of the scallop of the gingival tissue relative to the scallop of the interproximal alveolar bone. The gingival scallop is always equal to or greater than the underlying osseous scallop, which is greatest in the anterior teeth and flattens out posteriorly. Furthermore, it is important to remember that the osseous crest parallels the cementoenamel junction circumferentially and that the biological width follows the shape of the osseous crest.

Surgical Crown Lengthening

Crown lengthening is a surgical procedure performed on a healthy periodontium that requires exposure of adequate tooth structure for restorative purposes (**Figure 3**). Several techniques are available, depending upon the proposed location of the restorative margin, the location of the alveolar crest and gingival margin, the width of the keratinized attached tissue, and the amount of exposed tooth structure available.

Indications

Indications for surgical crown lengthening are periodontal, restorative, and esthetic. Periodontal considerations include cases of "delayed passive eruption" and where intracrevicular placement of the restorative margin encroaches on the gingival attachment and may lead to inflammatory periodontal disease. Restorative considerations include lack of retention due to short clinical crowns; treatment of overerupted teeth to correct the occlusal plane; presence of subgingival caries; and presence of a subgingival crown or tooth fracture, root perforation, or subgingival root resorption (**Figure 4**). Esthetic considerations include changing a "gummy smile," and marked discrepancies in the height of the gingiva around teeth in the esthetic zone.

Esthetic needs may also demand orthodontic eruption prior to surgical crown lengthening to maintain existing

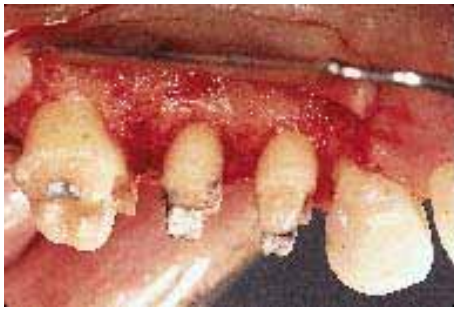


Figure 3g. Palatal view of maxillary left pre-osseous resection.

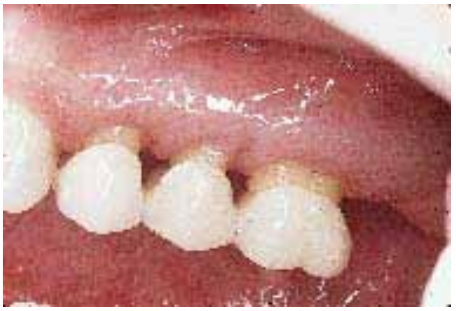


Figure 3h. Buccal view of healing post-crown-lengthening surgery showing amount of clinical crown length gained on buccal.

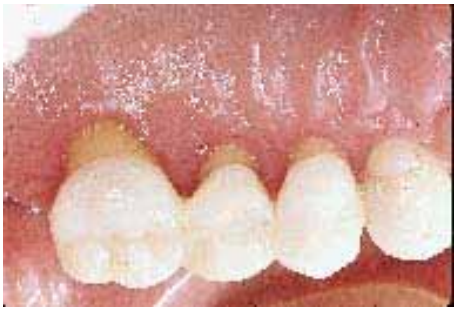


Figure 3i. Palatal view of healing post-crown-lengthening surgercy showing amount of clinical crown length gained on palatal.

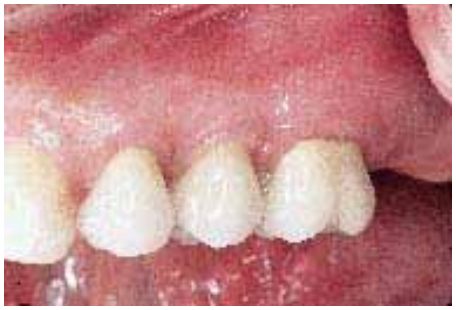


Figure 3j. Buccal view of final restoration (final restoration by Dr. Michelle IKoma).

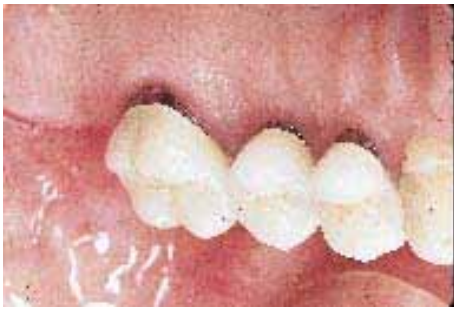


Figure 3k. Palatal view of final restoration.

gingival contours. This is most common in the anterior maxilla with a high smile line (**Figure 5**).

The traditional forced-eruption technique causes the gingival tissues to erupt with the tooth (**Figure 6**). A minor periodontal surgical procedure is then necessary to return the gingival margin to its proper location. There are several advantages that forced eruption prior to surgical crown lengthening provide as opposed to surgery alone that may be significant in varied clinical situations. In the maxillary anterior zone, forced eruption places the gingiva and underlying bone around the erupted tooth at a more coronal position. When surgical crown lengthening is completed, the surgeon is able to place the gingival tissues and osseous crest at a level that will be more conducive to a cosmetic result.

Another significant advantage to orthodontically erupting a tooth prior to surgical crown lengthening is that postsurgically, the crown-to-root ratio remains virtually the same or is improved compared to that obtained with surgery alone. In addition, supporting bone from the adjacent teeth does not have to be sacrificed to obtain sufficient clinical crown length for the tooth requiring treatment.

The disadvantage of forced eruption is that the diameter of the root decreases as the preparation moves apically. The final restoration will therefore exhibit a greater degree of taper from the gingival margin to the incisal edge. This will require greater attention to the gingival areas to avoid overcontoured margins. In addition, tooth preparation of the smaller root segment will require modification if one is to achieve a healthy blending of restorative materials, gingival health, and esthetics. Teeth with moderate to severe tapering of the root form are contraindicated for forced eruption.

Rapid orthodontic forced eruption[14] (two to six weeks) can be followed by surgical crown lengthening as long as the tooth is stabilized. It is not always necessary to wait for bone maturation of the attachment that follows with the eruption.

Another indication for forced eruption prior to crown lengthening with osseous resection is teeth adjacent to implants or future implant sites. Random alveolar crest reduction of partially edentulous ridges to achieve a flat positive bone architecture adjacent to the treated tooth is contraindicated.

Contraindications

Contraindications for surgical crown lengthening include teeth that are nonrestorable, teeth or adjacent teeth that would be compromised either functionally or esthetically, and teeth whose value is not compatible with the procedures necessary to save it. The advantages of retaining a tooth in terms of its significance to the overall treatment plan must be weighed against the extent of the procedures needed to properly restore the tooth. This is especially important today with the accessibility to highly predictable dental implants. Other factors to evaluate when considering crown lengthening procedures are the crown-to-root ratio after the tooth is restored and the ability of the patient to maintain the periodontium in a state of health after the restorative procedures have been completed.

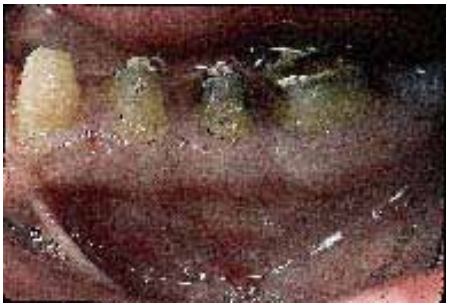


Figure 4a. Buccal view of mandibular left shwoing short clinical crowns.

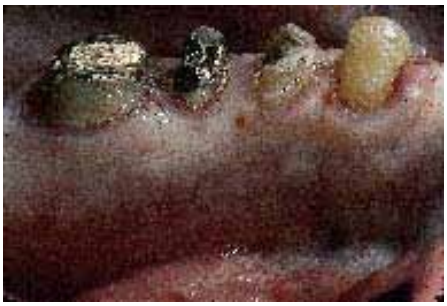


Figure 4b. Lingual view of mandibular left showing short clinical crowns.

Figure 4c. Buccal view of mandibular left showing gain in clinical crown length after osseous resective surgery.

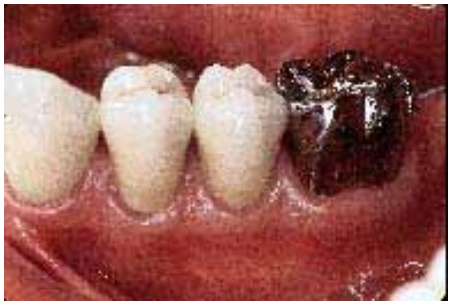


Figure 4d. Surgical crown lengthening to gain clinical crown length allows the restorative dentist to gain retention without placing restorative margins subgingival. Note the tissue health obtained when restorative margins are placed at thte ginfival margin (final restoration by Dr. Blake Mueller).

Restorative Requirements

It is extremely common to find short clinical crowns, which pose a problem to the restorative dentist during crown and bridge procedures. Schwartz and colleagues[23] found that loss of retention was the second most frequently encountered complication following caries development in fixed partial dentures. When undiagnosed for some time, loss of retention can lead to serious problems. In an attempt to reduce the risk of loss of retention, the abutments should be prepared following a carefully chosen design and not prepared further subgingival. A review of the ideal tooth preparation requirements follows:[24]

- Adequate length for proper retention and resistance. The ideal preparation must have at least 4 mm of axial wall height with a minimum of 2 to 3 mm of sound tooth structure circumferentially and a maximum convergence angle of 6 to 10 degrees.
- Sufficient axial reduction for adequate esthetic rendition. Metal-ceramic and all-ceramic crowns require 1.5 to 2 mm of tooth reduction to allow proper thickness of the ceramic veneer.
- Sufficient occlusal reduction for occlusal function and anterior guidance. Metal ceramic crowns require 2 mm of occlusal reduction, less if full cast gold restorations are utilized.

In the anterior maxilla, Kois[25] has advocated having at least 2 mm of solid tooth structure on the buccal and lingual surfaces of the tooth preparation. Kois recommends finishing the interproximal margin parallel to the cementoenamel junction without being concerned with the 2 mm rule of solid tooth structure, as long as the margin is on sound tooth structure. The scalloped tissue form follows the cementoenamel junction and underlying osseous crest, which is more coronally positioned in the interproximal zones. During crown preparation, it is easy to continue the interproximal crown margin at the same level as the buccal or lingual margin and violate the biologic width interproximally. This is most common when preparing anterior teeth, as crown margins are placed subgingivally for cosmetic purposes, and the underlying osseous scallop changes so dramatically from the straight to the interproximal surfaces.

Kois recommends avoiding the use of flat-end diamond burs when extending beyond the line angles. The tendency is to follow the flat shoulder margin from the straight buccal surface past the line angle into the interproximal. Adjustment in the depth of the interproximal margin using a round-end tapered diamond bur is recommended. This will prevent violation of the biologic width in the interproximal area, which is the most susceptible area.

Treatment Plan and Sequence

Prior to crown lengthening procedures, a combined periodontal and restorative treatment plan is essential. The sequence of therapy is very important to achieve desired clinical results. Diagnostic procedures include periodontal probing depths and radiographs to determine root form, root proximity, and bone levels. An esthetic examination includes evaluation of the smile line (position of the upper lip relative to incisal edge position and gingival facial levels, i.e., the amount of gingival exposure during speech as well as smiling).[13] The tissue type and the amount of keratinized tissue along with the patient's esthetic concerns, desires, and expectations are extremely

Figure 5a. Patient presents with short clinical anterior teeth, resulting in a "gummy smile."

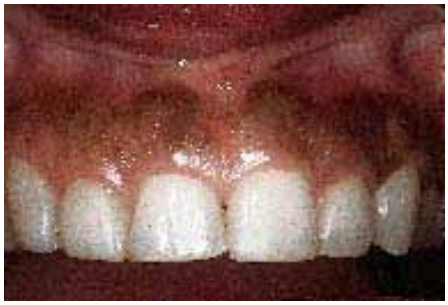


Figure 5b. Close-up view of patient's short clinical anterior teeth (not flat architecture of periodontium).

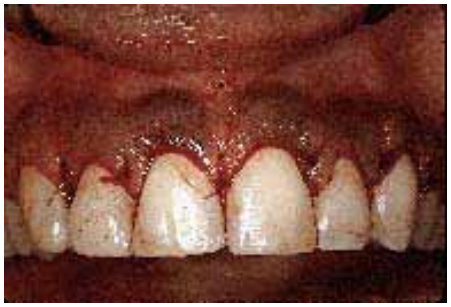


Figure 5c. Surgical crown lengthening was completed to lengthen anterior clinical crown length.

Figure 5d. Postsurgical results showing significantly improved anterior cosmetics.

important. The prognosis of the tooth or teeth to be treated along with the adjacent teeth is required. Compromising the adjacent dentition to save a tooth with a poor prognosis is a contraindication to treatment. Alternative options such as implants or fixed partial dentures should be considered. Mounted casts with a diagnostic wax-up of the future restorative plan are always indicated. Once the ideal future crown contours have been established, the restorative and periodontal team can work in reverse to achieve the desired clinical results.

Initial therapy includes scaling and root planing and providing the patient with oral hygiene instructions. A provisional restoration should be placed prior to the surgical procedure. When inadequate restorations exists, they should be removed and proper provisional restorations fabricated. This will help reduce the inflammatory component of the dentogingival complex and permit re-evaluation of tissue response before deciding what type of surgical correction is necessary. If endodontic treatment is indicated, root canal therapy should be completed prior to the surgery. This will prevent later surprises, especially if extensive decay exists under old restorations. It is always better and easier to make a judgment regarding prognosis when the restoration and decay have been removed. Removal of the provisional restorations at the time of surgery facilitates access to the interproximal areas and allows the periodontist to make the important decision about how much ostectomy (removal of supporting bone) is enough. It is important to remember that this is not a one-tooth procedure. Creating a level osseous contour that allows the soft tissue to follow is an important concept for long-term periodontal stability and maintenance.

If the margin is close to the osseous crest (as is often the case with fractured teeth or extensive decay) resulting in excessive ostectomy that will compromise the adjacent teeth, orthodontic forced eruption of single rooted teeth can be considered.[27] Decay extending close to furcations on molars can be evaluated for possible root amputation procedures. The restorative requirements postsurgery in the remaining furcations is the same as described. i.e., 3 mm margin to osseous crest. If this cannot be achieved, the nonrestorable tooth should be extracted.

Combined implant and conventional restorative treatment in the anterior zone should always be carefully analyzed. If crown lengthening is required on the teeth adjacent to implant sites, this procedure should precede the implant placement. Once healing has occurred with a stable desired gingival architecture, the implants can be placed with the hex platform at the desired vertical height relative to the teeth. It is very easy to misjudge this if the amount of osseous reduction has not already been determined and completed. Once the implant is placed, it is impossible to correct. If bone level changes are not anticipated, then implant placement can precede soft tissue alterations. This can usually be performed simultaneously with the second stage implant procedure.

Healing

In non-esthetic (posterior) areas, the patient should be re-evaluated six weeks postsurgery prior to continuing with final restorative procedures. Margins should be kept at the gingival margin. In the anterior esthetic zone, a longer

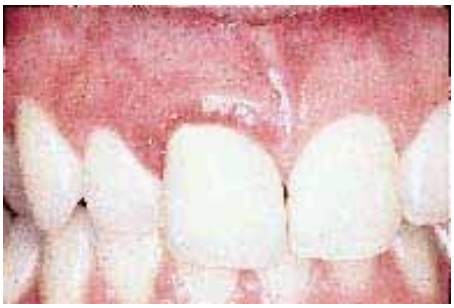


Figure 6a. Chronic gingival inflammation around crown restoration No. 8.

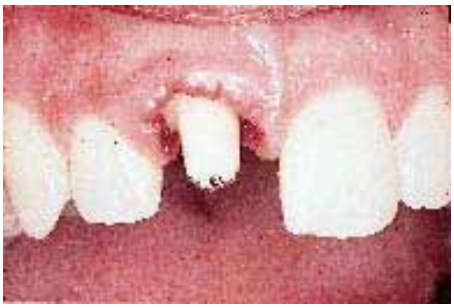


Figure 6b. REmoval of the restoration No. 8 shows restorative margin placed deep into gingival sulcus.

Figure 6c. Orthodontic extrusion was completed to coronally position osseous and gigival tissues prior to crown lengthening surgery.

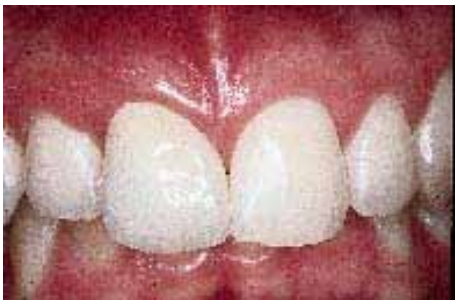


Figure 6d. Final restoration No. 8 just slightly below gingival margin shows improved gingival health (final restoration by Dr. Gary Solnit).

healing period is recommended. Wise recommends waiting 21 weeks for soft tissue gingival margin stability.[28] Kois has suggested waiting longer.[25]

During final tooth preparation, margins should be placed supragingival or, if cosmetic concerns direct, at the gingival margin. If cosmetics dictate subgingival margin placement, the dentist preparing the teeth should not only be aware of the cementoenamel junction and soft tissue form, but should also again sound to bone. Studies in the periodontal literature indicate that the postsurgical dimension of biologic width will approximate the amount present prior to surgery.[29,30]

It is as important postsurgically as it is prior to surgery to keep in mind that the location of a restorative margin relative to the crest of the alveolar bone is more critical for preserving gingival health that its distance below the free gingival margin. (Kois recommends keeping the margin 3 mm from the osseous crest.[5]) Prior to final margin placement, the restorative dentist should identify the level of the soft tissue in relation to the osseous crest before the retraction cord is placed. If this relationship is not properly identified, as the biologic width redevelops, the preparation margin can easily end up being too far subgingival. As described earlier, this relationship can set the stage for progressive periodontal breakdown.

Use of a rotating instrument beneath the gingival margin traumatizes the gingival, sulcular epithelium, and possibly even the gingival attachment. The trauma caused by a rotating instrument may be reversible.[31] However, in some cases, permanent loss of periodontal attachment may result.10

Tissue retraction is a traumatic procedure requiring gentle manipulation of the soft tissues. Loe[10] showed that with normal pressure, part of the retraction cord could impinge into the biological width. Excessive tearing of the dentogingival complex and inappropriate use of chemicals (buffered 14 percent aluminum chloride, Hemodent solution) used to control gingival crevicular fluid and bleeding will induce recession, exposing crown margins. It is safe to leave the impregnated cord up to 15 minutes. Depending on the thickness of the gingival tissue and the sulcus depth encountered, Chiche[32] recommends a single string or selective double-string technique. It is important to distinguish between thick and thin gingival tissue because the tissue behaves differently when it is surgically manipulated. It is easier to maintain papilla height postsurgically with thick tissue. Thin tissue tends to shrink more. In respect to crown margin placement, it is easy to hide a margin subgingivally in a thick periodontium as compared to thin tissue. Depending on the thickness of the tissue, a certain amount of rebound of the soft tissue is expected. It can take one to three years for the final mature tissue architecture to reform. It is not practical to keep patients in provisional restorations for so long. The important aspect of this concept is to place a final restoration that does not impinge the gingival embrasure and allows space for the interproximal gingival tissue to rebound. The final restoration should be fabricated to allow for final maturation of the gingiva and rebound of the interproximal gingival tissue. As long as the apical end of the final crown restoration is 5 mm or less from the interproximal bone, Tarnow[1] found that a papilla was always maintained or reformed postsurgically.

Conclusion

In the past, esthetics usually demanded subgingival margin placement. Current porcelain materials should allow more restorative margins to be finished at the level of the soft tissue. If the ideal relationship between bone and margin is respected, a healthy periodontium will follow.

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The Emperor's New Dental Smock

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It should be obvious to anyone who has not spent his waking hours during the past year cataloguing the indiscretions of our national leader that notable changes are taking place within our profession.

Specifically, I speak of the costuming we embrace as health professionals. It is important that the public can readily differentiate us from other professional persons such as those engaged in public landfill projects or employed in the service of the Good Humor Company.

In the early days of dentistry, when long frock coats and vests festooned with gold watch chains and elks' teeth were considered de rigueur for dentists, black was the color that most nearly expressed the seriousness-of-purpose (SOP) we wished to project. It could also be worn daily for upward of a month without visible blood splatters -- certainly a plus in those pre-high-volume-evacuation days. Additionally, the attire was appropriate at a formal wedding, a funeral or the ribbon-cutting ceremony at a new livery stable. Its equal as an all-purpose uniform has never been matched.

Suddenly, however, black was out and white was in. White represented purity, sterility and a new SOP as opposed to, say, candy apple red or hot pink. Backed by a coalition of button manufacturers and laundry operators, the medical triumvirate of Marcus Welby, Ben Casey and Dr. Kildare dictated health care costuming for years despite the fact that not one of them knew a speculum from a matrix band. It was during the latter part of this era that skirts for auxiliary personnel disappeared almost overnight, much in the same way an impacting astral body is supposed to have wiped out the dinosaur population.

Enter the Sixties. This was a period when "doing your own thing" became paramount and, to people of my generation, indicated that the Decline of Civilization initiated by the Beatles had gone into warp speed. To dentists young enough not to recognize the names of Fred Allen, the Ritz Brothers, Horace Heidt and Glen Gray, the opportunity to state their new SOP was not to be missed. White was definitely passé. Pastels were hot, as were paisleys and tie-dyes. T-shirts with clever messages such as "I'm with Stupid" were common enough that we could all express our individuality in unison. The freedom from frequent barbering as well as an unlimited selection of footwear were the most precious things to have evolved since the advent of Bis-gma. Dentists, in many instances, were difficult to distinguish from members of the Cirque du Soleil. Not only did they suffer no embarrassment from this, they openly advertised it in the media, offering the opinion that cowards had little

to fear from them, providing they presented with a valid insurance plan. Little did we suspect that just around the corner a brand-new seriousness-of-purpose was about to be unleashed.

OSHA took a good look at our uniforms and opined that we were the laughingstock of the infectious germ world. Let us cover every bodily surface, it said, with something as impervious to bacteria as Kevlar is to bullets. Spray it with Lysol and discard it at the end of each patient encounter, the recommendation went. God forbid you should launder it at home, it warned.

At the same time OSHA was directing infection control, large corporate structures were sticking their noses into the tent with managed care ideas. Individualism was in the decline, but uniform purveyors were not caught napping at their Singers. The emphasis now shifts to the Dental Team.

To impress patients with the concept of intensive team efficiency, modern dental office personnel can appear united in SOP by wearing identical uniforms. It works for McDonalds and K-Mart, the reasoning goes.

Personalized with an office logo, a typical dental outfit features a little smiling molar brandishing a toothbrush in one "hand" and a floss container in the other. Embroidered over an area beneath which a compassionate heart lurks, the effect is enough to allay the qualms of the most fearful. Below the logo is the wearer's name (first only -- we're friendly folks) and rank. The dentist has his or her title of "DENTIST" boldly depicted so there's no question about who's the boss.

It is not unusual to see a dentist and staff all sporting the team outfit milling about in lockstep at one of the Scientific Sessions. It brings to mind ducklings that have passionately imprinted on their mother. Unfortunately, once the OSHA stuff has been donned, all this is lost to the patient, but the staff is aware of who and what they are and that's enough to sustain them between paydays.

We may be in the last throes of individualism on this planet. My observation of past and current sci-fi movies confirms that before long, all of us will be wearing a uniform of silver lamé spandex. All of us -- and that includes visiting aliens with the exception of those with multiple tentacles -- will be encased in seamless, shiny, form-fitting suits. Authority will be vested in the timbre of one's voice, which should be similar to that projected by James Earl Jones. It couldn't hurt to have one of those Star Wars wands that go woom, woom! when waved at an antagonist.

Dentists would be well-advised to keep this in mind when ordering supplies.