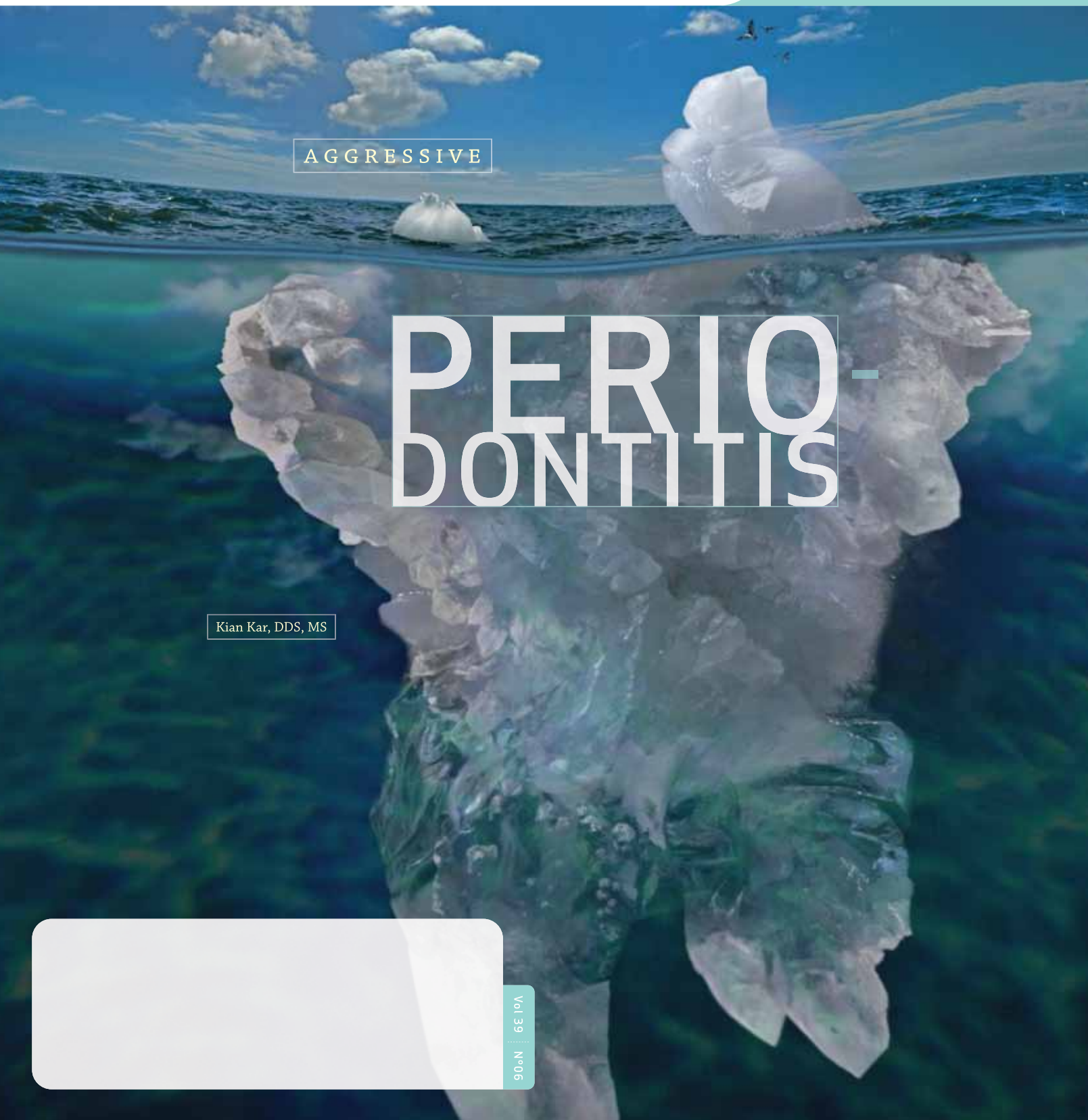
OF THE CALIFORNIA DENTAL ASSOCIATION

Journal

JUNE 2011



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Kian Kar, DDS, MS

Vol 39 No06

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DEPARTMENTS

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FEATURES

OF THE CALIFORNIA DENTAL ASSOCIATION

Journal

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JUNE 2011

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SEPTEMBER: *Xerostomia*

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CDA JOURNAL, VOL 39, Nº6

Bumble Bee: My Nemesis

KERRY K. CARNEY, DDS

Tuna fish. What would my life have been without tuna fish? I always liked tuna fish sandwiches. That was fortunate, because a tuna fish sandwich was what I had every day for my school lunch. That would be six ounces of tuna, five days a week, 36 weeks a year, or approximately 180 servings of tuna fish annually. Times that by 12 years of school (I did not have the advantage of kindergarten) for a grand total of 2,160 tuna fish sandwiches or 12,960 ounces or 810 pounds of tuna. That would be an underestimate of my lifetime total intake since the occasional extra scholastic tuna fish sandwich has not been taken into consideration.

The FDA recommendations for tuna consumption are no more than one six-ounce serving of albacore tuna a week. Add in the fact that the mayo and tuna mixture in my school lunch sandwiches incubated at room temperature for approximately six hours before ingestion. Thinking back on what I consumed as a child, it is amazing I can put a noun and a verb together at all. I am lucky to be alive.

Maybe if I had not ingested all that methylmercury-laced tuna, I might have led a different life. How much smarter would I have been? What other college might I have gone to? Maybe I would have realized my calling to dentistry earlier. Maybe I would know more poetry by heart and have fewer of my brain cells occupied with the archiving of the lyrics to way too many songs by the Turtles.

I am not trying to argue facetiously that mercury ingestion is a good thing. Far from it, on my vet's recommendation, I don't even give my cats tuna more than twice a week. (I hate to think what trouble they would get into if they had even 10 fewer brain cells than they operate on right now.)

The mercury issue itself is like quicksilver: difficult to contain, scattering, and flowing into other areas.

A significant level of bio-available mercury in the environment has the potential for human developmental and neurological harm.

We all try our best to keep mercury out of the environment. I have always felt pretty self-righteous about safely disposing of technology that may contain amounts of free mercury. Then one day, I received a letter from the local water treatment department advising me that as the owner of a dental office I would be part of a new program to encourage the installation of amalgam separators.

The letter proceeded to advise me that dental offices were the source of a significant proportion of the mercury that flows into the municipal water treatment plant. We have had an amalgam separator installed at our office for a number of years but I was incensed. Everyone knows that industry and mining are the major sources of the mercury in the bay water system. Immediately, I was on the phone to CDA to get the documentation I needed to prove to the local bureaucrat who wrote the letter that he was mistaken.

One of the benefits of being a member of CDA is that thanks to the expertise and availability of the staff, one can usually get the necessary information in a timely enough manner to keep from embarrassing oneself publicly. The staff specialist agreed that yes, dental offices produced a very small proportion of the mercury in the environment, but that from municipality to municipality, dental offices may represent as much as 40 percent of the treatment facility intake. Needless to say, this put a real damper on my righteous indignation.

Amalgam separators have been part of the ADA's best management practices since October 2007. Some estimates of voluntary participation are as high as 50 percent of the targeted offices. The rate of adoption varies from one region to another. The current rate of compliance is very difficult to verify precisely.

In California, there is significant regional difference in the regulatory agencies' interest and involvement in the issue of amalgam separators. In Northern California, where gold mining played a large role in our history and politics, mining has played a large part in the mercury contamination of the Bay Area water system. Because of the heightened awareness and concern for this contamination, the northern part of the state has had more regulatory activity than the southern part. The only agencies that have mandated separators are in Northern California.

Regulation is handled on a regional basis by the local water treatment districts. Our municipality used a "carrot" to entice the dental community to install separators. Last year, we could receive up

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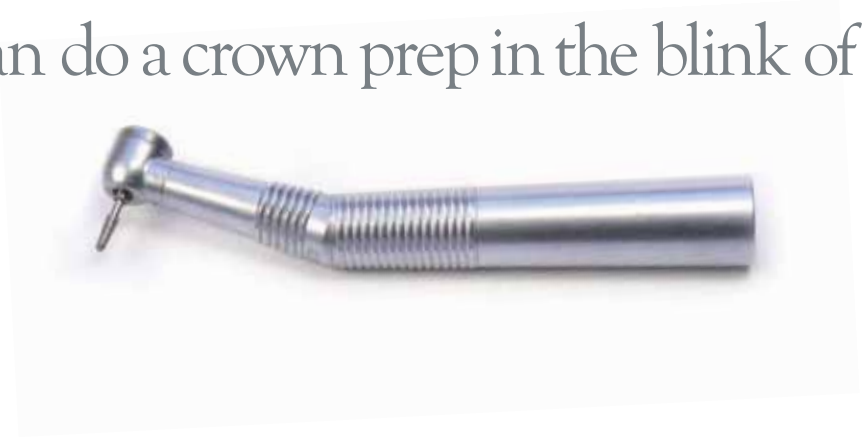

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to $1,000 in the form of a rebate for buying and installing an approved system. The rebate was even available for those who could prove they already had a compliant amalgam separator installed. Within four months, this program produced an increase in the participation rate from 36 percent to 93 percent of the eligible offices. Only one office declined to participate.

On Sept. 27, 2010, the Environmental Protection Agency announced plans to restrict discharges of mercury from dental offices in response to complaints by environmentalists and state regulators over the low voluntary utilization of amalgam separators by dentists.[1] The ADA will be engaging the EPA in a negotiated rule-making process regarding a national pretreatment standard for dental office wastewater. They will be working to make sure that any regulation will comply with ADA's best management practices and grandfather-in those offices that are already complying.

Other groups will be using the regulatory process as a purchase to move their own agendas. "The Mercury Policy Project (MPP) works to promote policies to eliminate mercury uses, reduce the export and trafficking of mercury, and significantly reduce mercury exposures at the local, national, and international levels," according to their website.[2] The MPP sees this regulation process as an opportunity to send a strong message to the United Nations' Environment Programme where the global mercury treaty is being debated right now.[1] That debate could have an impact on regulating the use of amalgam restorations.

A ban on amalgam use could have a substantial short- and long-term impact on the cost of dental care and the increase in untreated disease. Anti-amalgam arguments usually rest on questions of safety or environmental impact. The wholesale adoption of separators in dental offices undercuts the relevance of the environmental argument.

The mercury issue itself is like quicksilver: difficult to contain, scattering, and flowing into other areas. That reminds me of the time one of my uncles brought us a big vial of elemental mercury to play with on the dining room table. And I always thought he liked us. I am lucky to be alive. ■■■■

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1. Vaidyanathan G, Mercury: EPA plans to regulate discharges from dentists' offices, E&E News PM, Sept. 27, 2010. http://www.eenews.net/login. (Member log-in required to access site.)
2. The Mercury Policy Project, mercurypolicy.org/. Accessed April 4, 2011.

The *Journal of the California Dental Association* welcomes letters. *We reserve the right to edit all communications and require that all letters be signed. Letters should discuss an item published in the* Journal *within the past two months or matters of general interest to our readership. Letters must be no more than 500 words and cite no more than five references. No illustrations will be accepted. Letters may be submitted via e-mail to the* Journal *editor-in-chief at kerry.carney@cda.org. By sending the letter to the* Journal*, the author certifies that neither the letter nor one with substantially similar content under the writer's authorship has been published or is being considered for publication elsewhere, and the author acknowledges and agrees that the letter and all rights of the author with regard to the letter become the property of the California Dental Association.*

CDA JOURNAL, VOL 39, Nº6

Reader Provides More Options for Dental Patients Taking Antibiotics

Your editorial, "On the Same Page," (39(4):201-2, April 2011), correctly pointed out the dilemma when a patient's physician recommends antibiotics for dental treatment and you do not agree that should be done. Perhaps the legal strategy would be to refer the patient to a different dentist. An alternative is that after discussing your concerns with the patient and the physician, you require that the patient receive premedication prescriptions from the physician. That seems to be the best choice under the circumstances, although certainly not perfect, if your prime motivation is to help the patient. A custom-made informed consent for this situation would also be valuable.

ARTHUR SCHULTZ, DDS, JD
Manhattan Beach, Calif.

Dental Home Needed Earlier for Children

It is with great frustration that I observe the rapidly growing epidemic, if you will, of childhood caries. The Centers for Disease Control and Prevention documented that between 1994 and 2004 the cavity rate of children aged 2 to 5 grew by 30 percent. Reports continue to show it is worsening at the same rate. In 2000, the U.S. Surgeon General's report indicated the most common chronic infectious disease affecting children today is dental caries, which is five times more common than asthma. Oral disease is the most common reason children miss school.

Twenty-five years ago, the American Academy of Pediatric Dentistry recommended children establish a dental home by their first birthday. Still, the common response parents hear when asking their dentist or physician is to start at age 5, occasionally age 3. My pediatric colleagues and I routinely find and treat cavities in 2 year olds. About 20 of my dental colleagues and I in the Sacramento area alone spend a couple days a month treating children under general anesthesia. The average patient is 3 years old and requires about eight stainless-steel crowns, several pulpotomies, a few fillings, and a couple of extractions. Clearly, establishing a dental home at age 5 or 3 is too late.

Too late is too late. And yet, babies don't come with cavities. The pediatricians across this country, since the U.S. Surgeon General's Report, have been training themselves to do oral health assessments and refer children to the dentist with their first tooth. The infant in the car seat in the corner of the hygienist's room is a new patient seeking a dental home.

For my general dental colleagues I ask these questions: Are you and your staff prepared to counsel these new parents to ensure those new teeth grow into healthy cavity resistant mouths? Can you identify the low-risk infants and toddlers who you want to keep as the nucleus of a cavity-free young population in your growing hygiene department? Can your team counsel effectively to prevent cavities before they begin? Can you identify the high-risk patients who you would rather refer early to a pediatric colleague? Can you identify incipient problems in these very young children and refer to a pediatric colleague for management before they are a major treatment challenge?

Pediatricians begin counseling on "oral health" at the four-month "Well Baby Visit" and refer to a dental home when

the first tooth appears. Does your office have a well-thought and planned response for these new parents in your practice? Your favorite pediatric dental colleague in your area can provide you with all the information you want.

We all know that restorative and surgical intervention only repairs the damage and does nothing to address or reduce the oral diseases for our patients. If we want to truly impact the cavities and periodontal disease of our young patients, we need to start before the disease starts. Oral disease prevention begins with good dietary and hygiene practices before a cariogenic biota develops. The first tooth deserves a dental home.

SCOTT THOMPSON, DDS
Meadow Vista, California

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Matt Mullin

The Duty to Punish

BY DAVID W. CHAMBERS, PHD

The teenager murdered her parents and then threw herself on the mercy of the courts because she was an orphan. Something is morally wrong with this picture. Any society that puts too much distance between breaking the rules and accepting the consequences will be a failure. Most people realize that, but we disagree on how to make it work.

We expect everyone to play by the rules in the game of life. If we know that the rules and penalties are only pretend, we will pretend to follow them just to keep the playing field level. Failure to enforce the rules of society is an ethical failing on society's part. Technically, society reneges on its promises to ensure the conditions for common moral behavior.

Good theory, but why is it so often honored in the breach?

CONTINUES ON 361

Damage Still Evident Among Infrequent Smokers

Yes, even "a little" can kill you.

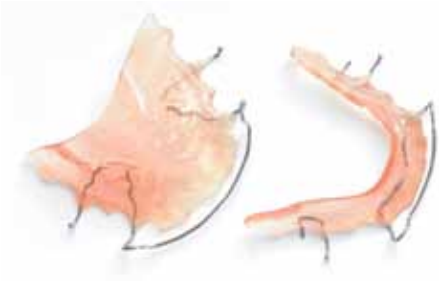
According to a recent issue of the *Harvard Heart Letter,* there is still measurable harm to the body and heart found among "light" or "intermittent" smokers as there is with their "heavy" habit counterparts.

Nearly half of those individuals who smoke a few cigarettes a day or now and then don't consider themselves as smokers, say they can quit anytime, and don't believe smoking poses much risk to their health. Studies state otherwise. Among the hazards with those "light" or "intermittent" smokers include increased risks for:

- Heart disease due to high blood pressure and cholesterol-clogged arteries;
- Premature death from cardiovascular disease;
- Lung, esophageal, stomach, and pancreatic cancer;
- Respiratory tract infections;
- Delayed conception in women and poor sperm function in men; and
- Slower recovery from injury.

There are no formal guidelines to help light and intermittent smokers quit, according to the *Harvard Heart Letter,* and nicotine replacement may work for these types of smokers.

To read the entire article, "Light and Social Smoking Carry Cardiovascular Risks," go to health.harvard.edu/newsletters/Harvard_Heart_Letter/2010/November/light-and-social-smoking-carry-cardiovascular-risks?utm_source=heart&utm_medium=pressrelease&utm_campaign=heart1110.



"We found little written about the kinds of retainers prescribed and how compliant patients are in using them."

MANISH VALIATHAN, BDS, DDS, MSD

Research Conducted on Orthodontia Compliance

Case Western Reserve University School of Dentistry is on the case about retainer usage.

"We found little written about the kinds of retainers prescribed and how compliant patients are in using them," said Case Western Reserve's Manish Valiathan, BDS, DDS, MSD, an assistant professor of orthodontics and a member of the American Board of Orthodontics.

Although retainers are commonplace in orthodontic practices, information about them is harder to come by. So, Valiathan and fellow researchers initiated three studies relating to retainer usage: how individuals use them, what types are prescribed, and the results when patients don't comply.

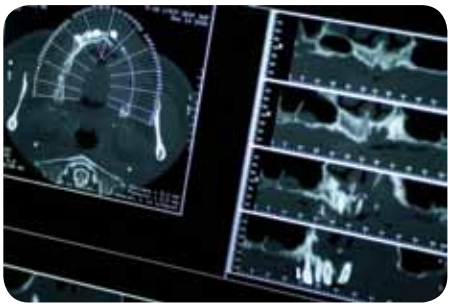
Of the 2,000 surveys that were randomly sent to orthodontists across the nation, 658 practitioners responded. Nearly 59 percent said they prescribed removable retainers and an estimated 40 percent opted for fixed lingual retainers that typically are worn for life.

The majority of orthodontists said they required wearing removable retainers full-time for the first nine months and then part-time afterward. They also advocated part-time retainer use throughout life, according to a news release.

Without retainers, Valiathan said, specific prior conditions may return but that definitive research does not exist as to what conditions require ongoing retainer use. More evidence is needed.

In another survey study of 1,200 patients on patient compliance two years after prescribing retainers, 36 percent responded to the researchers' questions regarding the type of retainer used, age, gender, length of time since braces were removed, and the hours per day and night the retainer is worn, according to the news release.

Sixty percent wore retainers more than 10 hours a day in the first three months and 69 percent wore them every night. By the time retainer users reached 19 to 24 months, 19 percent were not wearing retainers but 81 percent were — even if it was only one night a week.



New Use of CT Scans Help in Surgical Recovery

Computerized tomography is proving beneficial in oral surgery recovery. In a recent issue of the *Journal of Oral Implantology,* authors related two cases in which CT scanning, prior to and up to one year following the patients' oral surgery, was conducted.

Using the scans, oral and maxillofacial surgeons could measure the healing process following surgery that reconstructs the jaw and prepares it for future implants. In one case, CT scanning evaluated the successfulness of the implant and also checked the condition of the graft donor sites. In the second case, using these scans aided in determining the patients' readiness to receive three implants six months after the initial surgery, evaluating new bone formation, the condition of the sinus membrane, and radiographic change, according to a news release.

Although radiolucency was noticeable in the area where the two halves of the jawbone meet, the size of the defect was visibly reduced at 5½ months after surgery in one case, and one year postsurgery in another case.

For the two patients, healing was uneventful. What's more, past the one-year point of this study, CT scanning also could be used to evaluate progress of remineralization and other long-term changes.

To read the full article, "Computerized Tomographic Evaluation of Symphyseal Donor Sites Used in the Reconstruction of the Posterior Maxilla: a Case Report of Two Patients," go to joionline.org/doi/full/10.1563/AAID-JOI-D-09-00094.1.

ADA E-books Now Offered for Nook, Kindle

Three of the most popular dental practice management publications from the American Dental Association have hit the Kindle and the Nook.

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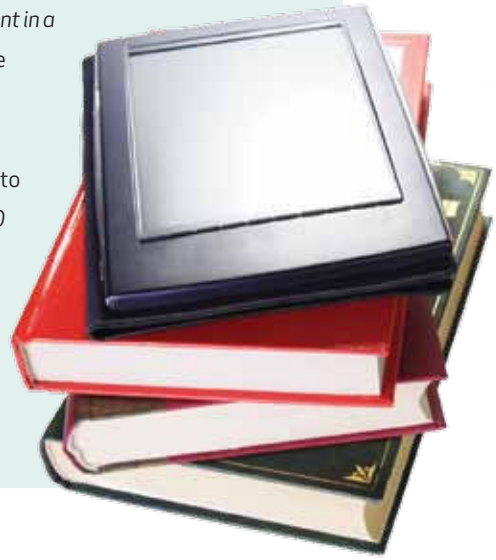
All three books are available in hard copy at adacatalog.org or (800) 947-4746. The cost of *CEO Crash Course* and *Terminating Employment in a Dental Office* is $39.95 each for ADA members, $59.95 each for non-ADA members. *Starting Your Dental Practice* in hard copy is $49.95 for members, $74.95 for nonmembers.



PUNISH, CONTINUED FROM 359

Punishment has a cost that must be borne. Somebody should do something about this. It has always been fashionable for legislators to pass laws to demonstrate their moral concern and then underfund enforcement. "People should just do what is right" is the wimpiest of false ethics imaginable. "I talked to them about it" does not get much work done either.

The fact that we get as much morality as we can afford is a doubly difficult problem. The benefit of ethical behavior is to society generally, the cost is to specific individuals or groups. We often seek to pass the cost of punishment onto others. The hangman of old wore a mask and was excluded from society.

Individuals can actually filch personal prestige by short-circuiting punishment. In every culture, granting clemency is a sign of high social status. Only the governor can commute the death sentence. When we say "I am letting it go this time," we elevate our own status. In surveys of cheating in colleagues, the most common response to detected cheating is for faculty members to "deal with the problem on a personal and individual basis."

In addition to wanting to be judge and jury by personally dispensing mercy, we like to be legislators as well. When we selectively wink at punishment, we are changing the rules. It is perfectly appropriate in a democracy to work to change the rules, say the laws requiring reporting of suspected child abuse. It is not appropriate to ignore the rules and expect to be exempt for the consequences.

Finally, society is lousy at matching punishment to unwanted behavior. Extreme forms of punishment have no more effect on behavior than do barely effective ones. One of my favorite cartoons shows the hangman placing a noose on the criminal's head and saying, "I hope this teaches you a lesson." On the other side, penalties that society is not willing to enforce are useless.

The nub:

1. In ethics, don't expect to get anything you are not willing to pay for.
2. Don't expect to control other's ethical behavior by paying the costs with others' resources.
3. Beware private justice.

David W. Chambers, PhD, is professor of dental education, Arthur A. Dugoni School of Dentistry, San Francisco, and editor of the Journal of the American College of Dentists.

Journal Wins Maggie Award

The *Journal of the California Dental Association* has been honored with a prestigious Maggie Award for the sixth time in seven years by the Western Publications Association.

This is the *Journal*'s first Maggie Award for an issue produced under the direction of current Editor Kerry K. Carney, DDS.

The September issue on Perinatal Care in the Office was honored for excellence in the trade publication category of Medical, Dental and Related Services for publications produced in 2010.

Lindsey Robinson, DDS, served as the guest editor for the issue. Her efforts furthered work initiated by the CDA Foundation when it convened the Perinatal Oral Health Guidelines project.

The *Journal* is available online at cda.org/publications/journal_of_the_california_dental_association

UPCOMING MEETINGS

2011	
June 9-12	Cleft 2011 International Cleft Lip and Palate Conference, San Francisco, cleft2011icpf.org.
June 16–18	ADA New Dentist Conference, Chicago, 800-621-8099, ext. 2779, ada.org/goto/newdent.
July 28-31	AGD Annual Meeting and Exhibits, San Diego (888) AGD-DENT or agd.org/sandiego.
Sept. 12-17	American Association of Oral and Maxillofacial Surgeons, Philadelphia, aaoms.org.
Sept. 14-17	FDI Annual World Dental Congress, Mexico City, www.fdicongress.org. Please also view this related video: http://www.youtube.com/watch?v=3N4okaVMYhs.
Sept. 22–24	*CDA Presents the Art and Science of Dentistry*, San Francisco, 800-CDA-SMILE (232-7645), cdapresents.com.
Oct. 10-13	ADA 152nd Annual Session, Las Vegas, ada.org.
Nov. 6–12	United States Dental Tennis Association, Palm Desert, Calif., dentaltennis.org.
Nov. 6–12	United States Dental Tennis Association, Palm Desert, Calif., dentaltennis.org.
2012	
March 29–April 1	CSPD/WSPD Annual Meeting, Portland, Ore., drrstewart@aol.com.

To have an event included on this list of nonprofit association continuing education meetings, please send the information to Upcoming Meetings, CDA Journal, 1201 K St., 16th Floor, Sacramento, CA 95814 or fax the information to 916-554-5962.

Enzyme Could Rival Toothbrushes, Dental Floss

Two enzymes of an oral bacteria have shown to inhibit the formation of plaque. Researchers from Japan demonstrated that the bacterium, *Streptococcus salivarius*, a non-biofilm forming and otherwise harmless inhabitant of the human mouth, reduced the development of dental biofilms.

"FruA [one of the enzymes] may be useful for prevention of dental caries," said author Hidenobu Senpuku, DDS, PhD, National Institute of Infectious Diseases, Tokyo. "The activity of the inhibitors was elevated in the presence of sucrose, and the inhibitory effects were dependent on the sucrose concentration in the biofilm formation assay medium."

"We show that FruA produced by *S. salivarius* inhibited *S. mutans* biofilm formation completely in the in vitro assay supplemented with sucrose," the researchers wrote in an article, "Inhibition of Streptococcus Mutans Biofilm Formation by *Streptococcus salivarius* FruA," that was published recently in an issue of *Applied and Environmental Microbiology*. *S. salivarius* is the primary species of bacteria inhabiting the mouth, according to the report.

FruA, the authors suggested, may actually regulate microbial pathogenicity in the oral cavity.

Periscope offers synopses of current findings in dental research, technology, and related fields.

IMAGING

SANJAY M. MALLYA, BDS, MDS, PHD, AND SOTIRIOS TETRADIS, DDS, PHD

Predicting IAN Damage

Susarla SM, Sidhu HK, et al, Does computed tomographic assessment of inferior alveolar canal cortical integrity predict nerve exposure during third molar surgery? *J Oral Maxillofac Surg* 68(6):1296-303, 2010.

THE CLINICAL PROBLEM: Injury to the inferior alveolar nerve (IAN) is a potential complication of third molar extraction. Panoramic radiographic findings may suggest an increased risk but are poor predictors of IAN injury. Frequently, patients determined to be at an increased risk for IAN damage are evaluated with computed tomography (CT). However, there is a lack of standard criteria that could help predict the probability of nerve injury.

AIM: This study evaluated whether the CT-demonstrated integrity of the cortical outline of the inferior alveolar canal (IAC) could be used to predict inferior alveolar nerve (IAN) exposure during third molar extraction.

METHOD: This retrospective study examined clinical and radiographic records of 51 patients who had been previously evaluated and managed for impacted mandibular third molar removal. All patients had been identified as high risk for mandibular nerve exposure, based on panoramic radiographic findings, and were evaluated preoperatively by maxillofacial CT. A total of 80 third molars were evaluated. Two observers evaluated coronal reconstructions of the CT examination for integrity of the cortical outline of the IAC. Defects in the cortical outline were measured. The presence and size of these defects were correlated with status of IAN exposure, as determined by direct visualization during or immediately after removal of the third molar.

RESULTS: Interruption of the IAC cortex was associated with a 13-fold higher risk for intraoperative IAN exposure. As a diagnostic predictor for IAN exposure, interruption of the IAN cortex was highly sensitive but was not very specific. However, when only interruptions of ≥3 mm were considered, the specificity was improved and yielded a positive predictive value of 79 percent.

CONCLUSION: Interruption of the IAC cortex ≥ 3mm in length is a strong predictor for IAN exposure during third molar removal.

IMPLANTS

RICHARD T. KAO, DDS, PHD, AND DAVID W. RICHARDS, DDS, PHD

Implant/Abutment Mismatching and Bone Loss

Canullo L, Fedele GR, et al, Platform switching and marginal bone-level alterations: the results of a randomized-controlled trial. *Clin Oral Impl Res* 21(1):115-21, January 2010.

AIM: Through a randomized-controlled trial, the marginal bone level alteration with different implant/abutment mismatching was examined.

METHOD: Four equal groups of 20 implants with 3.8 (control), 4.3 (test grp[1]), 4.8 (test grp[2]), and 5.5 mm (test grp[3]) diameter were randomly placed in posterior maxilla. After three months, 3.8 mm diameter abutments were placed on all implants. Radiographic evaluation indicated a mean bone loss of 0.99 mm for control group, 0.82 mm for test grp(1), and 0.56 mm test grp(3). A similar pattern was found after 33 months.

CONCLUSION: This study suggests marginal bone loss can be minimized by the extent of implant/abutment mismatching.

CLINICAL RELEVANCE: This clinical trial gives support to the platform-switching concept that using a smaller diameter abutment compared to the implant platform may result in less marginal bone loss.

CDA JOURNAL, VOL 39, Nº6

PERIODONTICS

GERALD I. DRURY, DDS

Free Gingival Grafting Prevents Recession

Agudio G, Nieri M, et al, Periodontal conditions of sites treated with gingival-augmentation surgery compared to untreated contralateral homologous sites: a 10- to 27-year long-term study. *J Periodontol* 80(9):1399-405, September 2009

BACKGROUND: This study compared the periodontal conditions of sites treated with gingival-augmentation procedures to untreated homologous contralateral sites over 10- to 27-years.

METHODS: A cohort of 55 patients took part in this study. Two sites were included: one surgically treated site and a contralateral homologous untreated control. Variables were recorded for each patient at baseline: one year after surgery and at the end of the follow-up period. Gingival-augmentation procedures were performed on the test areas. A marginal-free gingival graft (MFGG) was applied when the existing free gingiva was judged as very thin. A submarginal-free gingival graft (SMFGG) was applied when it was thick.

RESULTS: A total of 55 teeth were treated with SMFGG, and 18 teeth were treated with MFGG. At baseline, all 55 SMFGG test sites had gingival margin recession. In the control group, 30 of 55 sites had gingival recession. Keratinized tissue width was 1.0 mm in the test group and 2.6 mm in controls. At one year, roots were completely covered in 14 test sites and 18 control sites. KT was 5.3 mm in the test group and 2.6 mm in controls. At the end of the study, 49 of the 55 test sites had a reduction on recession and 33 had complete root coverage; only one site had increased recession; and all others remained stable. KT was 4.1 mm at test and 2.1 mm at control sites. At baseline, all 18 MFGG test sites had gingival recession and all but four controls had recession. At the end of the follow-up period, 14 test sites showed recession reduction, seven sites achieved complete root coverage and four remained stable at year 1 levels. KT increased from 1.0 mm in test sites at baseline, to 5.1 mm at year 1, and 4.8 mm at the end of the follow-up period. Control KT was 2.6 mm at baseline, 2.4 mm at year 1, and 1.9 mm at the end of the follow-up period.

CONCLUSIONS: This long-term split-mouth study showed that gingival-augmentation surgery improved the gingival conditions of sites with gingival recessions and minimal or absent attached gingiva at baseline. The surgery was meant to increase the width of keratinized gingiva. The untreated groups showed a progressive deterioration with an increase in gingival recession depth. The sites treated with gingival augmentation surgery showed a tendency for coronal displacement of the gingival margin with a reduction in recession. The contralateral untreated sites showed a tendency for apical displacement of the gingival margin with an increase in the existing recessions.

ORAL AND MAXILLOFACIAL SURGERY

D.D.R. YAMASHITA, DDS

Earlier Grafting Reduced Operative/ Anesthesia Time and Associated Morbidity

Miller LL, Kauffmann D, et al, Retrospective review of 99 patients with secondary alveolar cleft repair. *J Oral Maxillofacial Surg* 68(6):1283-9; June 2010.

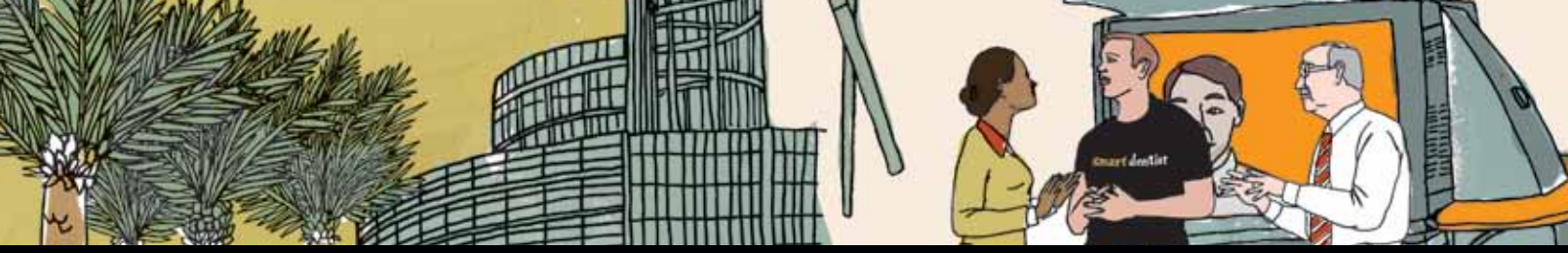
AIM: To evaluate the protocol and technique used in a large population of patients with cleft lip and palate when secondary grafting is performed during the early mixed dentition stage as determined by the eruption of the maxillary central incisor rather than strict age criteria.

METHODS: A retrospective chart review of 99 patients undergoing secondary cleft bone grafting at a single institution over a seven-year period from 2000-2008. Patients were assigned to one of two groups based on age: group 1, aged 6-8 (n=61) and group 2, 9-years and older (n=38). All patients underwent extraction of retained primary teeth adjacent to the graft site at least four weeks prior to placement of bone graft harvested from the anterior iliac crest. Data from each group was compared to determine the presence of statistically significant differences in operative time, length of hospital stay, follow-up period, complication rate, as well as the distribution of gender, race, and cleft site between groups 1 and 2.

RESULTS: There were no statistically significant differences in the distribution of gender, race, or cleft site between groups. The average operative time for groups 1 and 2 was 86 and 103 minutes, respectively, which was statistically significant at $P<0.05$. The difference in length of stay (x=1.06 days), follow-up period (range=one week to 52 months), and complication rates (9.8 percent for group 1 versus 13.2 percent for group 2) were not statistically significant between groups at $P<0.05$.

CONCLUSIONS: Secondary cleft repair with iliac crest bone graft completed in the early mixed dentition period at or before the eruption of the maxillary central incisors provides adequate bone support for the future dentition and decreases operative times with comparable postoperative outcomes to those achieved when surgery is delayed until 25 percent of the permanent canine root is formed.

CLINICAL RELEVANCE: Secondary cleft repair has traditionally been recommended at the 9- to 11-year age range or before the eruption of the permanent canines. Earlier grafting may be indicated to prevent eruption of the central or lateral incisors into a residual cleft or the collapse of alveolar ridge segments, which may jeopardize the periodontal support of the central and lateral incisors. The combination of earlier cleft repair with minimally invasive iliac crest bone harvest would benefit patients through reduced operative/anesthesia time and associated morbidity.



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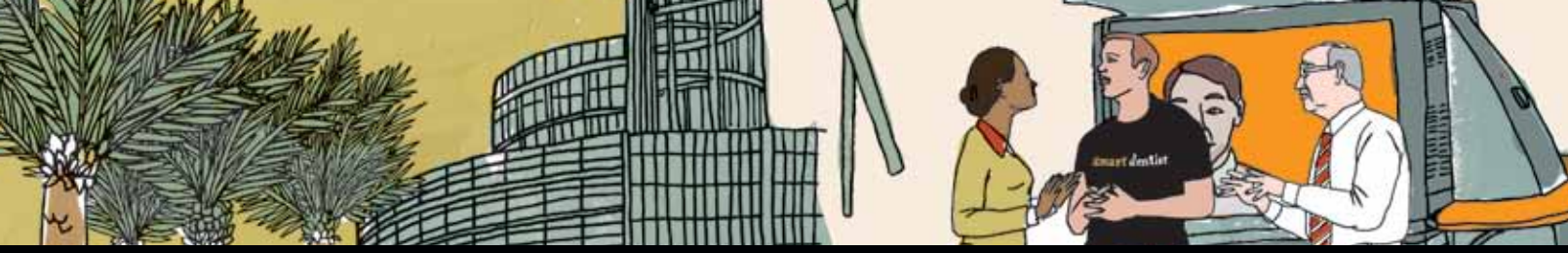

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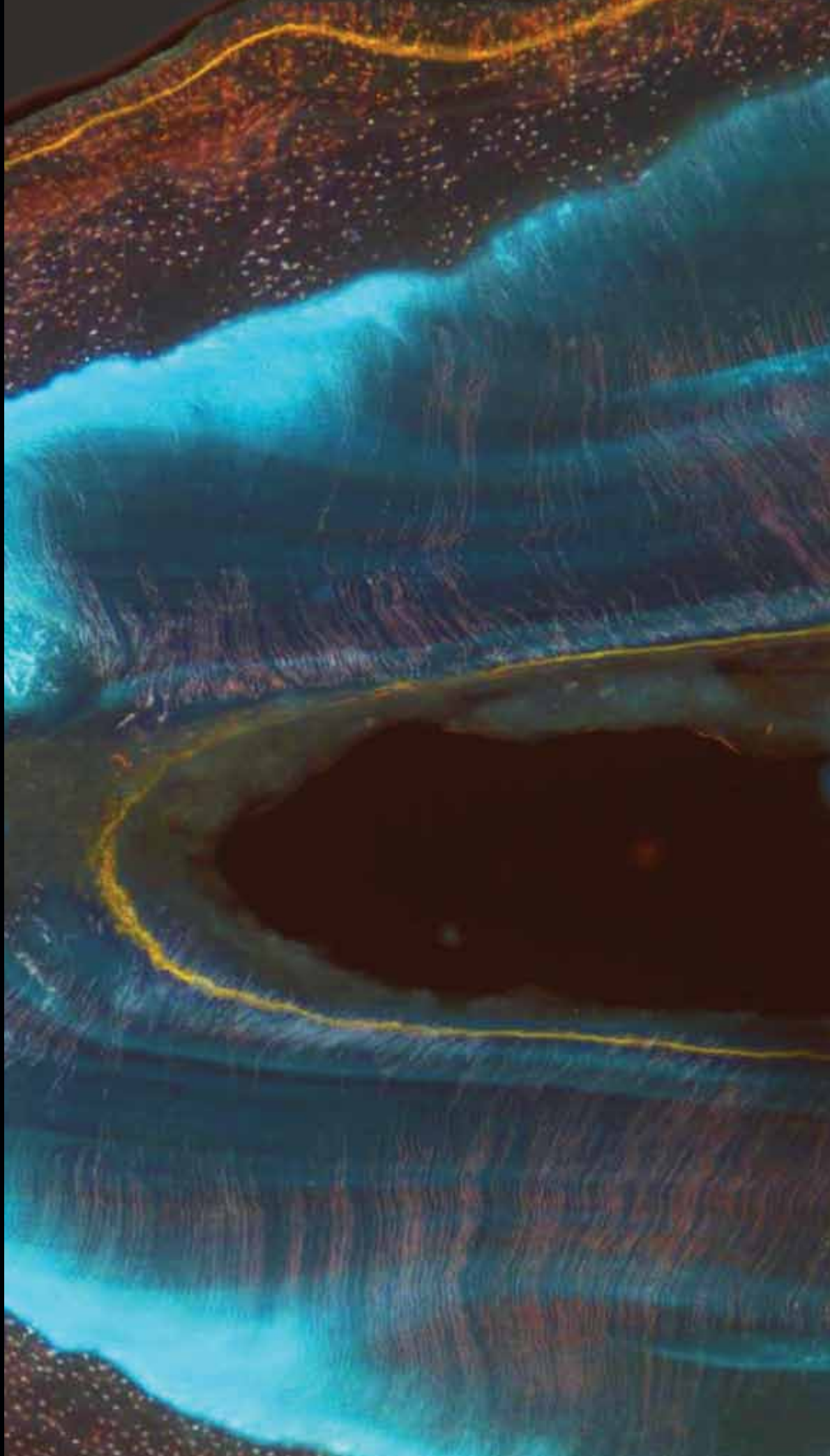

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CDA JOURNAL, VOL 39, Nº6

Aggressive Periodontitis

KIAN KAR, DDS, MS

GUEST EDITOR

Kian Kar, DDS, MS, is an associate professor of Clinical Dentistry, and clinical director of Advanced Periodontology, Ostrow School of Dentistry, University of Southern California. He also is a diplomate, American Board of Periodontology, and in private practice limited to periodontics and dental implant surgery in Mission Viejo, Calif.

Periodontal disease continues to be one of the most common oral infections among adults. According to new research published in the *Journal of Dental Research* from the Centers for Disease Control and Prevention and the American Academy of Periodontology, the prevalence of periodontal disease in the United States may have been underestimated by as much as 50 percent than originally projected. It is likely that many of these patients developed periodontal diseases as adolescents or young adults who were nevertheless diagnosed or treated until later in life. These types of periodontal infections present common features that are classified as aggressive periodontitis. The pathogenesis of these types of periodontitis continuous to be investigated and has a complex correlation to local factors such as: specific bacterial pathogens, viruses, pathogenic immuno-modulation, host-parasite interactions and individual immuno-susceptibility.

Additionally, systemic cofactors and genetic influences may further complicate this local host-parasite interaction. Often, advanced periodontal defects seen amongst middle-aged and older individuals may be the scars of a past history of an early onset periodontal infection (aggressive periodontitis) that is masked or transformed into chronic periodontitis later in life. Identifying these patients is a diagnostic challenge, especially when historic data, as is often the case, is not available. The goals of this issue of the *Journal* are to provide historical background, therapeutic guidelines, and preventive insight concerning aggressive periodontitis.

Frydman and Simonian will provide the historical perspective in understanding aggressive periodontitis as a disease entity. In the article by Nowzari, the issue of early-in-life periodontal infection, its socioeconomical and psychological impacts amongst young population of patients is addressed. The importance of awareness about global concerns for periodontal disease and the necessity in providing educational and preventive resources, also are discussed in an educational documentary by Nowzari titled, *The Enemy of The Smile — AA — (An Ancient Bacterium)*, which is available to view on the Internet Movie Database website. In the article by Slots, the potential influence of viruses in pathogenesis of aggressive periodontitis is presented. The influence of viruses is an emerging concept that, when better understood, provides useful insight in pathogenesis as well as diagnostic and therapeutic approaches in the treatment of patients with aggressive periodontitis.

Moreover, Simonian, Nowzari, and I provide literature review and clinical guidelines in treatment of individuals affected by aggressive periodontitis. Considering the complexity in diagnosis and treatment with an aim to individualized therapy a "dynamic therapeutic approach" in management of individuals affected by this disease is discussed. Additionally, Chee reviews maintenance and peri-implant tissue concerns when treating individuals with a history of aggressive periodontitis.

It is my hope that this issue will provide further awareness and insight among dental professionals about an often undetected or misdiagnosed periodontal disease entity. Understanding the complexity in management and treatment of individuals with aggressive periodontitis would pave a path in collaborative efforts among restorative dentists, periodontists, and other specialties, to provide appropriate periodontal and peri-implant care for these individuals. ■■■■

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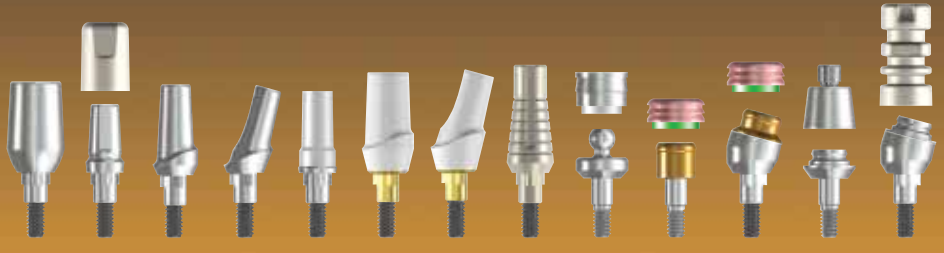

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CDA JOURNAL, VOL 39, Nº6

Aggressive Periodontitis: the Historic Quest for Understanding

ALON FRYDMAN, DDS, AND KRIKOR SIMONIAN, DDS

ABSTRACT Aggressive periodontitis has had many names as well as theories behind its etiology. The current paper looks to investigate this history and show the evolution of both nomenclature and understanding. Going beyond academic debates, emerging trends and conventions are also examined and placed into a clinical perspective. The aim of the paper is to recognize the origins of the elusive disease in order to form a concrete understanding of this multifactorial phenomenon.

AUTHORS

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Krikor Simonian, DDS, is a diplomate, American Board of Periodontology, and a clinical assistant professor, Advanced Periodontology, Herman Ostrow School of Dentistry, University of Southern California, Los Angeles. He maintains a private practice limited to periodontics and dental implants in Pasadena, Calif.

DISCLOSURE

This work is not supported by any grants.

Between a disease and its characterization there is an ocean of interpretation. That interpretation is biased by the tools with which the interpreter identifies, observes, collects, and infers data. Ultimately, recognizing the limitations of these tools, the judicious observer assembles his body of summations and constructs a theory. Throughout the history of periodontology, there have been advancements and trends in science that have affected the terminology, understanding, and approach to the aggressive forms of the disease. The end result for the clinician is a collection of disease classifications and diagnosis. The static that exists between the systems of classification and diagnosis is academic at best, yet the influence these classification systems have upon diagnosis goes without saying.

The clinician is faced with the task of organizing a wide array of information, choosing what is important, and delivering the best estimate as a diagnosis. That diagnosis, however, is then funneled back into an accepted treatment protocol based on research anchored in disease classification. As the two are intertwined, it is important that the inception of the classification systems and their history are understood. The evolution of therapy not only parallels that of classification and diagnosis, but is the common thread between the two. At some point the debate ends and the patient receives treatment. The following review investigates aggressive periodontitis etiology, clinical presentation, and its related history.

Historical Survey

There was little understanding of periodontal diseases prior to 1920. Often, dentists used their clinical observations to theorize etiology and proper treatment, or they followed the opinion of

a seasoned speaker. Perhaps the most prominent of the latter group was John M. Riggs, who lectured extensively on periodontal disease. His influence was so widespread that dentists often referred to periodontitis as "Riggs' disease."[1] Still, clinicians observed different types of periodontal disease. In 1879, C.G. Davis distinguished between periodontal destruction due to "lime deposits" and "Riggs' disease," which he described as "necrosed alveolus" without any visible mechanical irritant.[2] A few years later, when he published his classification of periodontal diseases, G.V. Black distinguished "phagedenic pericementitis," with little presence of calculus deposits and irregular bone destruction, from "calcic inflammation of the peridental membrane," which he described as presenting with a slower and generalized form of bone loss with heavy calculus presence.[3]

The scientific basis of periodontology began to emerge after the 1920s. With histopathological studies, Gottlieb first described what would be later called juvenile periodontitis. He reported it as "diffuse atrophy of the alveolar bone." Later, he hypothesized that the cause of the disease was a defect in continuous cementum development, which he called "deep cementopathia." He saw the attachment and tooth loss as an attempt by the body to eliminate the defective structure out of its system.[4] Gottlieb's influence was widespread and a degenerative form of periodontal disease was accepted as distinct from inflammatory disease.

Studying an autopsy case study in 1942, Orban and Weinmann fortified the degenerative disease hypothesis, while adding that a secondary inflammatory process appears later and is often the symptom commonly first observed by clinicians. They termed the disease "periodontosis."[5]

While recognizing that a distinct disease entity other than the periodontitis commonly seen in adults may occur in younger patients, the 1966 World Workshop in Periodontics, citing there was no scientific evidence of a degenerative disease entity, recommended that the term "periodontosis" be abandoned.[6]

In 1971, Baer redefined "periodontosis" as rapid bone loss around more than one permanent tooth "in an otherwise healthy adolescent." He further described two forms of the disease: localized, which affected the incisors and first molars; and generalized, which involved most of the dentition. He also stressed that "the amount of destruction manifested is not commensurate with the amounts of local irritants present."[7] The term "juvenile periodontitis," which was introduced by Butler in 1969, soon replaced "periodontosis." Scientific research in the '60s and '70s, starting with the experimental gingivitis studies by Harald Loe and his colleagues, followed by the evidence of microbial specificity at periodontitis sites by Newman, Socransky, and Slots, finally substantiated juvenile periodontitis as an infection, and the degenerative theory was laid to rest.[8-11] It was also during this period that evidence was presented that juvenile periodontitis patients had defective neutrophil function.[12]

At the 1989 World Workshop in Clinical Periodontics, the classification "early onset periodontitis," was introduced to differentiate from "adult periodontitis," emphasizing both the age of onset and progression rate of the disease.[13] "Prepubertal," "juvenile" and "rapidly progressive" were included under this classification as distinct disease categories. However, this classification faced criticism because of the difficulty in defining or documenting rate of disease

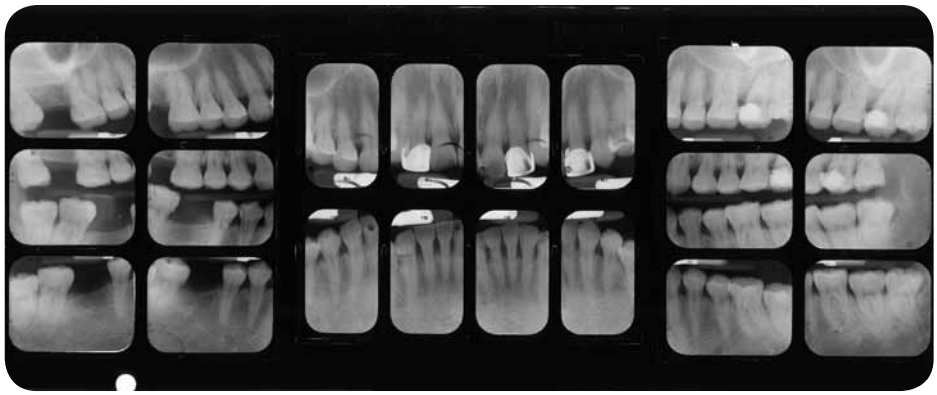

FIGURE 1. Typical appearance of chronic periodontitis. Note the pattern of bone loss related to the accumulation of radiographic calculus in the mandibular anterior region.

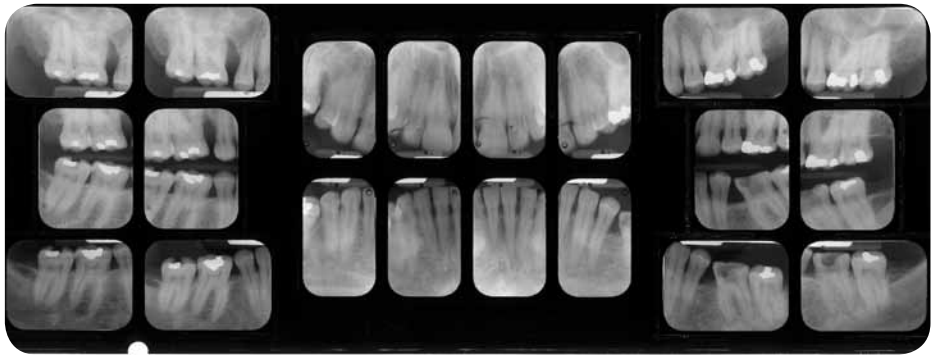

FIGURE 2. Localized defect on the mesial of No. 3 in this case of localized aggressive periodontitis along with missing first molar No. 19.

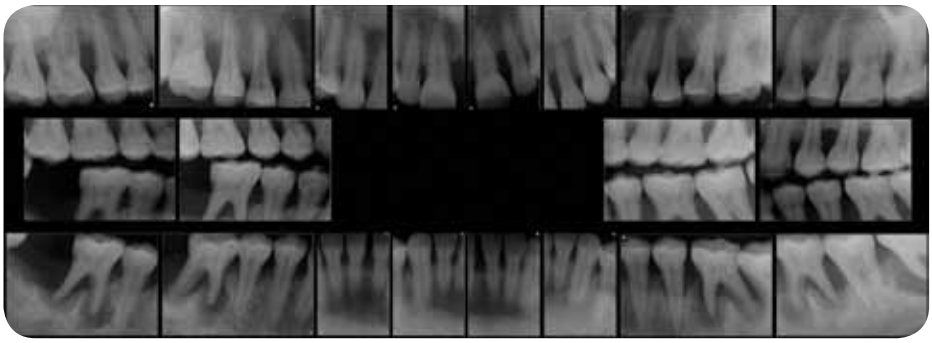

FIGURE 3. Note the pattern of bone loss localized to the first molars while adjacent premolars are relatively unaffected. Tooth loss and super-eruption are also present while there is little calculus to explain the severity of disease. Also notable is the minimal amount of carious involvement.

progression, and, also the fact that a clear distinction could not be made between "rapidly progressive" and "generalized juvenile," among other concerns.[1,14]

The "1999 International Workshop for a Classification of Periodontal Diseases and Conditions" attempted to addresses the problems of the 1989 classifications. It eliminated using patient age and rate of disease progression for classification of periodontal diseases. Due to the inability for objective and fixed designations, terms such as "adult" and "juvenile" were seen as inappropriate descriptions for diagnostic categories. Therefore, the classifications "aggressive" and "chronic" periodontitis were adopted.[15]

Current Understanding: Emerging Science and Trends

Tests and Their Utility

Current definition and understanding of aggressive periodontitis are complicated due to the lack of a single diagnostic test that can deliver a diagnosis on its own. Investigators studying risk factors have encountered difficulty in distinguishing true risk factors from associated cofactors.[21] Accepted risk factors have not been studied with long-term longitudinal studies. As Garcia pointed out, "Simply looking at risk factors without understanding their effect under the influence of time, we may arrive at a false or even exaggerated representation."[21] With a disease that presents early in life, research results may not have enough time to accurately gauge the cause of aggressive disease. Still, the American Academy of Periodontology statement on risk assessment argues for attention when it comes to smoking, poor diabetic status, inadequate home care, age, gender, extent, and severity of alveolar bone loss, as well as proportion of pocket probing depths >6 mm.[22] With aggressive periodontitis, the utilization of salivary tests for immunoglobulins and glycoproteins all show elevated levels paralleling the inflamed tissues.[23,24]

However, the practitioner is challenged with utility of these tests outside of research purposes. The scientific push to investigate for specific bacteria classically associated with aggressive periodontitis has been met with questions of utility due to variability in sensitivity detection methods.[25] Currently, the trend among clinicians is to use the results of microbial testing, as studies often show its benefit in both treatment planning and overall outcome.[26] Most importantly, microbial sampling has been used in aggressive cases to aid the practitioner in deciding upon the type of antibiotic to use when the decision has been made to use them as adjuncts to therapy as well as a method of follow up after the fact.[27,28]

Genetic Influence

With the emergence and advancement in medical genetics, dentistry, also attempted to discover a new understanding in the manifestation of aggressive periodontitis. Investigations into genetic predisposition to periodontal disease have looked at specific markers with some degree of success and now there is a growing trend to support such a case in aggressive periodontitis.[29-31] Genetic sequences for cytokine production have been the focus of genetic investigations into periodontitis susceptibility.[32] Genetic disorders leading to systemic challenges are often excluded from consideration as aggressive periodontitis because of the definition itself.[15] However, recognizing that kinks in the immune system may not manifest as systemic conditions, genetics can be key in understanding the manifestation of aggressive periodontitis. Specifically, genetic evidence for predisposing factors in aggressive periodontitis have shown association with neutrophil dysfunction, immune cascade overreaction to bacterial endotoxin, and a connective tissue turnover imbalance.[33-38] Overall, the current understanding seems to show that genetics influences the patient's immune system response and, hence, the ability to fight rather than contract disease.

Clinical Features

Clinical recognition of aggressive periodontitis has classically been through comparison to chronic periodontitis, as disease rate is one of the most distinct differences between the two. Chronic periodontitis is generally considered to have a slow and steady pace (**FIGURE 1**), while the aggressive forms have a more rapid rate of progression[7] (**FIGURE 2**). Most studies suggest a rate of 0.2 mm of attachment loss per year in chronic periodontitis cases while the rate of attachment loss for aggressive periodontitis is greater.[7,39-42] This pattern is one of the cornerstones of understanding aggressive periodontitis that has been constant despite the changes in nomenclature.

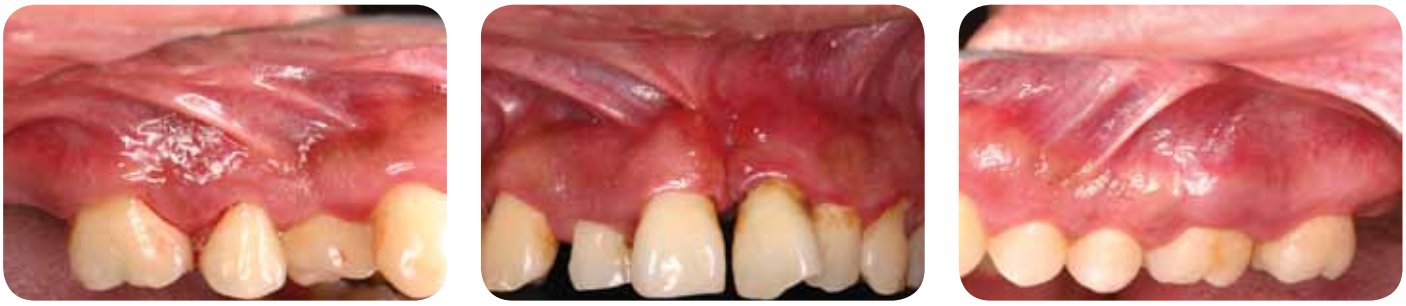

FIGURE 4A.

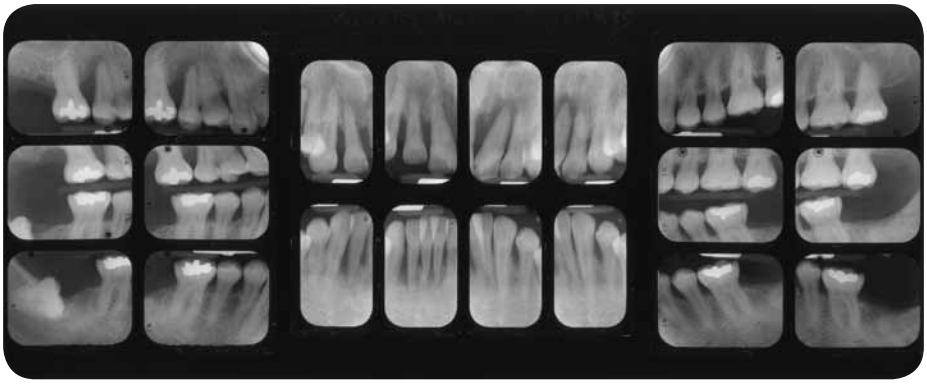

FIGURE 4B.

FIGURES 4A-4B. Minimal plaque biofilm is present clinically (**FIGURE 4A**) relative to the amount of bone destruction seen around the maxillary anterior as well as No. 4 in Figure 4b.

As the current theory for aggressive periodontitis rests on the assumption of a complex infection, there is no single type or group of bacteria that warrant a specific diagnosis. However, there is evidence that certain bacterial profiles are associated with greater amounts of disease, while some other profiles seem to be associated more with health. The bacteria investigated mostly have been *Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis,* and some other exogenous bacteria. Often, however, the profiles seen in disease overlap with those seen in healthy subjects, lending further indication of a multifactorial disease.

Radiographic Presentation

The radiographic appearance of aggressive periodontitis, whether in its localized or generalized form, can be distinctive. Usually, in the localized form, vertical bone loss is seen around the first molars, as well as the incisors[43,44] (**FIGURE 3**). The breakdown can also be limited to other teeth. Radiographic appearance alone may often not present with the pathognomonic signs expected and, therefore, should not be depended upon solely. Other radiographic findings that hint at the possibility of aggressive periodontitis include a low caries incidence.[44-47] As with other radiographic signs, this holds greater value when put into context of the entire radiographic presentation.

Theories offering explanation for the specific presentation of localized bone loss usually revolve around the eruption pattern of the teeth. Case reports of aggressive periodontitis affecting the primary dentition do support this theory, leaving a possible mechanism by which the first permanent teeth to erupt to be those most affected.[48-50] As the primary dentition interacts with the earlier permanent teeth, the first molars and incisors will be exposed first, allowing for the pattern of destruction. Despite the amount of damage seen in aggressive cases, often absent is the concomitant presence of plaque.[7,51,52] (**FIGURES 4A-4B**). Low plaque levels are often seen in aggressive periodontitis patients, and that presentation specifically is often what aids in diagnosis. The biofilm itself can vary in its quality from chronic periodontitis as is seen with the presence of more gram-negative forms of bacteria as well as exogenous species.[11,52-54]

Viral Involvement

The newest frontier in the understanding of aggressive periodontitis is the possibility of viral involvement. Gaining support is the possibility of synergistic co-destruction of the periodontium in aggressive cases.[55,56] Viruses present in high numbers throughout the world population, such as cytomegalovirus and herpes simplex, seem to be the viruses most involved in aggressive periodontitis cases.[57-60] Herpesviruses have been shown to have an inclination for cellular and tissue-tropism, capacity for immune evasion, dormancy, and reactivation. These phenomenons may explain the site-specific destruction as well as latency periods of attachment loss seen in aggressive periodontitis.[61,62]

When considering the tissue tropism that viruses exhibit, it is possible to deduce that this may be one of the reasons for the traditional patterns of destruction seen in localized aggressive periodontitis. Further, the evidence for co-destruction offers some explanation as to the amount of destruction seen in these cases.

TABLE 1

Classification and Nomenclature

Classification	Year	Author	Theory
Chronic suppurative pericementitis	1914	G.V. Black[16]	Bone destruction/ absence of calculus
Periodontosis	1920	Gottlieb[17]	Alveolar atrophy/ cemental pathosis
Juvenile periodontitis	1969	Butler[18]	Localized bone loss
Precocious periodontitis	1977	Sugarman[19]	Age
Rapidly progressive periodontitis	1979	Lavine[20]	Age and rate
Early onset periodontitis	1989	AAP Workshop 1989[13]	Age
Aggressive periodontitis	1999	Armitage[15]	Age independent

Conclusion

Recognizing aggressive periodontitis can be as elusive as understanding it. The disease has had an evolution not only in the way we classify it, yet also in the way we assume that it works (**TABLE 1**). Overall, the standing theory points to a multifactorial disease that gains influence from oral and opportunistic bacteria, viruses, the immune system, genetic dispositions, systemic interactions, familial exposure, and hormonal influences. Despite sharing many of the same etiologic factors as chronic periodontitis, aggressive periodontitis results in a different manifestation that is often contrasted in order to diagnose it. ■■■■

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Timothy G. Giroux DDS/Broker

ASK THE BROKER

Question:

I have a practice with an associate, a hygienist and several key employees. Should I have something in writing about their employment that would facilitate a sale or transition?

Fred J, DDS

Even though we have an attorney on staff, we are not lawyers and you need to consult your attorney on these matters. The law in different states also varies. However, I will expound on a few issues as to how I understand the situation here in California.

California is an "at will" employment state, but most attorneys would advise their clients have an "at will" clause signed by their employees as a stand-alone agreement or as part of an office manual. Along with an "at will" clause of employment, some attorneys would recommend that some type of "proprietary information" protection language also be part of the employment agreement. If one were to lose a key employee that has built relationships with the patient base, it would be wise to have some type of agreement in place that makes it clear that all patient information is proprietary to the practice.

Your attorney could also draw up language that would specifically address any efforts by the employees to solicit patients away from the practice. I have seen language that also includes proprietary management systems that are in place. Most of us dentists in California understand that covenants "not to compete" for employees (including dental associates) are not defensible in California, but obviously the proprietary information special to any practice could possibly be protected.

The language may have to be crafted differently for each type of dental employee, but any agreement that would help protect the practice goodwill in this fashion would obviously be beneficial for any buyer with these concerns. After all, the goodwill of most practice transitions represents up to 80% of the value of the practice. Maintaining the continued probability of patient visits to the practice, even in the event of employee turnover, is paramount to the value of the practice.

Again, I want to reiterate that these legal Human Resource questions should be directed to your attorney. It might be wise to address these matters well in advance of any planned transition and have your plan reviewed by your attorney on a regular basis to see if there are any changes to the law regarding these matters.

Timothy G. Giroux, DDS is currently the Owner & Broker at **Western Practice Sales (westernpracticesales.com)** and a member of the nationally recognized dental organization, ADS Transitions. Do you have any **Questions? Email them directly to *Dr Giroux at:* wps@succeed.net** or Call **800.641.4179**

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Latin America: Native Populations Affected by Early Onset Periodontal Disease

HESSAM NOWZARI, DDS, PHD, AND JAVIER ENRIQUE BOTERO, DDS, PHD

ABSTRACT Millions of individuals are affected by early onset periodontal disease in Latin America, a continent that includes more than 20 countries. The decision-makers claim that the disease is not commonly encountered. In 2009, 280,919 authorized immigrants were registered in the United States versus 5,460,000 unauthorized (2,600,000 in California). The objective of the present article is to raise awareness about the high prevalence of the disease among Latin Americans and the good prognosis of preventive measures associated with minimal financial cost.

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In Latin America, early onset periodontal disease presents as a silent disease process that affects millions of children and young adults. While the affected individuals can be identified in many villages and towns in Latin America, the study of early onset periodontal disease has been neglected for decades and for invalid reasons. In Latin America, a vast continent that includes 20 countries, the information regarding the pathogenesis of this silent disease in young people remains scarce. The decision-makers claim that localized aggressive periodontitis (LAgP, formerly reported as juvenile periodontitis) is not commonly encountered; yet, it is reported in many investigations in populations of Latin American background.

Latin America, once part of the Spanish and Portuguese empires, refers to territories in the Caribbean, and in Central and South America where Romance languages are primarily spoken (Spanish, Portuguese, and variably French). Due to globalization and population migrations, currently large numbers of Latin Americans seek treatment in the United States and particularly in California. **TABLE 1** provides information about the number of people obtaining legal residence in the United States in 2009 by country of birth and state of residence. The estimated number of unauthorized immigrants by state is also presented in **TABLE 1**. While 280,919 authorized immigrants were registered, the number of unauthorized immigrants was approximately 5,460,000 (2,600,000 in California).

In the 21st century, the health profession must be prepared to meet the challenges necessary to provide care to patients affected by infection-induced periodontal attachment loss and bony

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TABLE 1

Number of People Obtaining Legal Residence in the United States in 2009 by Country of Birth and State of Residence and Estimated Unauthorized Immigrants by State

Country of Birth	Total	Arizona	California	Florida	Texas
Argentina	5,780	42	713	2,392	295
Bolivia	2,837	14	269	499	162
Brazil	14,701	116	1,680	3,620	619
Chile	2,250	28	341	596	111
Colombia	27,849	192	1,386	11,139	1,513
El Salvador	19,909	125	6,509	674	2,632
Guatemala	12,787	189	4,282	884	641
Mexico	164,920	9,168	59,814	4,407	38,597
Peru	16,957	147	2,403	4,293	627
Uruguay	1,775	6	82	633	46
Venezuela	11,154	59	390	6,381	1,112
Unauthorized Immigrants		460,000	2,600,000	720,000	1,680,000

Source: U.S. Immigration Statistics (http://dhs.gov/files/statistics/). Revised August 2010.

defects. In the United States, the evidence strongly suggests that protecting and restoring the smiles of children and adolescents can enhance health and confidence diminish anxiety and negative feelings and, thus, encourage them to explore new ways of moving forward.[1-3]

In order to avoid controversy, the terms "early onset periodontal disease" used within the context of this article refer to the establishment of periodontal inflammation, which can derivate to gingivitis and periodontitis, at an early unknown age.

General Overview

FIGURE 1 provides an estimation of periodontal disease in Latin America. The reported prevalence of cases compatible with aggressive forms of periodontitis in Latin America ranges from 0.32-28 percent. Gingivitis is the most frequent form of periodontal disease ranging from 2.7-100 percent in young individuals and has a tendency to increase with age and thus should be carefully considered. It is difficult to assess whether these values represent the true periodontal condition of the population due to a lack of homogeneity in the studies presented. Several problems can be noted:

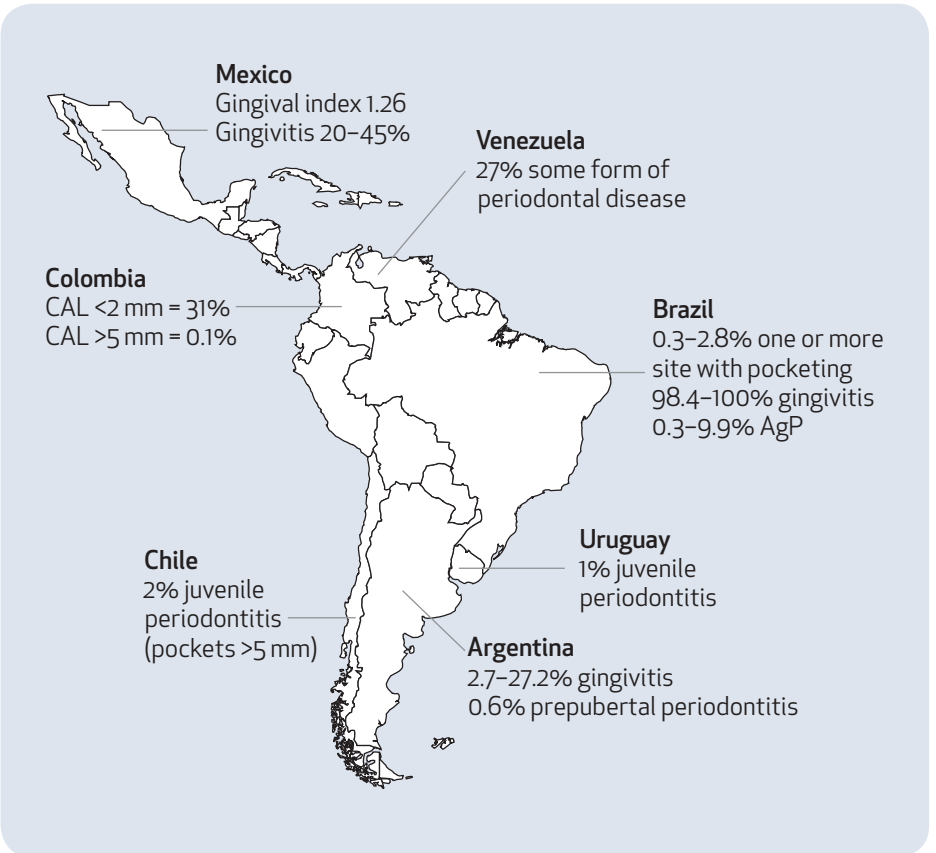

FIGURE 1. Estimated frequencies of gingivitis and aggressive periodontitis in Latin American countries. AgP: aggressive periodontitis.

1. Availability and local pertinence of the information: It is possible that more studies of periodontal disease in Latin America exist but are not easily available internationally. These papers, written in Spanish and Portuguese and not in English, may be published in local nonindexed journals but with great impact within the community.

2. Standardization of periodontal diagnosis and clinical parameters: Change in terminology used to classify periodontal disease has caused multiple terms to be used to describe the same diagnosis, making the analysis of studies complicated.

3. Case definition: The use of many different periodontal parameters threshold levels makes case definition difficult.

4. The use of multiple periodontal indexes: Full-mouth clinical examinations are costly and time consuming. The use of partial-mouth examination has led to underestimation of the disease if the index teeth are not affected.

An Overview of the Literature from Mexico to Chile

Studies from Latin America evaluating periodontal disease in young individuals are reported in **TABLES 2 AND 3**. Starting from Mexico, three published studies have reported a prevalence of 20-61 percent gingival inflammation in a computed sample of more than 3,000 individuals aged 6-19. Although pocketing appeared more frequently with advancing age, in the young population it was not observed.[4-6] From Venezuela, only one study was available, which reported that 27 percent presented with some forms of periodontal disease among 214 examined individuals.[7] Although a detailed description of the periodontal status was not presented, the majority of the examined individuals were affected by gingival inflammation assessed by the Russell's Periodontal Index.

A large study in Colombia involving a representative sample (3,988 subjects 15-19 years old) of the population was performed by the Health Department in 1999.[8] The most common form of periodontal disease affecting the young population was gingivitis. In addition, the description of the periodontal status using a partial clinical examination performed by calibrated clinicians revealed that clinical attachment loss of <2 mm was present in 31 percent while clinical attachment loss of >5 mm was measured in 0.1 percent of the sample. Even though the determination of the final periodontal diagnosis was not reported, it gave an indication that, in some cases, pocketing and clinical attachment loss may have occurred.

IT IS DIFFICULT TO assess whether these values represent the true periodontal condition of the population due to a lack of homogeneity in the studies presented.

Brazil, which is the largest country in South America, provides more substantial studies accounting for a computed sample of more than 9,000 subjects aged 7 to 29. Gingivitis was found to be the most prevalent form of periodontal disease in the young population ranging from 98-100 percent.[9,10] One study reported a 28 percent prevalence of interproximal bone loss assessed in bitewing radiographs, which was indicative of periodontitis.[11] Later studies found that the prevalence of AgP (aggressive periodontitis) may have been overestimated and that the prevalence ranged from 0.3-9.9 percent using a full-mouth examination and radiographic confirmation.[12,13] In a subsequent study, the prevalence of aggressive periodontitis was evaluated in an urban population in southern Brazil.[14] A representative sample of 612 subjects aged 14 to 29 was evaluated. Subjects in the age groups 14 to 19 years and 20 to 29 years were classified with aggressive periodontitis if they had four or more teeth with attachment loss ≥4 mm or ≥5 mm, respectively.

Aggressive periodontitis was diagnosed in 5.5 percent of the subjects. The disease occurred equally among males and females, but was two times more prevalent among non-whites than whites. In the age groups 20 to 24 years and 25 to 29 years, the aggressive periodontitis subjects had a significantly higher prevalence of tooth loss (90.2 percent versus 40.4 percent and 86.1 percent versus 43.4 percent, $P<0.01$) and mean number of missing teeth (2.6 versus 0.9 and 3.4 versus 1.5, $P<0.05$) than subjects without attachment loss. A more recent study assessed the risk indicators for aggressive periodontitis in an untreated and isolated young population from southeastern Brazil.[15] One-hundred-thirty-four subjects aged 12-29 were selected by a census. Of those eligible, 101 subjects received a full-mouth clinical examination. Cases were defined as individuals with four or more teeth with attachment loss >4 mm or >5 mm in the age groups 12-19 and 20-29, respectively.

Overall, 9.9 percent of the subjects were diagnosed with aggressive periodontitis (10.3 percent of the 12-19-year-olds and 9.7 percent of the 20-29-year-olds). The authors concluded that this population from Brazil presented a high prevalence of aggressive periodontitis.

In Argentina, one study suggested that the prevalence of gingivitis ranged from 2.7-27.2 percent and that increased with age.[16] Information regarding aggressive

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TABLE 2

Latin American Studies Reporting the Prevalence of Gingivitis in Young Individuals

Authors	Country	Subjects	Prevalence (%)	Comments
de Muniz et al., 1985	Argentina	2,279 subjects aged 7-8 and 12-13	2.7-27.2%	Frequency of gingivitis increased with age.
Cunha et al., 1998 a,b	Brazil	811 subjects aged 7-14	98-100%	High prevalence of gingivitis in low-income populations.
Gonzalez et al., 1993	Mexico	700 subjects aged 11-17	GI 1.26	Gingival inflammation increased with age.
Carrillo et al., 2000	Mexico	361 subjects aged 11-77	20-45% in 10-19 years old	The presence of periodontal pockets increased with age.
Hernandez et al., 2000	Mexico	2,140 subjects aged 6-14		61.01% of the sample presented some form of periodontal disease as evaluated by the Russell's index.
Ortiz, 2000	Venezuela	214 subjects aged 6-13		27 % of the sample presented some form of periodontal involvement.

GI: Gingival index.

TABLE 3

Latin American Studies Reporting the Prevalence of Periodontitis in Young Individuals

Authors	Country	Subjects	Prevalence	Comments
Funosas et al., 1999	Argentina	152 subjects aged <6	0.66%	One case of AgP was documented.
Gjermo et al., 1984	Brazil	304 subjects aged 15	28%	Periodontal destruction was assessed in bitewing radiographs.
Tinoco et al., 1997	Brazil	7,843 subjects aged 12-19	LAgP 0.3%	Localized aggressive periodontitis cases harbored high levels of *A.actinomycetemcomitans*.
Cortelli et al., 2002	Brazil	600 subjects aged 15-25	1.66% LAgP 3.66% GAgP	Full-mouth examination and confirmed radiographically.
Susin et al., 2005	Brazil	612 subjects aged 14 to 29	5.5%	Full-mouth clinical examination. Low socioeconomic status is an important risk indicator.
Corraini et al., 2009	Brazil	101 subjects aged 12-29	9.9%	Full-mouth examination. High prevalence of AgP in an untreated population.
Lopez et al., 1991	Chile	2,500 subjects aged 15-19	0.32%	Localized aggressive periodontitis was more frequent in low socioeconomic status. Initial screening and full-mouth clinical and radiographic examination.
Tovar et al., 1999	Colombia		Clinical attachment loss <2 mm=31%, clinical attachment loss >5 mm=0.1%	Clinical attachment loss increased with age. Gingival inflammation as the most common sign of periodontal disease.
Campi et al., 1996	Uruguay	100 subjects aged 11-18	1% LAgP	CPITN index was used; only 1 subject presented clinical, radiographic and microbiologic characteristics indicative of localized aggressive periodontitis.

AgP: aggressive periodontitis, LAgP: localized aggressive periodontitis, GAgP: generalized aggressive periodontitis, CPITN: community periodontal index and treatment needs.

periodontitis from Argentina is limited and remains unknown. A later study in a much smaller population sample reported a case of LAgP equivalent to 0.66 percent and confirming that gingivitis is more frequent in children.[17] Similar values were observed in Chile where a random sample of 2,500 subjects aged 15-19 were initially screened for pockets in central incisors.[18] Subsequent to initial screening, a full-mouth clinical and radiographic examination was used to confirm the diagnosis. The prevalence was estimated to be 0.32 percent for LAgP and was more frequent in low socioeconomic status. In Uruguay, LAgP was reported to be 1 percent and confirmed with clinical, radiographic, and microbiological aids.[19]

In summary, although there are limited numbers of studies aimed to determine the prevalence and etiopathogenic variables of early onset periodontal disease in young individuals, and despite heterogeneity in study designs, information suggests that gingivitis in the young population is quite common with a tendency to increase with age. In contrast, information regarding periodontal destruction is more limited and precludes generalizing conclusions. Nonetheless, increase in periodontal probing with or without bone loss can occur and needs further investigation. The high prevalence of periodontitis in adults is often related to lack of diagnosis of early onset periodontal disease and proper oral health measures, which may often lead to irreversible problems in adulthood.

FIGURE 2A.

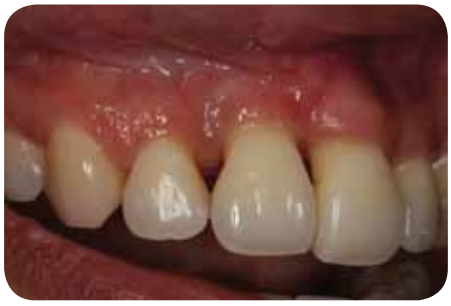

FIGURE 2B.

FIGURE 2. Early onset periodontal disease was neglected in this patient who is now 28 years old. Protruded maxillary incisors touch the lower lip. Note loss of papillae and formation of black spaces between the anterior teeth.

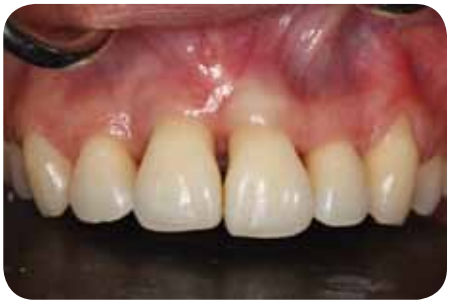

FIGURE 2C.

FIGURE 2D.

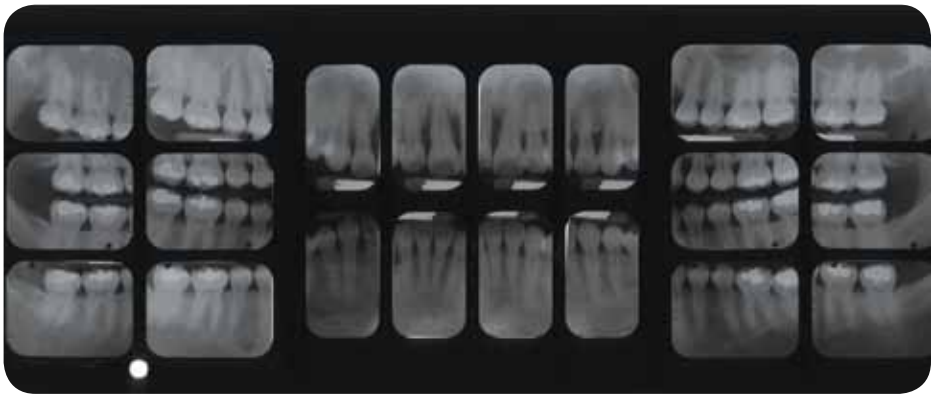

FIGURE 3. Radiographic appearance of the same patient affected by early-in-life oral infection. Note severe bone loss around maxillary anterior teeth.

The Negative Impact of the Disease on Facial Expression

The initiation of early onset periodontal disease is silent and targets the supporting periodontal tissues. The early onset periodontal disease is distinguished from the chronic form of periodontal disease affecting adults in that it is established in children and adolescents and has a rapid rate of tissue destruction. With the establishment of periodontal inflammation, teeth start to shift position and in severe cases, they may be lost. The extrusion or protrusion of maxillary incisors following periodontal bone loss, destruction of anterior papillae, or loss of maxillary or mandibular central incisors seriously damages facial expression (**FIGURES 2 AND 3**). This impairment in the dentition is emotionally and socially relevant for interactions with other members of society. The

self-concept (self-esteem, confidence) and social interactions may also be affected by smiling patterns. Araujo et al. measured the impact of periodontal disease on quality of life of 401 Brazilian patients of both genders aged 19 to 71.[20] Functional limitation was the predominant item, affecting 91.5 percent of the sample. The patients with aggressive periodontitis achieved the highest scores in negative impact. The authors reported that chronic periodontitis affected 56.7 percent of study participants.

Periodontal disease can also be responsible for pain. Further, the condition can lead to emotional pain from rejection and alienation. How are these children interpreting what is happening to them? How do affected adolescents face personal challenges associated with their facial appearance? How do young adults relate to each other? How are relationships influenced by the speech problems and oral malodor?

The reactions eagerly anticipated by the smiling individual with missing teeth and inflamed gingiva may not be forthcoming from others. Expectations and opportunities can be limited because of miscommunication and misinterpretation due to self-consciousness about appearance. Children may show dependent behavior or may regress to behaviors appropriate to a much younger child. Therefore, school-age children affected by destructive early onset periodontal disease may be at risk for delayed educational development due to a poor social interaction and responses from the public, peers, parents, and teachers.

Pathogens Initiating Periodontal Disease in Children and Young Adults

Several microorganisms such as *Aggregatibacter actinomycetemcomitans,* enteric rods, *Porphyromonas gingivalis, Tannerella forsythia, Campylobacter rectus, Eikenella corrodens, Prevotella nigrescens* and *Treponema denticola* have been implicated as important initiators of periodontal disease in children and young adults. Some new evidence also implicates herpesviruses as modulators of the progression of periodontal destruction.

Multiple studies have been performed in Latin America in order to study the association of periodontal disease with periodontal pathogens. Vieira et al. examined the occurrence of *A. actinomycetemcomitans* in Brazilian Indians belonging to the Umutina Reservation, Mato Grosso, Brazil.[21]

THE REACTIONS
eagerly anticipated by the smiling individual with missing teeth and inflamed gingiva may not be forthcoming from others.

Forty-eight native Brazilians with gingivitis and 38 with chronic periodontitis were studied. *A. actinomycetemcomitans* was isolated from 8.33 percent of saliva, supragingival and subgingival samples from patients with gingivitis and from 18.42 percent of saliva and supragingival biofilm, and 26.32 percent subgingival biofilm from patients with chronic periodontitis. The bacterial DNA was detected in 8.33 percent of saliva, supragingival and subgingival biofilm from patients with gingivitis and from 23.68 percent of saliva, 28.95 percent supragingival biofilm and 34.21 percent subgingival biofilm from patients with periodontitis.[21] The results suggested that *A. actinomycetemcomitans* could be associated with attachment loss in this population. Cortelli et al. assessed the occurrence of *A. actinomycetemcomitans* in Brazilians with chronic periodontitis.[22]

A total of 555 (mean age 33.04±12.45) individuals, living in two large areas of the São Paulo state and diagnosed with mild (180 (mean age 29.59±10.94)), moderate (241 (mean age 31.18±11.45)) or severe (134 (mean age 33.04±12.45)) chronic periodontitis were enrolled. *A. actinomycetemcomitans* was detected in 18.4 percent of individuals: 16.1 percent mild; 17.4 percent moderate; and 23.1 percent severe chronic periodontitis. A higher occurrence of this bacterium was found both in the youngest group ($p<0.05$) as well as in the female group ($p<0.05$).
In Colombia, Lafaurie et al. and Botero et al. described the composition of the subgingival microbiota in chronic periodontitis and aggressive periodontitis patients in a Colombian population.[23,24] The authors detected a significant number of Colombian patients being affected by gram-negative enteric rods, in particular aggressive periodontitis patients ($P<0.01$). *P. gingivalis, T. forsythensis,* and *E. corrodens* showed higher frequencies in aggressive periodontitis patients compared to chronic periodontitis and healthy subjects ($P<0.05$). Moving further south, Gajardo et al. measured the prevalence of periodontopathic bacteria in 36 aggressive periodontitis patients in a Chilean population.[25] *C. rectus, P. gingivalis, E. corrodens, P. micros,* and *Capnocytophaga sp.* were the most predominant periodontopathic bacteria.

Various studies have addressed the presence of herpesviruses in periodontitis patients. Botero et al. also examined 30 Colombian subjects diagnosed with periodontitis (20 with chronic periodontitis and 10 with aggressive periodontitis) and

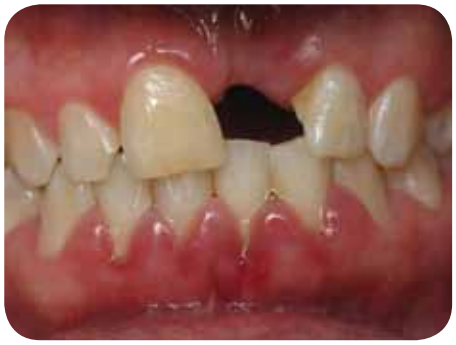

FIGURE 4. Gingivitis in a 16-year-old-male. Note intense gingival inflammation leading to increase sulcus depth.

22 periodontally healthy individuals and correlated subgingival human cytomegalovirus with increased attachment loss and bacterial co-infection.[26] Human cytomegalovirus was more prevalent (P<0.05) in periodontally diseased subjects compared to healthy ones. Furthermore, attachment loss was increased in human cytomegalovirus-positive sites. In the periodontitis groups, higher frequencies and levels of specific periodontopathic bacteria were detected in virus-positive sites. Imbronito et al. in a case-control study, detected HSV-1 (herpes simplex virus), HCMV (human cytomegalovirus), and EBV-1 (Epstein-Barr virus) in 86.7 percent, 46.7 percent, and 33.3 percent of the aggressive periodontitis group, respectively, and in 53.3 percent, 40.0 percent, and 20.0 percent of the gingivitis group, respectively.

A. actinomycetemcomitans was detected significantly more often in the aggressive periodontitis group (P<0.005). Herpesviruses may be related to the etiology of periodontitis by facilitating bacterial infection and by altering the functioning of gingival fibroblasts and promoting the release of inflammatory cytokines and metalloproteinases.[27-30]

The subgingival microbiota in patients with early onset infection in Latin American countries is constituted with a high frequency of known periodontal pathogens. Gram-negative enteric rods appear in high quantities and their etiopathogenic role in periodontal destruction requires further investigations.

Why Gingivitis Should Be Taken Seriously?

A simple and logical analysis of current literature in Latin America reveals that gingival inflammation is evident as a serious epidemic. Children and adults affected by gingivitis are at risk of harboring active periodontal pathogens (**FIGURE 4**). The overgrown and inflamed gingival tissue can be considered sites of active bacterial and viral replication. However, little attention is given to gingivitis as compared to periodontitis perhaps because its treatment seems easy and its potential negative impact not taken seriously.

Once gingival inflammation starts, it may happen undetected and many changes that predispose to periodontitis appear. It has been clear for decades that not all subjects develop gingivitis at the same rate and it is reversible with good plaque control.[31] This is true for most subjects having a fairly good plaque control at home. But for those periodontal sites where plaque removal is insufficient (interproximal sites, faulty restorations), gingival inflammation perpetuates silently.

With gingival inflammation present, the size of the gingiva increases due to edema and increases the depth of the sulcus, and, thus providing the perfect environment for the growth of periodontopathic bacteria (*A. actinomycetemcomitans, P. gingivalis, T. forsythia, T. denticola*). The microbiota shifts from a predominantly gram-positive and aerobic streptococci-composed biofilm to a gram-negative and anaerobic rods biofilm.[32] Yumet and Polson reported that more mitotic epithelial activity is associated with the presence of inflammation in the underlying connective tissue.[33]

Although periodontopathic bacteria can be detected at clinically healthy sites, it is at inflamed sites that they colonize subgingivally.[34-38] An increase in the number of periodontopathic bacteria results in the production of highly toxic virulence factors and antigens, promoting inflammation in a chronic manner. This can be shown in studies of LAgP where a specific and highly virulent serotype of *A. actinomycetemcomitans* (JP2) is frequent and positively correlates to advanced periodontal destruction and reduced response to treatment.[39-45] But little is known on what levels of *A. actinomycetemcomitans* or any other periodontopathic bacteria are necessary to start periodontal destruction. Inflammation occurs not only locally but an increase in circulating neutrophils (PMNs) is observed in experimental gingivitis.[46] These two lines of evidence indicate that gingivitis is an inflammatory-infectious disease with a chronic nature that could progress to periodontitis.

It is only when periodontitis is detected that the importance of gingivitis is recognized. Today, there is evidence that poor intraoral conditions affect the systemic health of individuals. Contreras et al. have provided evidence for a potential association between subgingival *P. gingivalis, T. forsythia,* and *E. corrodens* and preeclampsia affecting Colombian pregnant women (P<0.01).[47] In a case-control study carried out in Cali, Colombia, that included 130 preeclamptic and 243 nonpreeclamptic women between 26 to 36 weeks of pregnancy, 63.8 percent of preeclamptic women and 36.6 percent of controls were affected by periodontitis (P<0.001). The average newborn birthweight from preeclamptic mothers was 2,453 gr, whereas in controls it was 2,981 gr (P<0.001).

The average age in this study was 24 but there were also younger women who were also affected. Leon et al. examined the amniotic fluid of 26 pregnant women with a diagnosis of threatened premature

labor at a gestational age ranging between 24 and 34 weeks.[48] Eight women presented with gingivitis, 12 with chronic periodontitis, and six without periodontal disease. Microbial invasion of the amniotic cavity by *P. gingivalis* was 30.8 percent. In these eight patients, *P. gingivalis* was present in both the subgingival samples and the respective amniotic fluid sample. The authors suggested that the presence of microbial invasion of the amniotic cavity by *P. gingivalis* could indicate a role for periodontal pathogenic bacteria in pregnant women with a diagnosis of threatened premature labor. Furthermore, in Mexico, Martinez-Martinez et al. detected periodontal bacterial DNA in serum and synovial fluid of patients affected by refractory rheumatoid arthritis.[49]

One hundred percent of patients showed periodontal bacterial DNA in subgingival dental plaque and serum and synovial fluid and 83.5 percent in serum. *P. intermedia* and *P. gingivalis* were the species most frequently identified. The authors suggested that the possible pathway of transport of periodontal bacterial DNA from the oral cavity to joints could be via the free-form of DNA. As a result of the completion of these studies, the awareness on the importance of good oral health is being considered by the governments in order to take action and promote high oral hygiene standards.

An Economic Fact

The gross domestic product on a purchasing power parity basis divided by population reflects the quality of living standards of a particular country. A list of countries ranked shows that while the United States is ranked 11 ($46,000 U.S. dollars), the first Latin American country to appear in the list is Chile (ranked 76, $14,600 U.S. dollars) followed by Argentina (ranked 80, $13,400 U.S. dollars) and Mexico (ranked 83, $13,200 U.S. dollars). Paraguay is ranked 148 ($4,600 U.S. dollars) just above African countries.[50] The poor living standards of many Latin American countries affect access to health services and thus prevention of periodontal disease is limited. This creates a situation where the limited financial resources available are used for different government priorities other than health of the population. As a result, thousands of unaware families and their children suffer from early-in-life infection.

A Framework for Action

Research has led to a variety of approaches to improve periodontal health through prevention and early diagnosis. Education, prevention and early intervention ensure the greatest chance for success. Periodontal health can be improved through a simple awareness campaign at the family levels regarding the overall good prognosis associated with preventive measures. Mothers are in a unique position to reduce or eliminate the risk of gingival inflammation in children and reduce or eliminate early onset periodontal disease in Latin America. By learning how to prevent gingivitis in children, mothers or other caregivers can improve the overall health of infants and young children.

Conclusions

Periodontal disease is a serious problem in Latin American countries, affecting the smiles of young individuals and creating self-esteem problems that could reflect in poor behavioral attitudes. It is when the governments take oral health as a serious priority in their policies that the problem can begin to be resolved.

As many young individuals are affected, it is the responsibility of the parents to promote good oral hygiene habits and to visit the dentist regularly for dental and periodontal checkups. ■■■■

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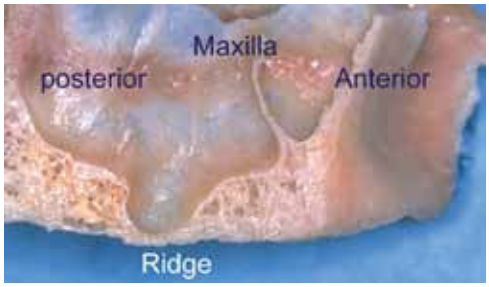

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Herpesvirus Periodontitis: Infection Beyond Biofilm

JØRGEN SLOTS, DDS, DMD, PHD, MS, MBA

ABSTRACT Herpesviruses, including Epstein-Barr virus and cytomegalovirus, occur at high copy counts in aggressive periodontitis, and may interact synergistically with periodontopathic bacteria in the etiology of the disease. Herpesvirus active periodontal infections may impair local host defenses and thus increase the aggressiveness of resident periodontopathic bacteria. The bacteria, in turn, may augment the virulence of the herpesviruses. The abundance of herpesviruses in periodontitis redefines the pathogenic paradigm of the disease and may have significant clinical implications.

AUTHOR

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Periodontitis is a complex infectious disease that is associated with specific bacterial species.[1] Healthy gingival sites are predominated by facultative gram-positive bacteria, whereas periodontitis lesions mainly harbor anaerobic and proteolytic gram-negative species.[2] Microbiological culture and culture-independent molecular studies have identified more than 1,200 bacterial species and 19,000 phylotypes in the oral cavity, and at least 400 bacterial species in subgingival sites, but despite the long list of different bacteria in periodontitis, less than 20 species are designated major periodontal pathogens.[3-6]

It is becoming increasingly clear that major clinical characteristics of periodontitis are difficult to explain solely on the basis of a bacterial infection.[7] It remains an enigma why most patients show periodontitis in relatively few teeth despite an omnipresence of periodontopathic bacteria in saliva. Also, many periodontitis lesions are self-limiting with short-duration morbidity despite a persistent presence of periodontopathic bacteria in the periodontal pocket. Moreover, as clearly evidenced in localized aggressive (juvenile) periodontitis, periodontal tissue destruction tends to occur in a bilateral symmetrical pattern around the midline of the mouth, and may almost reach the apex in one tooth while barely affecting a neighboring tooth sharing the same interproximal space.

The conventional explanation is that periodontitis-prone teeth exhibit anatomies predisposing to enhanced plaque accumulation, but studies have failed to identify a close relationship between supragingival plaque amount and destructive periodontal disease.[8] Case in point is

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TABLE 1

Herpesviruses in Gingival Biopsies From Periodontitis Lesions and Clinically Healthy Sites*

Herpesvirus	Periodontitis (14 subjects)	Healthy periodontium (11 subjects)	P (χ^2 test)
Herpes simplex virus type 1	8 (57)**	1 (9)	0.04
Epstein-Barr virus	11 (79)	3 (27)	0.03
Human cytomegalovirus	12 (86)	2 (18)	0.003
Human herpes virus-6	3 (21)	0 (0)	0.31
Human herpes virus-7	6 (43)	0 (0)	0.04
Human herpes virus-8	4 (29)***	0 (0)	0.17
Presence of herpesviruses	14 (100 %)	5 (45 %)	0.007

* Contreras et al.[16]
** No. (%) virus-positive samples.
*** Three patients were confirmed HIV-positive.

the lingual surface of mandibular molars, which frequently shows massive biofilm build-up but little or no attachment loss, and localized aggressive (juvenile) periodontitis that reveals little plaque formation at sites with rapidly advancing disease. As periodontal pockets of all morphologic types amass high microbial densities, the mere anatomy of the subgingival area is also unlikely to be a key determinant of periodontitis disease severity. Because of the many puzzling clinical features of periodontitis, and as mostly indirect evidence exists for a bacterial etiology of the disease, it is our contention that a pure bacterial cause of periodontitis has been overemphasized.

A Swedish epidemiologic study of 30 years duration may provide indirect evidence for that argument.[9] The study found the percentage of periodontally healthy individuals increased from 8 percent to 44 percent with a parallel decrease in the proportions of subjects with gingivitis and moderate periodontitis, but the prevalence of advanced periodontitis patients remained unchanged at 6-8 percent during the 30-year study period.[9] Apparently, several cases of advanced periodontitis have an etiology that is unresponsive to conventional mechanical therapy.

Recent studies showing the presence of herpesviruses in severe periodontitis sites may provide new important insights into the causation of the disease.[10] Individual periodontitis lesions can harbor millions of genomic copies of herpesviruses as well as papillomaviruses, human immunodeficiency virus (HIV), human T-lymphotropic virus type 1, torquetenovirus, and hepatitis B and C viruses.[11,12] Viruses reside in high levels within gingival tissue. Saliva can contain several additional viruses of medical importance, but their relationship to periodontitis has still to be determined.[13] New gene sequencing technologies will undoubtedly expand the list of viruses in the periodontal virome.[14] Taken together, periodontitis sites can harbor viral copy counts that approach the total bacterial count. The abundance and the variety of pathogenic viruses in periodontitis lesions suggest that viruses are not merely an epiphenomenon of gingival inflammation but are causally related to disease development.

The present article discusses the relationship between herpesviruses and periodontitis, and proposes that a co-infection of active herpesviruses and periodontopathic bacteria constitutes a major cause of periodontitis. Herpes simplex virus type 1, Epstein-Barr virus, and cytomegalovirus are the most studied herpesviruses in periodontology, and they are the main focus of this review. The concept of a herpesviral-bacterial combined etiology of periodontitis may explain a number of the clinical characteristics of the disease and provide new tools for the management of the disease.

Herpesvirus Characteristics

The biological characteristics of the herpesvirus family and of other oral viruses were outlined in a recent review and will here only be summarized briefly.[12] Herpesvirus virions vary in size from 120 to 250 nm and consist of a double-stranded linear DNA molecule surrounded by an icosahedral capsid, a proteinaceous tegument, and a host-derived lipid-containing envelope with embedded viral glycoproteins. Eight herpesvirus species with distinct biological and clinical characteristics infect humans: herpes simplex virus type 1 and 2, varicella-zoster virus, Epstein-Barr virus, cytomegalovirus, and human herpesvirus 6, 7 and 8. Herpesviruses establish a lifelong infection and occur in a latent and in an active stage. Herpesviral persistence as a latent infection requires subversion or evasion of the host's innate and adaptive immune systems, and of the intrinsic antiviral defense that operates at the intracellular level. Reactivation from latency may happen spontaneously or be triggered by a concurrent infection, fever, drugs, tissue trauma, emotional stress, exposure to ultraviolet light, or other factors that impair the host immune defense. Herpesvirus reactivation in turn induces additional immunosuppression, possibly leading to bacterial or viral superinfections, which may not be resolved until the herpesvirus active infection is subdued by the immune system or by pharmacotherapeutics.

Herpesvirus infections show a distinct tendency to cellular and tissue tropism.

TABLE 2

The Prevalence of Subgingival Herpesviruses*

Herpesvirus	Aggressive periodontitis	Chronic periodontitis	Healthy periodontium
Herpes simplex virus type 1	78 %**	26 %	0 %
Epstein-Barr virus	58 %	46 %	8 %
Cytomegalovirus	42 %	52 %	8 %

*Adapted from Slots.[10]
**Median percentage value obtained from more than 20 worldwide studies.

Herpesviruses target various cells of the immune system and subvert host immune functions to their own advantage. Herpes simplex virus type 1 is usually associated with primary infections of the orofacial area and with latent infection of the trigeminal and spinal ganglia. Epstein-Barr virus infects B-lymphocytes, where it establishes latency. Cytomegalovirus infects several cell types and establishes latency in macrophage-granulocyte progenitor cells and in peripheral blood mononuclear cells.

Herpesvirus infections induce strong antiviral innate and adaptive immune responses, which, although incapable of eradicating the infection, are generally effective in controlling viral replication and preventing clinical disease.[11,15] The cellular immune response plays a key role in controlling herpesvirus infections by means of major histocompatibility complex class I-restricted cytotoxic CD8+ T-lymphocytes that recognize viral peptides on the surface of infected cells. Individuals having an Epstein-Barr virus-cytomegalovirus dual infection tend to show markedly stronger T-lymphocyte responses and more severe disease than subjects who are monoinfected by either of the viruses. To evade antiviral immune responses herpesviruses encode genes that interfere with the activation of major histocompatibility complex-restricted T-lymphocytes and of natural killer cells, modify the function of cytokines and their receptors, interact with complement factors, modulate signal transduction and transcription factor activities, suppress apoptosis, and alter various other cellular functionalities. Herpesviruses may participate in disease development by manipulating the regulation of these cellular processes.

Most individuals become infected with herpesviruses early in life, and 60-100 percent of adults are carriers of herpes simplex virus type 1, Epstein-Barr virus, and cytomegalovirus. Herpesvirus infections are a major cause of morbidity in patients with deficits in innate and adaptive immunity, and may also cause clinical disease in immunocompetent persons. The clinical outcome of herpesvirus infections ranges from subclinical or mild disease to encephalitis, pneumonia, and even to cancer, including lymphoma, sarcoma, and carcinoma.[12] Herpes simplex virus type 1 produces herpetic gingivostomatitis and cold sores, and herpes simplex virus type 2 causes genital ulcerous disease and occasionally oral disease. The Epstein-Barr virus is the causative agent of infectious mononucleosis and oral hairy leukoplakia, and is implicated in the etiology of nasopharyngeal carcinoma and various lymphomas. Cytomegalovirus infection is of major clinical significance in pregnant women, newborn infants with congenital infection, immunosuppressed transplant patients, and HIV-infected individuals.

Herpesviruses in Periodontal Disease

The occurrence of herpesviruses in various types of periodontal disease has been studied by qualitative and quantitative polymerase chain reaction identification techniques. **TABLE 1** shows a significantly higher occurrence of herpesviruses in biopsies from periodontitis lesions than from healthy periodontal sites.

TABLE 2 summarizes findings from more than 20 studies worldwide on the prevalence of herpes simplex virus, Epstein-Barr virus, and cytomegalovirus in subgingival sites. Aggressive periodontitis lesions tend to show herpesviruses in a reactivated state, and individual lesions may yield subgingival copy counts as high as 8.3×10^8/ml for Epstein-Barr virus and 4.6×10^5/ml for cytomegalovirus, and the gingival tissue of periodontitis lesions may house even higher viral loads.[10,17] A recent study associated the Epstein-Barr virus and cytomegalovirus with peri-implantitis.[18] In contrast, infected healthy periodontal sites and gingivitis lesions typically harbor herpesviruses in a nontranscriptional phase and in copy counts of only 1,000 to 20,000/ml.[10] Other viruses of the herpesvirus family and various nonherpesviruses can also inhabit advanced periodontitis lesions.[10] The remarkably high copy counts of pathogenic viruses in aggressive periodontitis lesions makes it unlikely that these infectious agents are acting merely as harmless bystanders present in proportion to the severity of the underlying periodontal pathosis.

Herpesvirus Periodontopathic Potential

It is assumed that periodontitis debuts in genetically or environmentally predisposed individuals who are infected with virulent infectious agents and reveal persistent gingival inflammation and distinct immune responses.[11,19] Fitting that concept, herpesviruses are implicated in the development of periodontitis. The

pathogenicity of herpesviruses is executed through direct virus infection and replication, and via a virally induced alteration of the host immune defense. The early phases of periodontitis in immunologically naïve hosts may predominantly involve cytopathogenic events, whereas most clinical manifestations in immunocompetent individuals are secondary to cellular or humoral immune responses. A periodontal herpesvirus infection may induce a significant portion of the immune reactions in periodontitis.[10]

The Epstein-Barr virus and cytomegalovirus can infect and alter functions of periodontal monocytes, macrophages and lymphocytes, and may exert direct cytopathic effects on periodontal fibroblasts, keratinocytes, endothelial cells, bone cells, and polymorphonuclear leukocytes. A periodontal herpesvirus infection may increase the pathogenicity of the periodontal microbiota by expressing herpesvirus proteins on eukaryotic cell membranes that may serve as new bacterial binding sites, or by inducing abnormalities in the adherence, chemotaxis, phagocytic and oxidative, secretory, and bactericidal activities of polymorphonuclear leukocytes.[11] However, the interaction between herpesviruses and bacteria is most likely bidirectional, with bacterial enzymes or other inflammation-inducing factors having the potential to activate periodontal herpesviruses.[11] Experimental mice infected with murine cytomegalovirus-*Porphyromonas gingivalis* exhibited a significantly higher mortality rate than mice infected with murine cytomegalovirus-*Escherichia coli*.[20] The potential of *P. gingivalis* to suppress interferon-gamma antiviral host response, probably by means of proteolytic enzymes, may partly explain the observed increase in cytomegalovirus pathogenicity.[21]

A statistical relationship has been found between various herpesviral-bacterial consortia and periodontal disease severity, indicating a periodontopathogenetic synergy between the infectious agents. Numerous studies of medical diseases and experimental infections have revealed that a viral-bacterial coinfection produces more severe illness than a single infection by either of the two types of infectious agents.[11] Periodontal herpes simplex virus type 1, the Epstein-Barr virus, and cytomegalovirus have been linked to an elevated occurrence of the putative periodontal pathogens *P. gingivalis, Tannerella forsythia, Dialister pneumosintes, Prevotella intermedia, Prevotella nigrescens, Treponema denticola, Campylobacter rectus* and *Aggregatibacter (Actinobacillus) actinomycetemcomitans*.[7,11] The Epstein-Barr virus and cytomegalovirus seem to be most closely associated with *P. gingivalis* and *T. forsythia,* two bacterial species with high periodontopathic potential, and the linkage between cytomegalovirus and *P. gingivalis* appears to be particularly strong.[7,11,22-24]

PERHAPS PERIODONTITIS can teleologically be viewed as the biological price paid by the host to control periodontal herpesviruses and avoid viral dissemination and serious systemic diseases.

Herpesvirus infections induce an expression of pro-inflammatory cytokines and chemokines as part of the host defense against the viral infection.[25] The Epstein-Barr virus and cytomegalovirus infections can up-regulate interleukin-1β and tumor necrosis factor-α gene expression of monocytes and macrophages.[26] In turn, interleukin-1β and tumor necrosis factor-α may upregulate matrix metalloproteinase, downregulate tissue inhibitors of metalloproteinase, and activate osteoclasts.[27] Increased levels of pro-inflammatory cytokines in periodontal sites have been associated with an enhanced risk of periodontal tissue destruction.[27,28]

Even though pro-inflammatory cytokines have the potential to initiate collagen degradation and alveolar bone resorption, the periodontal cytokine response may actually be beneficial overall by preventing the activation and widespread dissemination of virulent viruses. Perhaps periodontitis can teleologically be viewed as the biological price paid by the host to control periodontal herpesviruses and avoid viral dissemination and serious systemic diseases.

Herpesvirus-Bacterium-Host Response Model of Periodontitis

FIGURE 1 depicts a model for the development of periodontitis, which as its core, has a sequential infectious process that proceeds from bacteria to herpesvirus to bacteria.[11] In the herpesviral-bacterial model of periodontitis, herpesvirus-related cytopathogenic effects, immune evasion, immunopathogenicity, latency, reactivation from latency, and tissue/site tropism comprise important aspects of periodontal pathosis. Initially, bacteria in the dental biofilm induce gingivitis, which permits latent herpesviruses embedded in macrophages, T-lymphocytes and B-lymphocytes to enter the periodontium.[29] Cytomegalovirus can replicate in gingival tissue, which may help to sustain the periodontal infection. Reactivation of the latent herpesviruses may occur spontane-

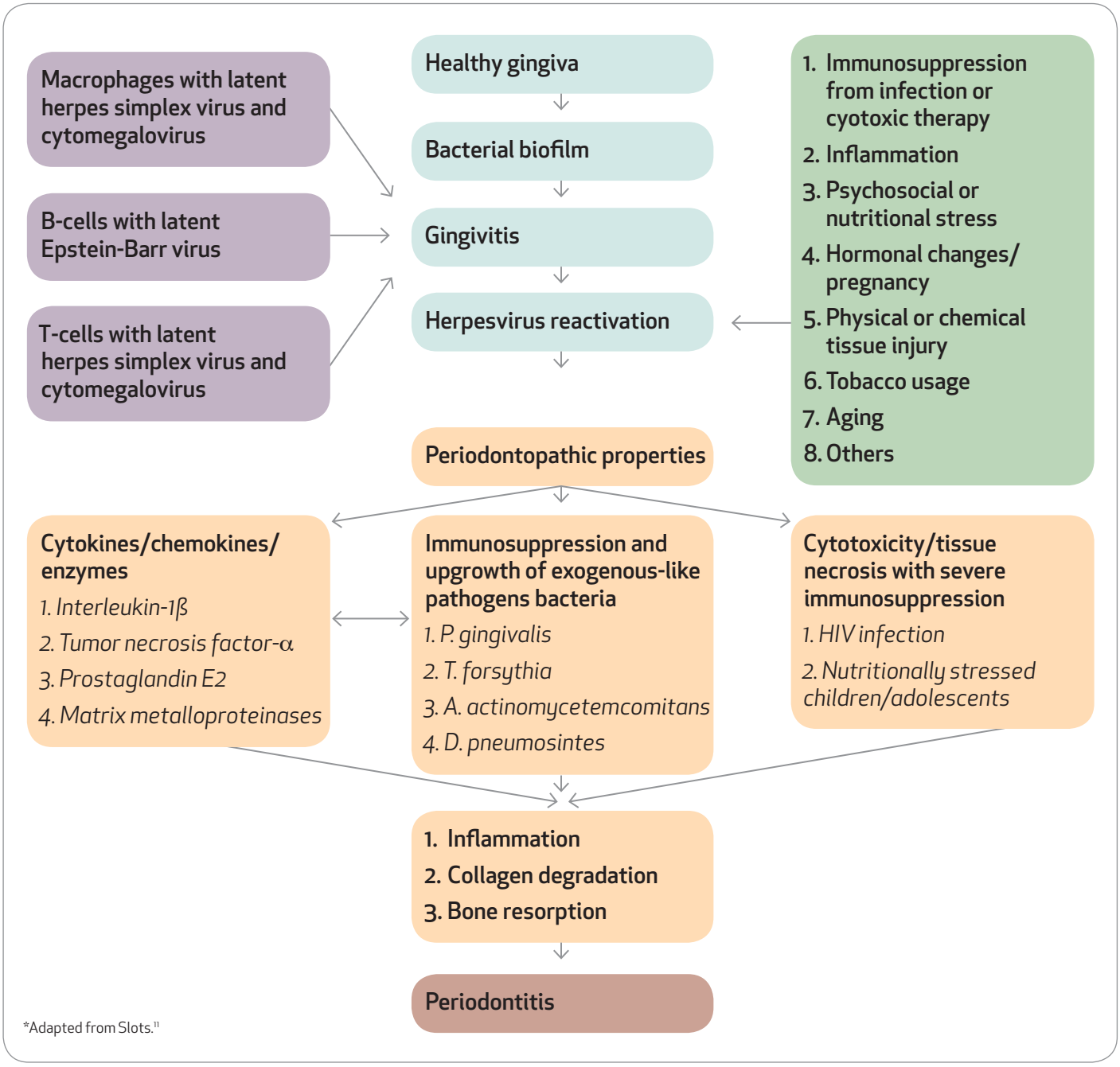

FIGURE 1. Herpesvirus model of periodontitis.*

ously or during periods of decreased host defense, resulting from drug-induced immunosuppression, concurrent infection, unusual and prolonged emotional stress, hormonal changes, physical trauma, etc. Perhaps not coincidentally, most herpesvirus activating factors are also suspected risk factors/indicators for periodontitis.[30] As described above, the pro-inflammatory cytokines released during the herpesvirus infection can potentially activate matrix metalloproteinases and osteoclasts and impair the immune defense against periodontopathic bacteria.

Therapeutic Concepts

Periodontal scaling and root planing, or other means of instrumental removal of dental biofilms, can lower herpesvirus counts in the periodontal pocket and in saliva. Topical antiseptics that are active against both herpesviruses and bacteria (e.g.,

sodium hypochlorite and povidone-iodine) can further reduce the periodontal load of pathogenic agents.[31-36] Selective patients may also benefit from systemic treatment with antiviral and antibacterial medications. Sunde et al. described a refractory periodontitis patient with high Epstein-Barr virus copy counts who was treated with the anti-herpesvirus drug valacyclovir-HCl (Valtrex, 500 mg twice a day for 10 days).[37] The treatment suppressed the Epstein-Barr virus to an undetectable level for at least one year and resulted in a "dramatic" clinical improvement.[37] As periodontal herpesviruses may trigger overgrowth of bacterial pathogens, a systemic antiviral therapy should probably precede an antibiotic therapy against the periodontopathic bacteria.

Effective anti-infective periodontal therapy includes professional administration of well-tolerated antimicrobial agents, each exhibiting high activity against periodontal pathogens, and delivered in ways that simultaneously affect pathogens residing in different ecological niches of the oral cavity.[38,39] It is recommended to employ oral rinsing with dilute sodium hypochlorite (bleach) or chlorhexidine for general disinfection, povidone-iodine or dilute sodium hypochlorite for subgingival irrigation, and systemic antibiotics for infectious agents that reside within periodontal tissue and in difficult-to-reach subgingival and extradental sites. The follow-up maintenance program should have a strong anti-infective emphasis.[40] It is recommended that patient self-care includes subgingival irrigation with dilute sodium hypochlorite and oral rinsing with dilute sodium hypochlorite or chlorhexidine two to three times per week.

Summary and Perspectives

Periodontal research has long been a significant strength of the University of Southern California School of Dentistry, and periodontal viral infection is an area of particular expertise. The etiopathogeny of periodontitis includes virulence factors of herpesviruses and bacteria, host immune responses against viral and bacterial infections, and manipulation of host cell processes by the infectious agents.

Herpesviruses may induce periodontitis by activating tissue-destroying pathways of the immune system and by predisposing an individual to bacterial carriage and increased bacterial load. Conventional periodontal treatment can cause a multiple-fold reduction in copy counts of periodontal and salivary herpesvirus species, and treatment with the anti-herpesvirus drug valacyclovir may decrease the periodontal copy count of the Epstein-Barr virus to virtually undetectable level and give rise to a marked improvement in the periodontal conditions. As anti-herpesvirus immunity may be an important determinant of a stable periodontium, a future availability of herpesvirus vaccines makes the topic of periodontopathic herpesviruses particularly interesting. Control of herpesviruses by vaccination has the potential to reduce the need for the traditional periodontal therapies of surgery and antibiotics. The concept of herpesviral-bacterial co-infection in periodontitis may unlock many of the intricacies of the disease, and constitutes an important theme for further research. ■■■■

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Dynamic Therapeutic Approach for Individuals Affected With Aggressive Periodontitis

KIAN KAR, DDS, MS; KRIKOR SIMONIAN, DDS; AND HESSAM NOWZARI, DDS, PHD

ABSTRACT Management of patients affected with aggressive periodontitis is complicated by several poorly understood etiological and modifying factors that create difficulty in establishing a universal treatment recommendation. The goal of this manuscript is to underscore the complexity of therapy and to provide some guidelines in the decision-making process and interdisciplinary therapy. A dynamic approach is presented to formulate strategies in diagnosis and treatment planning that is both patient- and site-specific.

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In assessing a patient with any disease, it is important to identify causative and modifying factors that contribute to the disease initiation and progress in order to offer a treatment. However, because of a complex combination of incompletely understood etiological and risk factors in periodontitis, it is not possible to assign a simple cause-and-effect diagnosis (e.g., streptococcal sore throat). Therefore, a classification system is utilized to study disease patterns and types in large populations of patients to provide a framework for studying the epidemiology, etiology, and treatment outcomes for a given group of similar diseases.[1-3] As a starting point, such a system can serve to generate a clinical framework for periodontal diagnosis. In clinical management of specific patients, a diagnosis should be made for the individual within the classification framework.[4]

The classification of "aggressive periodontitis" was adopted by the 1999 workshop of American Academy of Periodontology to describe a specific pattern of diseases previously classified as periodontosis, localized juvenile periodontitis, generalized juvenile periodontitis, early onset periodontitis, and rapidly progressive periodontitis.[5,6] This classification was adopted to avoid using a patient's age as criteria for categorizing periodontal disease. Instead, the classification is based on clinical, radiographic, historical, and laboratory findings.

Clinical Features of Aggressive Periodontitis

Aggressive periodontitis is a specific type of periodontitis with identifiable clinical and laboratory findings that are not characteristic for chronic periodontitis.

According to the consensus report of American Academy of Periodontol-

ogy on aggressive periodontitis the common features of the disease are:

- Patients are clinically healthy except for the presence of periodontitis;
- Rapid attachment loss and bone destruction; and
- Familial aggregation.

The following secondary features may also be present:

- Amounts of microbial deposits are inconsistent with the severity of periodontal tissue destruction;
- Elevated proportions of *Aggrigatibacter Actinomycetemcomitans* and *Porphyromonas gingivalis* in some populations;
- Phagocyte abnormalities;
- Hyper-responsive macrophage phenotype, including elevated levels of PGE2 and IL-1;
- Progression of attachment loss and bone loss may be self-arresting; and
- Lack of caries or low caries index.

The diagnosis and classification is based on clinical, radiographic, and historical data and, some, or possibly all, of the above characteristics. Laboratory testing may not be essential for assigning a diagnosis for aggressive periodontitis even though it could be helpful in clinical decision-making. The influence of modifying risk factors (e.g., cigarette smoking, emotional stress, drugs, sex hormones, etc.) should be considered to manage individual patients who are affected with aggressive periodontitis.

There are enough specific features to classify aggressive periodontitis into localized and generalized forms. These features are presented in **TABLE 1**.

A thorough review of medical and family history will aid to identify individuals whose periodontal disease may be associated with specific syndromes or systemic conditions. Individuals with significant systemic modifiers of the innate and adaptive immune responses should be classified as "periodontitis as a manifestation of systemic disease." Negative effects of certain systemic conditions will increase the patient's susceptibility to microbial plaque and consequently to severe and extensive periodontal attachment loss and early tooth loss. This group of diseases includes neutropenia, hypophosphatasia, leukemias, Chediak–Higashi syndrome, leukocyte adhesion deficiency, Papillon–Lefèvre syndrome, trisomy 21, histiocytosis, and agranulocytosis. Proper management of these patients requires management of systemic diseases that may be responsible for the patient's severe periodontitis in conjunction with periodontal infection control.[6,7]

TABLE 1

Characteristics of Localized and Generalized Aggressive Periodontitis According to American Academy of Periodontology

Localized Aggressive Periodontitis

- Age of onset around puberty
- Robust serum antibody response to infecting agents
- Localized first molar/incisor presentation with interproximal attachment loss on at least two permanent teeth, one of which is a first molar, and involving no more than two teeth other than first molars and incisors

Generalized Aggressive Periodontitis

- Usually affecting individuals under the age of 30 but patients may be older
- Poor serum antibody response to infecting agents
- Pronounced episodic nature of the destruction of attachment and alveolar bone
- Generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors

Treatment Planning

The first factor in developing a treatment plan for patients who are affected by aggressive periodontitis is to identify the esthetic and functional needs amongst the patient, restorative dentist, and periodontist. It is critical to identify patient expectations, realistic attainability of the treatment desires, treatment limitation, risk of future breakdown, and dynamic nature of the therapy. When functional or esthetic concerns are present, treatment strategy needs to address the strategic value (risk and cost benefit considerations) of the remaining teeth in providing a functional and esthetic outcome, if infection control and periodontal stability is to be achieved. In these circumstances, a longer provisional phase period of nine to 12 months is advisable to evaluate the outcome of periodontal therapy and periodontal stability before committing to a definitive reconstructive phase. Occasionally, alternative restorative suggestions, such as shortened dental arch or transitional fixed or removable prosthesis, may be considered. This communication is especially important when there are major restorative treatment needs, particularly when considering implant therapy.

Patients with a history of severe chronic periodontitis and aggressive periodontitis may be at additional risk of adverse peri-implant soft-, and hard-tissue outcomes. Peri-implant infections share both bacteriological and histopathological similarities to both aggressive and chronic periodontitis.[8-14] Patients with a history of periodontitis pose a risk for peri-implant diseases, thus, in younger patients with aggressive periodontitis, especially the general-

ized type, it is prudent to defer major implant therapy to later stages when the patient is older.[15-17] This strategy may reduce the chance of early peri-implant complications and provide a better long-term prognosis of implant prosthesis.

When no major functional or esthetic concerns are present, the aims of the therapeutic approaches are primarily on maintaining the periodontium. Localized aggressive periodontitis has been reported to be self-limiting, whereas individuals with the generalized form continue to lose periodontal attachment and teeth over time; however, the residual periodontal lesion and bony defects are major contributing factor for future periodontal disease as the patient ages.[5,18,19] Also, some cases of localized aggressive periodontitis progress to generalized aggressive periodontitis, that may resemble or transform to chronic periodontitis at a later stage.[19-21]

Additionally, risk factors have similar long-term influences on both chronic periodontitis and aggressive periodontitis, although one could argue that with younger patient age and greater initial attachment loss may dictate a poorer long-term prognosis in aggressive disease. Since patients are mostly evaluated at a cross-section of a time, it requires a careful assessment of history and the clinical presentation of periodontitis to identify a past history of aggressive disease to identify a "burn out" stage that may be masked or confused with chronic periodontitis among the middle-aged or older population or with periodontitis as a manifestation of systemic disease.

Since the aggressive forms of periodontitis have distinctive features from chronic periodontitis and potentially respond differently to therapy, the authors are suggesting a tailored treatment planning approach in treatment of patients with aggressive periodontitis.[4,22] This approach is dynamic, both in planning and therapeutic recommendations. Unlike chronic periodontitis where there are more established protocols for therapy, the authors are suggesting that within the overall classification of localized or generalized aggressive periodontitis, existing therapeutic approaches need to be tailored individually based on clinical features and perceived patient- and site-specific diagnoses. This decision-making process is adaptive to following clinical parameters:

- Systemic association;
- Chief complaint and patient expectations (functional and esthetic demands including restorative/reconstruction needs);
- Onset of the disease and patient's age;
- Pattern (distribution) of the disease;
- Severity of attachment loss (pocket depths and clinical attachment loss, radiographic bone loss and bony defects);
- Quantity of etiological factors (plaque and calculus index/code);
- Severity of gingival inflammation (gingival index and bleeding on probing);
- Bacteriological association and consideration of systemic antibiotic therapy and adjunctive local anti-infective therapy;
- Initial mechanical debridement (scaling and root planing) protocol;
- Periodontal re-evaluation and supportive (maintenance) therapy;
- Surgical therapy for infection control and repair of periodontal defects; and
- Implant therapy.

This dynamic approach should be applied to both diagnostic (such as radiographic and laboratory tests) and therapeutic procedures. Following is a review and implementation recommendations considering above clinical parameters to provide guidelines for infection control and periodontal repair of individuals with aggressive periodontitis.

Mechanical Debridement

Scaling and root planing, as initial phase nonsurgical therapy, is traditionally performed by quadrants at different appointments. The efficacy of this approach has been demonstrated by a classical series of studies by Badersten et al.[23-25] Furthermore, a number of systematic reviews on the efficacy of different modalities of mechanical nonsurgical periodontal therapy have been published.[26-30] However, the effectiveness and the efficiency of the traditional mechanical therapy have mostly been analyzed in a patient population affected by chronic periodontitis. Aggressive periodontitis is considered a more site-specific and bacterial-specific periodontal infection that is strongly correlated to the host or early-in-life periodontal infection.[16,31,32] While mechanical debridement is effective in the reduction of bacterial plaque, specific bacteria repopulate periodontal pocket within three to seven days after treatment, restoring bacterial counts to almost pretreatment levels.[33]

Periodontal pathogens commonly associated with aggressive periodontitis, such as *A. actinomycetemcomitans, Bacteroides species,* and *P. gingivalis,* colonize different intraoral habitats in addition to periodontal pockets, including the tongue, buccal mucosa, saliva, and tonsils.[34-37] Saliva probably acts as the major vector of bacterial transmission in most inter-individual cases.[38] When there is an increase in periodontal pathogens around teeth, similar microbial flora are observed around neighboring implants as well, indicating an intraoral transmission of those presumptive periodontal pathogens.[39,40] Therefore, if periodontal treatment does not result in elimination of pathogens from the mucous membranes as well as periodontal pockets, these surfaces may function as:

TABLE 2

Original Protocol of Full-Mouth Disinfection Introduced by Quirynen et al.[49]

- Full-mouth scaling and root planing (the entire dentition in two visits within 24 hours, i.e., two consecutive days) under local anesthesia
- Brushing of dorsum of tongue for one minute with 1 percent chlorhexidine gel
- Mouthrinsing twice with 0.2 percent chlorhexidine mouth rinse for one minute (during the last 10 seconds, the patient had to gargle in an attempt to reach the tonsils)
- Subgingival irrigation of all pockets three times within 10 minutes with chlorhexidine 1 percent gel after both sessions of scaling and root planing and repeated on Day 8, using a syringe with marks at 6 and 8 mm
- Mouthrinsing at home with 10 ml of 0.2 percent chlorhexidine mouthrinse twice daily for one minute for the following two weeks
- Oral hygiene instructions including toothbrushing, interdental cleaning with interdental brushes or other aids, and tongue brushing

- Source of reinfection for the healing and healthy periodontium after treatment;[33,41]
- Source of transmission to family members;[42] and
- Reservoir for infection of tissues around implants.[39,40]

Quirynen described that periodontal pathogens are present in various ecologic niches and that the transmission of these may occur from individual to individual as well as within the oral cavity among sites.[41] This evidence reinforces the need for a full-mouth approach to periodontal infection control especially in cases of aggressive periodontitis rather than treating individual sites. Full-mouth disinfection control as described by Quirynen (**TABLE 2**) consists of mechanical debridement within a short span of time (24-48 hours) with adjunctive use of local anti-infective agents for additional disinfection (i.e., chlorhexidine) during the initial healing period (two weeks).[43] This protocol may be supplemented with systemic antibiotic therapy when indicated. This approach considerably reduces the chance of re-infection of treated pockets by bacterial translocation from other untreated pockets or the intraoral sites. Furthermore, Guerrero also reported greater clinical improvement using an enhanced mechanical debridement within 24 hours and full-mouth disinfection through use of systemic antibiotic therapy and chlorhexidine rinses for two weeks, compared to traditional quadrant scaling and root planing for treatment of patients with generalized aggressive periodontitis.[44]

Considering a dynamic approach, the decision to perform nonsurgical mechanical debridement is dependent on presentation of etiological and local factors (plaque, calculus, and pocket depths). In cases of generalized aggressive periodontitis, full-mouth scaling and root planing utilizing a full-mouth disinfection approach is performed preferably within 48 hours. In cases of localized aggressive periodontitis generalized scaling (debridement) and localized root planing will be performed in one session.

When initial heavy inflammation with tenacious and heavy calculus and deep pockets are present, the patient will be scheduled for an early re-evaluation within two weeks of initial scaling and root planing to retreat areas with residual detectable calculus, with the aim of mechanical disruption of biofilm (if indicated, systemic antibiotic will be administered after initial scaling and root planing regardless of the need for early re-evaluation). Patients without adequate home care (more than 20 percent O'Leary plaque index) will be scheduled for biweekly plaque control appointments. Remaining patients will be re-evaluated monthly for the first three months.

Antibiotic Therapy

A number of studies have demonstrated minimal improvement and high percentage of nonresponders when using mechanical debridement alone in treatment of patients with aggressive periodontitis.[45-48] This is due to the fact that nonsurgical therapy alone does not completely eradicate certain subgingival periodontal pathogens, including *A. actinomycetemcomitans* black-pigmented Bacteroides species, and Capnocytophaga species.[49-51]

Similarly, local antibiotic therapy does not seem to eliminate *A. actinomycetemcomitans* when used in treatment of localized aggressive periodontitis.[49,52,53] To target these specific presumptive periodontal pathogens that are highly associated with aggressive periodontitis, mechanical debridement needs to be supplemented with systemic antibiotics. Using this strategy, the number of spirochetes, *A. actinomycetemcomitans* and *Capnocytophaga* were reduced to undetectable levels and significant improvement in clinical outcomes were observed.[50,51,54,55] The use of systemic antibiotics may enhance gains in attachment level and alter the subgingival bacterial profiles.[56] Additionally, full-mouth scaling and root planing, along with systemic combination metronidazole and amoxicillin or metronidazole alone, and antimicrobial rinses have been advocated for patients with generalized aggressive periodontitis.[46-48,57-59]

Some authors propose systemic antibiotic therapy for all cases of moderate to severe periodontitis without microbial testing. This recommendation is made regardless of a diagnosis of chronic or aggressive periodontitis.[57,60,61] However,

TABLE 3

Recommendation for Use of Oral Systemic Antimicrobial Therapy Based on Detection of Putative Periodontal Pathogens (adopted from Slots)

Detection any of the following:

- *A. actinomycetemcomitans*
- Red complex[71]
  - *Tannerella forsythia*
  - *P. gingivalis*
  - *Treponema denticola*

Antibiotic recommendation:

- 250 mg amoxicilllin-375 mg metronidazole /TID/eight days
- In cases of penicillin allergy:
  - Metronidazole alone (500 mg/TID/8 days)
- In case of metronidazole and penicillin allergy:
  - Clincamycin 300 mg/TID/8 days or
  - Azithromycin (250-500 mg/QD/ 4-7 days)

Detection of:

- Enteric gram-negative rods

Antibiotic recommendation:

- Ciprofloxacin (500 mg/BID/8 days)

No detection of any of the following:

- *A. actinomycetemcomitans,*
- Red complex: [71]
  - *Tannerella forsythia*
  - *P. gingivalis*
  - *Treponema denticola*
- Enteric gram-negative rods

No systemic antibiotic therapy

QD=once daily, BID=twice daily, TID=thrice daily
Recommendation is for or systemically healthy adults with normal body weight.

widespread use of systemic antibiotic therapy in a large population of people affected by periodontitis has a potential of selecting antibiotic resistance species.[62] Moreover, patients with aggressive periodontitis benefit more than the patients with chronic periodontitis from adjunctive use of systemic antibiotic therapy.[63,64]

Clinically, the magnitude of change in some sites may be greater when antibiotics are used, making it a more viable treatment option.[22] The effectiveness of systemic antibiotic treatment increases when it is administered immediately after scaling and root planing.[65] Both the American Academy of Periodontology and the European Federation of Periodontology indicate that the adjunctive use of systemic antibiotic therapy benefits patients with aggressive periodontitis. However, both also emphasize that the optimal drug, dosage, and duration to provide the greatest effect is not completely understood.[63,66] Nevertheless, the choice of antibiotic treatment is best deferred to the result of bacteriological sampling.[67]

It should be mentioned here that there is growing evidence supporting the potential role of viruses in pathogenesis of aggressive periodontitis. It appears that a high periodontal load of active viruses such as the Epstein–Barr virus or cytomegalovirus is associated with aggressive periodontitis. There are hypotheses of synergistic viral and bacterial co infections in the pathogenesis of aggressive periodontitis; but their role, if any, in the initiation of the disease is not defined.[68-70] Therefore, the potential benefit of an antiviral treatment strategy is not currently established.

When utilizing the concept of dynamic therapeutic approach for the use of antibiotic therapy, one has to make a clinical judgment as to when and how (local or systemic) to administer antibiotic therapy. As mentioned earlier, the decision for the type of antibiotic is best to be deferred to bacterial sampling since the presence or absence of certain pathogens may change the decision to administer different choices or combination of antibiotic regiments. **TABLE 3** summarizes treatment recommendations based on the detection of putative periodontal pathogens. When a patient with classification of aggressive periodontitis is not positive for any of the red complex pathogens *(P. gingivalis, T. Forsythia, T. denticola), A. actinomycetemcomitans,* and enteric gram-negative rods, a retest may be indicated if clinical presentation of a given case would constitute suspicion of specific infection (i.e., in cases of severe periodontal disease specially among young individuals).[71] However, once a negative detection is confirmed, no systemic antibiotic therapy is advised. This strategy is employed to avoid unnecessary exposure of patients to a course of systemic antibiotic therapy. Yet, a subsequent bacterial retesting is recommended if no significant clinical improvement is observed after initial mechanical debridement and/ or subsequent surgical therapy. Use of local antimicrobial therapy may be considered for specific sites with supra bony pockets to reduce gingival inflammation only when no systemic antibiotic therapy is indicated.

Moreover, several local anti-infective agents are reported to provide favorable clinical outcome in control of inflammation and antibacterial property that might be of significance in management of periodontal infection among patients with aggressive periodontitis including 0.2 percent chlorhexidine, 10 percent povidone-iodine for professional use and 0.1-0.5 percent sodium hypochlorite for patient self-care.[43,72,73]

Surgical Therapy

There is a general agreement that favorable therapeutic outcome can be achieved treating patients with localized aggressive periodontitis even with cases of severe periodontal attachment loss. The rationale for surgery among patients with aggressive periodontitis is in part related to the perceived need to remove tissue invaded by *A. actinomycetemcomitans* as well as *P. gingivalis*.[74-77] Because the presence of these species within epithelial cells may not be eliminated after nonsurgical and systemic

antibiotic therapy, it is plausible that recolonization may occur, which might contribute to recurrent or refractory disease.[78] Furthermore, since periodontal lesions associated with deep pockets, molars, furcation sites, and angular bone defects respond less favorably to repeated nonsurgical instrumentation, a surgical approach may be considered for debridement versus repeated nonsurgical instrumentation on the sites with evidence of inflammation after nonsurgical therapy.[79-81] Surgical techniques for root debridement have been successfully utilized to treat localized aggressive periodontitis in combination with systemic antibiotics with significant improvements in probing depths and attachment levels, and evidence of radiographic bone fill after five years of maintenance.[51,82-85] Also to repair bony defects, osseoinductive surgical procedures with the use of autografts, allografts, alloplast, with or without barrier membranes, have been reported with successful and favorable outcomes in treating localized aggressive periodontitis lesions.[86-92] Each of the above studies contained few subjects and defects, making comparisons between the groups difficult. In contrast. Gunsolley reported that among patients with localized aggressive periodontitis who received treatment, there was no difference in periodontal attachment gain over the 15-year period for those who received scaling and root planing alone compared to those who were treated surgically.[19]

The fact that different authors report favorable results using different nonsurgical as well as surgical modalities (including different bone graft materials and membranes and in different combinations) may suggest that the healing of aggressive periodontitis lesions may be related to the diagnosis, site-specific healing potential, the timing of the surgical intervention, and defect morphology rather than the choice of materials and surgical techniques. In choosing inductive techniques, it is important to consider the potential for post surgical complication of using barrier membranes. Sites with membrane show a significant variability in result due to potential of exposure and bacterial contamination. Furthermore, membrane contamination is highly associated with less favorable results.[93-95]

IN CHOOSING inductive techniques, it is important to consider the potential for post surgical complication of using barrier membranes.

The result from Nowzari et al. underscores the importance of full-mouth infection control prior to considering surgical repair of periodontal lesions.

In cases of generalized aggressive periodontitis, there is an overall reluctance among clinicians to perform surgical therapy. Some of the reasons for this reluctance are severe attachment loss on presentation, possible risk of unknown or undetected systemic disease, a history of unfavorable surgical outcomes with previous experiences, or a reluctance to perform surgery in patients with unknown prognosis and risk factors.[22] While a cautious approach to surgery in patients with generalized aggressive periodontitis is prudent, in sites where there is continuous clinical inflammation and deep pockets of >5 mm with bleeding on probing, a surgical treatment for root debridement such as Modified Widman flap with no osseous resection is indicated.[96] However, any resective surgery should be avoided at this stage to allow a potential for repair of osseous defects. Many authors have reported good success of osseous inductive surgery to repair intraosseous defects in patients with generalized aggressive periodontitis.[65,97-99] Once the infection is under control, osseous corrective surgery may be indicated to correct residual osseous defects during the long-term maintenance phase of the treatment. An apically positioned flap with osseous recontouring (osseous surgery) is a very effective approach in correcting remaining shallow to medium intraosseous defects, reducing pockets and subgingival detection of *A. actinomycetemcomitans*.[100,101] Resective types of periodontal surgery are more effective than access flap surgery in combating subgingival *A. actinomycetemcomitans* apparently due to the excision of *A. actinomycetemcomitans*-infected gingival tissue and pocket reduction to levels permitting adequate oral hygiene measures in the long-term maintenance of patients.[102]

In choosing corrective/recective surgical approaches, it is critical to observe bony defect anatomy and morphology as well as esthetic implications of such treatment modalities with an understanding of the risk-benefit outcome, the ultimate goals of periodontal therapy and reconstructive treatment needs for a particular patient.

Maintenance and Reconstructive Restorative Therapy

When clinical improvement is achieved with initial infection control, a two-month maintenance schedule is planned for the first six to nine months.

After nine months, radiographic evaluation of residual bony defects may be indicated to correct remaining osseous defects and deep pockets (>5 mm specially on posterior teeth). Based on the clinical outcome of therapy patient should be scheduled on a long-term maintenance interval of two to three months for a close monitoring of periodontal condition, especially in cases of generalized aggressive periodontitis. Once periodontal prognosis and stability are established, reconstructive periodontal, implant restorative therapy can be planned.

A flow chart is illustrated in **DIAGRAM 1** to provide a guideline using a dynamic therapeutic approach. Use of such approach is exemplified in periodontal treatment planning of the following cases of individuals affected with generalized or localized aggressive periodontitis. The quotation marks are used to reference patients own words of their history and chief complaints for case description.

Case 1

This case presents a 40-year-old African-American male from a North African origin with no systemic condition (nonsmoker ASA I). He reported to Department of Advanced Periodontology with a chief complaint of "loose front tooth" and esthetic concern about maxillary anterior diastema. His dental history consists of almost a lack of any professional dental visits since he never had any pain or "cavities." He reports of oral hygiene regiment of daily Miswak (a teeth cleaning device/brush made from a twig of the *Salvadora persica* tree) use. He reported of "loosening" of his teeth within the past five years and enlargement of the space between his maxillary anterior teeth. Furthermore, he presented a history of early edentulism of both parents at a similar age due to "loose teeth and gum infections." Despite the lack of professional dental visit there are no existing caries and no restorative therapy for any previously existed caries. He was also positive for red complex bacteria (*P. gingivalis, T. Forsythia, T. denticola*) and *A. Actinomycetemcomitans*.

Clinically, generalized gingival inflammation, generalized deep pockets of 5-10 mm with generalized bleeding on probing were present. Radiographically, generalized advanced horizontal bone loss, vertical bone loss, and circumferential intraosseous defects were present.

ONCE THE INFECTION is under control, osseous corrective surgery may be indicated to correct residual osseous defects during the long-term maintenance phase of the treatment.

Although there are detectable etiological factors (plaque and calculus) they are not abundant and not consistent with the extensive attachment loss and deep vertical and circumferential defects. This patient does not have any contributing systemic or other local factors (occlusal traumatism or severe malpositioning or crowding) that can be associated with extent of periodontal attachment loss. Either the attachment loss started early in his youth or has rapidly progressed through adulthood, which, in both cases, would be classified as aggressive periodontitis. Hence, this case is an example of generalized aggressive periodontitis (**FIGURE 1**).

Considering No. 8 root fracture, esthetic demands, restorative needs and advanced loss of attachment; prognosis of the maxillary dentition is poor. However, the mandibular arch does not require a functional or esthetic rehabilitation. Therefore, a reasonable treatment approach will be a transitional maxillary removable prosthesis and maintenance of mandibular teeth from Nos. 19 to 30 (**FIGURE 1**). The infection control stage will consist of:

- Full-arch maxillary extraction and delivery of an immediate transitional complete denture;
- Extraction of Nos. 17, 18, 30, and 31;
- Mandibular right and left quadrant scaling and root planing in a single session, one week after extraction of nonmaintainable/poor prognosis teeth;
- Systemic antibiotic therapy with amoxicillin and metronidazole (250 mg each one every eight hours for eight days);
- Oral hygiene instructions and 0.2 percent chlorhexidine for two weeks; and
- Periodontal re-evaluation six weeks post scaling and root planing followed by three-month periodontal maintenance.

Surgical therapy was not indicated since neither deep pocket depths (>5 mm), nor clinically detectable inflammation was detected after initial therapy. **FIGURE 1** demonstrates the clinical outcome with generalized probing depths of 2-3 mm and localized 4 mm probing on mesiolingual of No. 19 and distolingual of No. 29, one-year post-treatment. Implant therapy of maxillary arch may be considered at this stage, since periodontal infection is deemed under control.

Case 2

This case is presentation of a 35-year-old Asian female with no systemic condition (nonsmoker ASA I). The patient reported with the chief complaint of protruded maxillary anterior tooth No. 10 and diastema. She reported of a

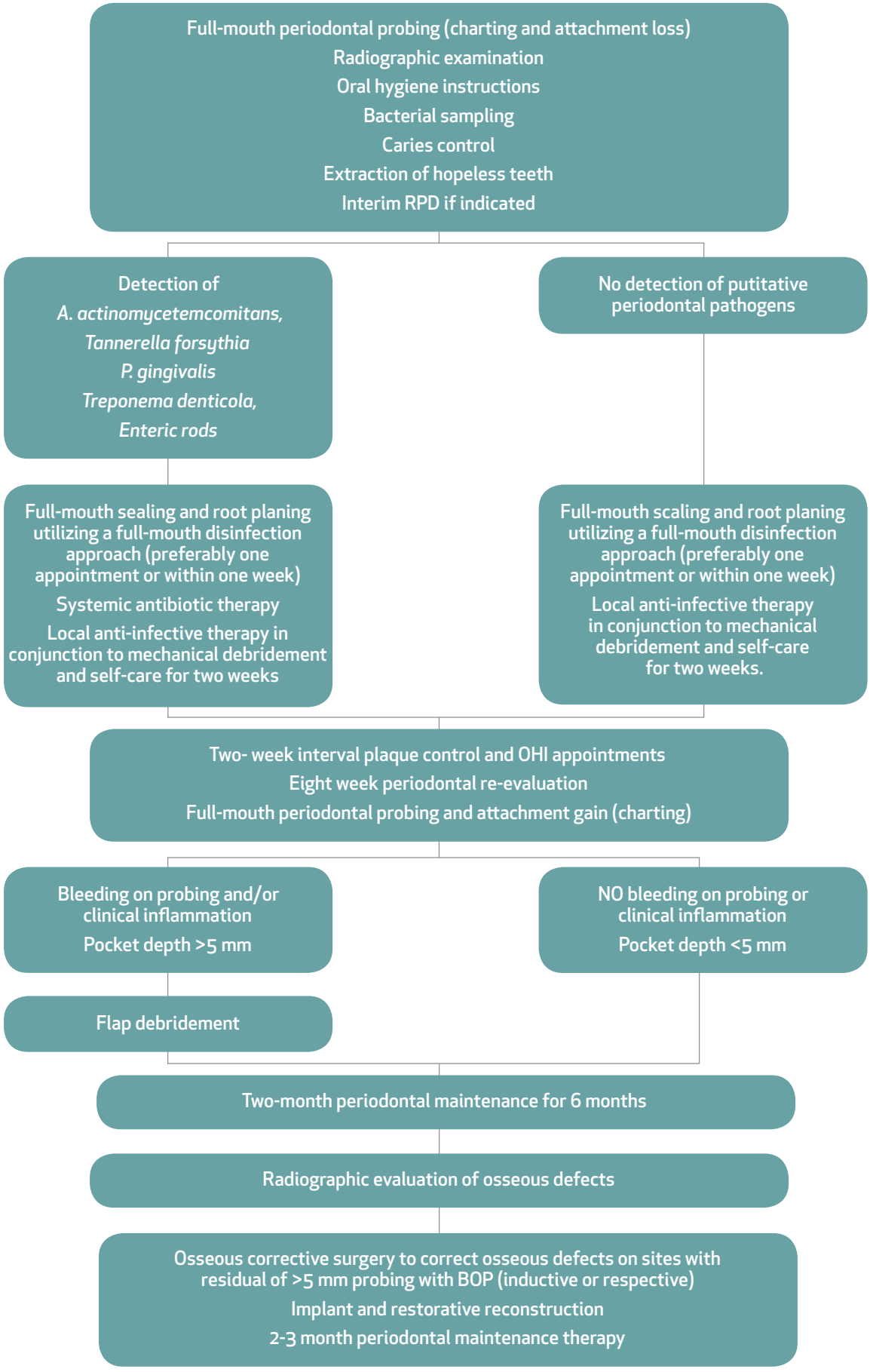

DIAGRAM 1. Dynamic therapeutic approach in treatment planning for individuals affected by aggressive periodontitis.

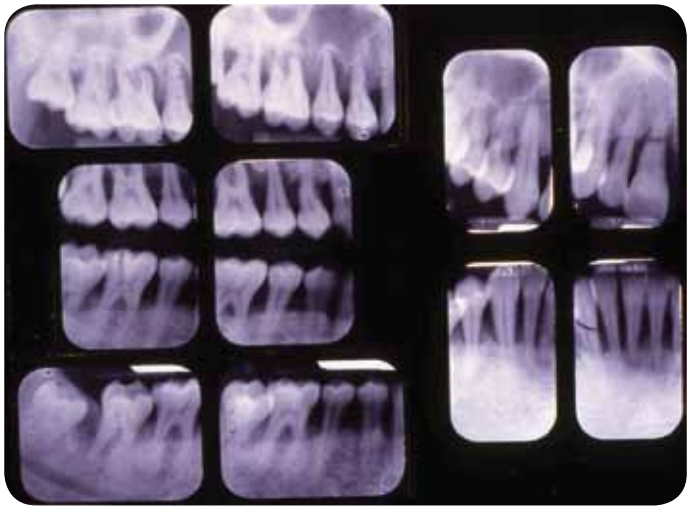

FIGURE 1A. Patient's right.

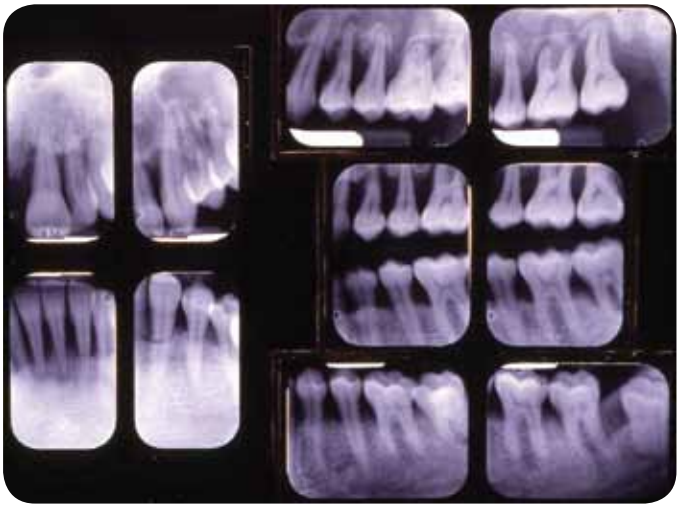

FIGURE 1A. Patient's left.

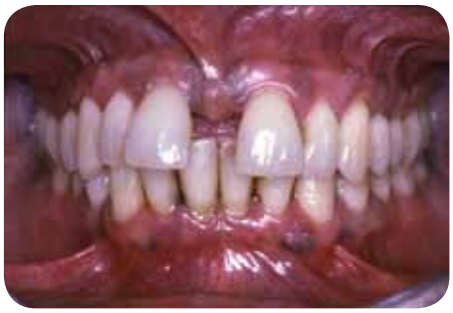

FIGURE 1B.

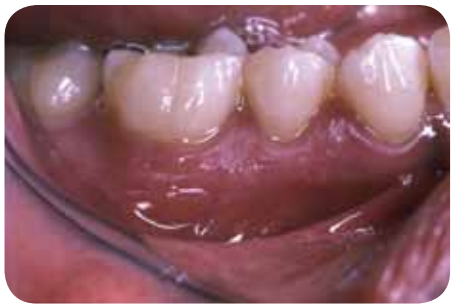

FIGURE 1C.

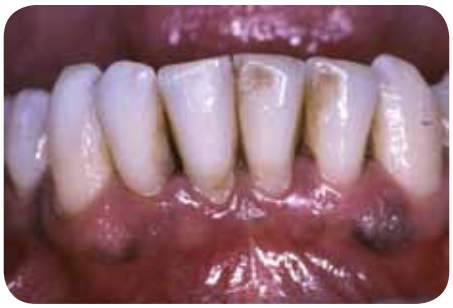

FIGURE 1D.

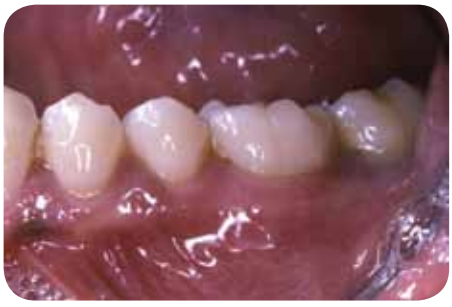

FIGURE 1E.

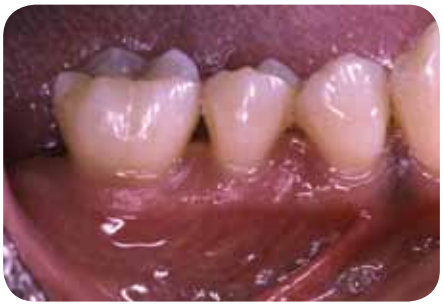

FIGURE 1F.

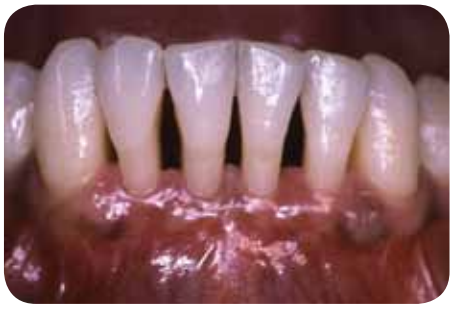

FIGURE 1G.

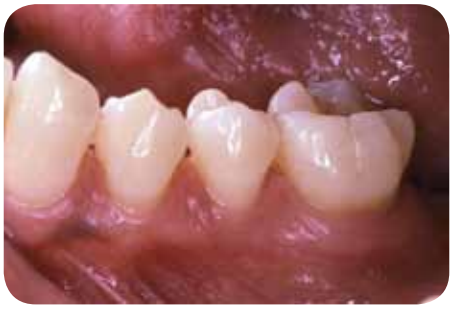

FIGURE 1H.

FIGURE 1. Clinical and radiographic presentation of case 1: Full-mouth preoperative radiographic (**A**) and clinical (**B**) presentation. Note lack of caries despite advanced periodontal infection. Preoperative clinical presentation of mandibular arch (**C, D AND E**) and one-year postoperative clinical presentation (**F, G, AND H**).

"gum infection" on her lower front teeth when she was in her late teens. She also reported that within the past year she had noticed an increase in maxillary anterior spacing between Nos. 9 and 10 and flaring of No. 10. Additionally tooth No. 31 was recently removed due to "gum abscess." There was a history of sporadic dental treatment and "a general cleaning" a few years prior to her periodontal consultation. She presented with severe

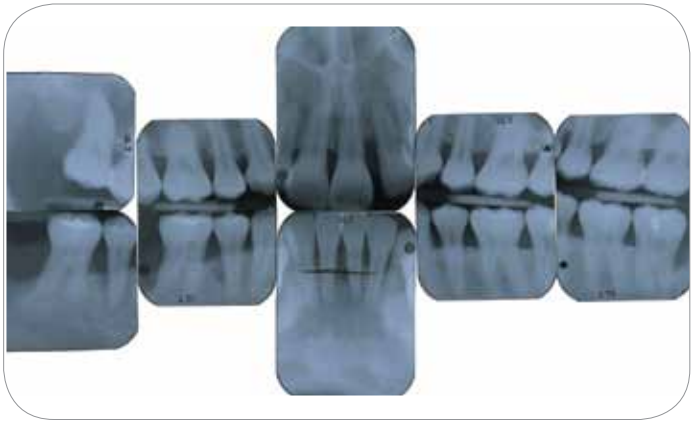

FIGURE 2A.

FIGURE 2B.

FIGURE 2C.

FIGURE 2. Radiographic and clinical presentation of case 2: Preoperative (**A**) and three-year postoperative radiographic (**B**) presentation of maxillary and mandibular teeth. Note radiographic bone gain and re-establishment of lamina dura of Nos. 2, 3, 10, 14, 18, 24, 25, 29, and 30 following resolution of infection and orthodontic therapy. (**C**) Clinical presentation of pre- and postorthodontic therapy to correct diastema and alignment of the teeth. *(Orthodontic treatment courtesy of James Clark, DDS, Mission Viejo, Calif. Restorative evaluation courtesy of Christopher Travis, DDS, Laguna Hills, Calif.)*

attachment loss on anterior and first molars and a generalized mild-to-moderate attachment loss. There were patterns of site-specific infection that may be correlated to presence of local etiological factors. However, despite the lack of professional dental care and inadequate home care (O'Leary plaque index of 100 percent) no caries or restorative treatment for previously existing caries were present. Initially generalized bleeding on probing with pocket depths of 6-10 mm and areas of moderate to advanced horizontal and vertical osseous defects were detected (**FIGURE 2**). Her bacterial sampling results were negative for red complex bacteria *(P. gingivalis, T. Forsythia, T. denticola)* enteric gram-negative rods and *A. actinomycetemcomitans.* Given her history and pattern of disease, her periodontal diagnosis was classified as a history of localized aggressive periodontitis that was transforming to a generalized form of chronic periodontitis.

Her treatment plan consisted of:

- Oral hygiene instruction (initial plaque index of 100 percent);
- Bacterial sampling;
- Four quadrants of scaling and root planing in two appointments within one week. No systemic antibiotic were administered since no *A. actinomycetemcomitans, P. gingivalis, T. Forsythia, T. denticola,* or enteric gram-negative rods were detected by microbial testing;
- Two weeks rinse with 0.2 percent chlorhexidine, re-evaluation for plaque control and OHI;
- Periodontal re-evaluation six weeks post initial scaling and root planing for pocket depth and attachment gain;
- Flap debridement No. 29 distal due to 7 mm pocket post scaling and root planing with bleeding on probing, suppuration, persistent gingival inflammation and intraosseous defect;
- No. 18 autogenous bone graft to correct distal 2-3 wall intraosseous defect;
- Nos. 14 and 15 palatal approach osseous surgery to correct shallow interproximal crater for pocket reduction and flap debridement of No. 4 to repair mesial vertical intraosseous defect; and
- Orthodontic therapy to realign teeth and reduce maxillary anterior diastema one year after periodontal therapy and assessment of periodontal stability.

At three-year postoperative evaluation, generalized 2-3 mm probing depths, clinically healthy gingiva, radiographic resolution of osseous defects, and re-establishment of a clear lamina dura indicating periodontal stability. At this stage the patient is ready for definitive restorative plan, which includes:

- Bonded restorations to contour inter proximal contact of Nos. 8, 9 and 10; and
- Implant-supported restoration No. 31 to increase occlusal support and masticatory function.

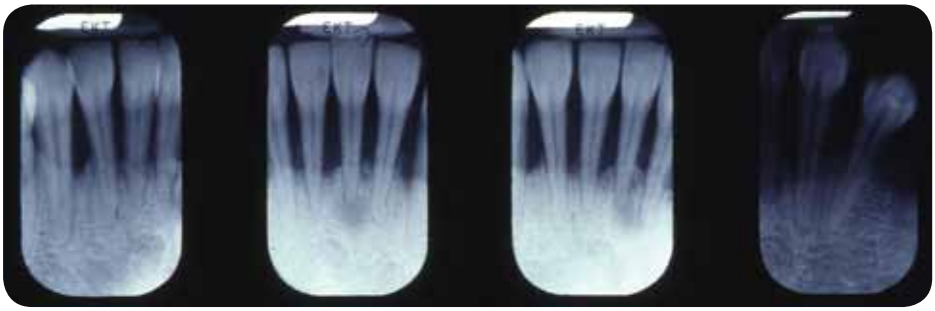

FIGURE 3A.

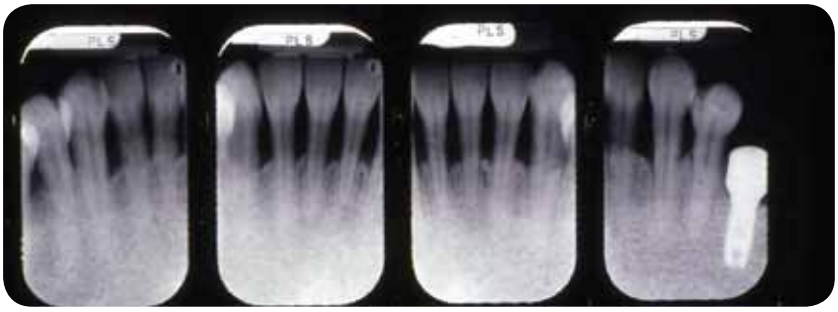

FIGURE 3B.

FIGURE 3. Radiographic presentation of case 3: Preoperative (**A**) and seven-year postoperative (**D**) presentation of mandibular anterior teeth. Note radiographic bone gain following resolution of infection.

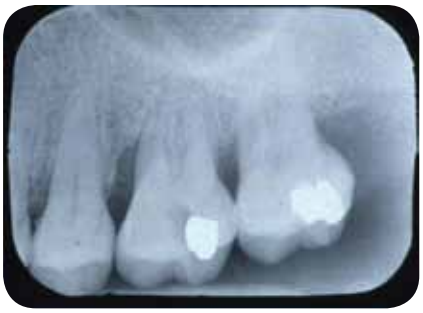

FIGURE 4A.

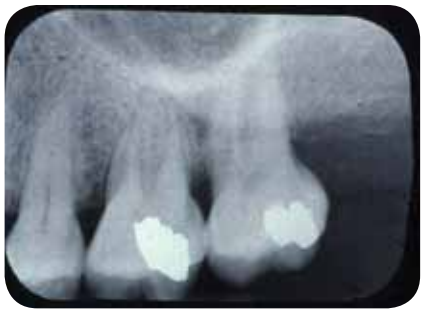

FIGURE 4B.

FIGURE 4. Preoperative (**A**) and postoperative (**B**) radiographic presentation of case 4: Note resolution of mesial vertical intraosseous defect of tooth No. 14 and interproximal craters of No. 12, 13, 14, and 15 one year following periodontal osseous surgery and flap debridement.

Case 3

Clinical and radiographic appearance of mandibular anterior sextant of a 28-year-old female affected with generalized aggressive periodontitis is presented on **FIGURE 3**. Putative periodontal pathogens *A. actinomycetemcomitans* and *T. forsythia* were detected by microbial sampling. Periodontal treatment included scaling and root planing in conjunction with amoxicillin and metronidazole. No surgical therapy was performed in this sextant since initial therapy resulted in reduction of probing depths and resolution of periodontal inflammation. Seven-year postoperative evaluation demonstrates radiographic bone repair and disappearance of periapical periodontitis of mandibular anterior teeth. At this point, the patient was ready to proceed with implant therapy to replace posterior missing teeth.

Case 4

The case presented on **FIGURE 4** exemplifies an adult patient (46-year-old female) with a history of localized aggressive periodontitis, which had been self-limiting and transformed to a secondary chronic periodontitis. There is a site-specific infection by gram-negative rods affecting mesial of tooth No. 14. Treatment for this patient included initial scaling and root planing, systemic antibiotic therapy for initial infection control followed by maxillary left osseous surgery to correct residual osseous craters between Nos. 12, 13, 14 and 15, as well as No. 14 vertical intraosseous defect. Postoperative radiographic appearance demonstrates periodontal healing and repair of osseous defects.

Case 5

The case presented on **FIGURE 5** exemplifies a 32-year-old female with a history of localized aggressive periodontitis. No other area of attachment loss was detected in this patient. There is a site-specific infection by *P. gingivalis* affecting mesial of tooth No. 19. Treatment for this patient included initial scaling and root planing and systemic antibiotic therapy for initial infection control. Upon re-evaluation, bleeding on probing, and residual pocket depth indicated a need for flap debridement of No. 19 mesial intraosseous defect. Surgical photograph indicates remaining calculus on mesial of No. 19 post scaling and root planing. One-year clinical and radiographic appearance demonstrated periodontal healing and repair of osseous defect.

Case 6

This case is presentation of a 24-year-old African-American male He is systemically healthy with no known systemic contributing factors (nonsmoker ASA I). This is a young individual with severe attachment loss and nonmaintainable

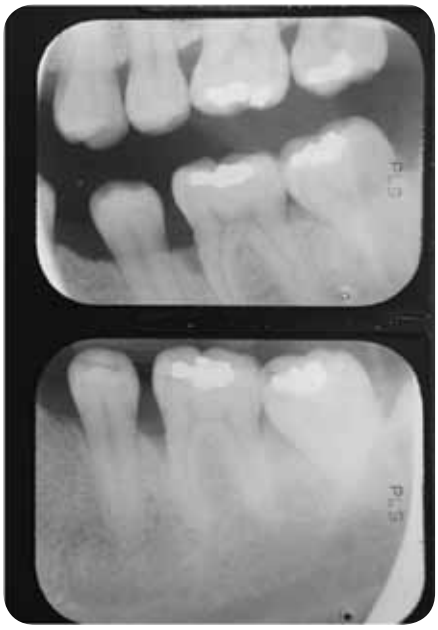

FIGURE 5A.

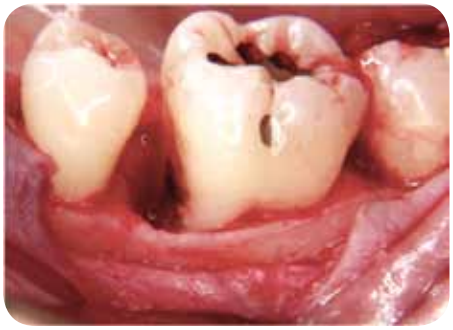

FIGURE 5B.

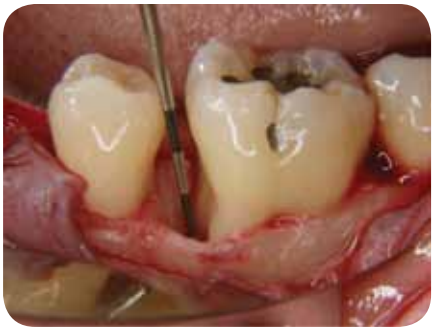

FIGURE 5C.

FIGURE 5. Clinical and radiographic presentation of case 5: Preoperative (**A**) radiograph, flap debridement, and removal of residual calculus (**B AND C**), one-week postoperative healing (**D**), and one-year postoperative clinical and radiographic presentation (**E AND F**). Note soft-tissue healing and radiographic bone gain.

FIGURE 5D.

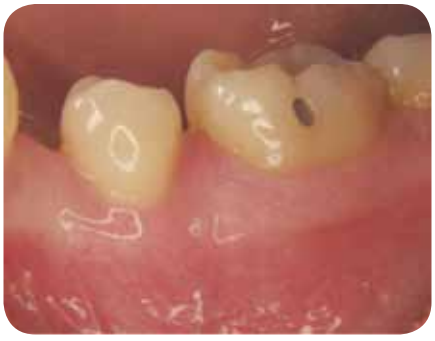

FIGURE 5E.

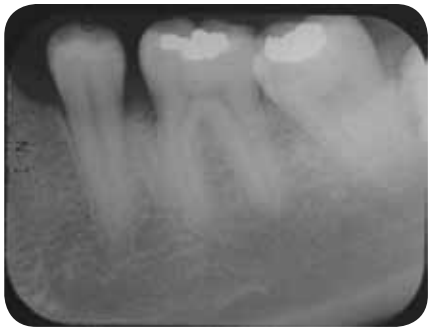

FIGURE 5F.

teeth: Nos. 3, 4, and 5. A fixed partial denture was previously fabricated to replace No. 7 that was previously removed two to three years prior to periodontal consultation because of "looseness" and "gum infection." Initially generalized bleeding on probing with generalized pocket depths of 5-10 mm and areas of moderate to advanced horizontal and vertical osseous defects were detected (**FIGURE 6**). His periodontal condition is classified as generalized aggressive periodontitis.

Loss of posterior maxillary right molar and premolar teeth as well as potential loss of Nos. 6 and 14 created both functional and esthetic restorative demands. To consider a reconstructive plan for this patient, control and stability of periodontal disease condition is essential. A transitional removable prosthesis would be considered during periodontal therapy as a provisional replacement of the missing teeth. His treatment plan includes:

- Oral hygiene instruction (initial plaque index of 100 percent);
- Bacterial sampling;
- Extraction of Nos. 3, 4, and 5;
- Four quadrants of scaling and root planing in two appointments within one week and intraoperative 10 percent povidone-iodine irrigation due to severity of gingival inflammation and therefore a perceived need for local antibacterial therapy;
- Systemic antibiotic therapy (ciprofloxacin 500 plus metronidazole 500 mg two times a day for eight days) since *T. forsythia* and enteric gram-negative rods were detected by microbial testing;
- Two weeks re-evaluation for plaque control and OHI for two months;
- Periodontal re-evaluation in six weeks post scaling and root planing for pocket depth and attachment gain;
- Flap debridement of mandibular right and mandibular left quadrants due to residual 5-7 mm pockets post scaling and root planing with bleeding on probing, suppuration, and persistent gingival inflammation.

Implant therapy will be considered after evaluation of resolution of peri-

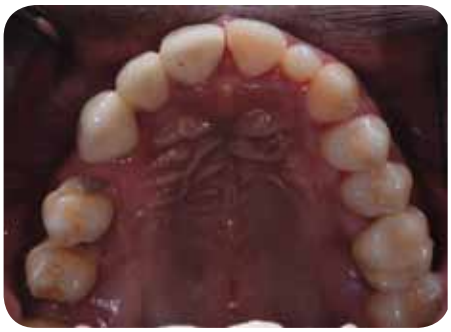

FIGURE 6A.

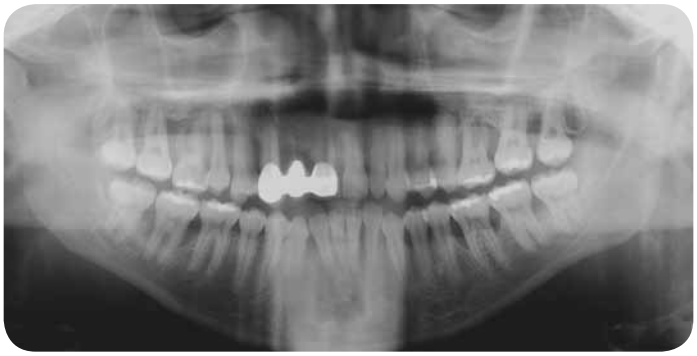

FIGURE 6B.

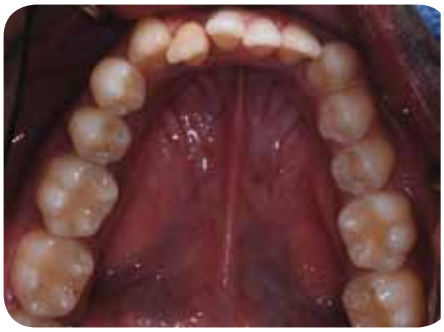

FIGURE 6C.

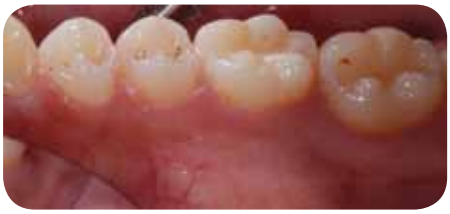

FIGURE 6D.

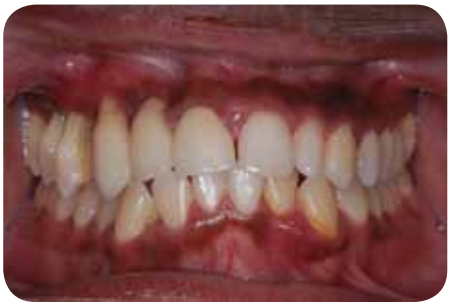

FIGURE 6E.

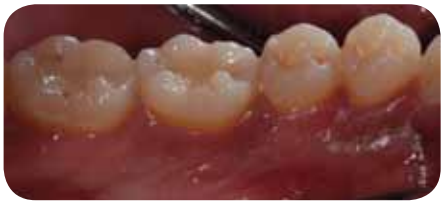

FIGURE 6F.

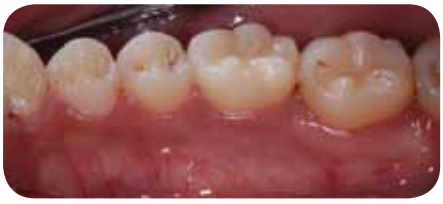

FIGURE 6G.

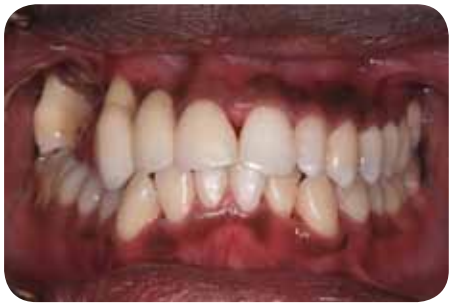

FIGURE 6H.

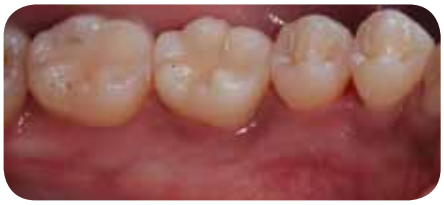

FIGURE 6I.

FIGURE 6. Clinical and radiographic presentation of case 6: Preoperative occlusal view (**A AND C**) and panoramic radiograph (**B**). Note lack of caries despite severe periodontal attachment loss. (**D**) mandibular right lingual; (**E**) retracted anterior intercuspal occlusion; (**F**) mandibular left lingual preinitial therapy; (**G, H, AND I**) postinitial therapy indicating overall resolution yet localized residual inflammation. *(Courtesy of Yvonne Tam DDS, USC Periodontology resident.)*

odontal inflammation, radiographic resolution of bony defects. This final assessment may be completed after a period of 12 months to evaluate potential breakdown of the periodontal condition.

Conclusion

Aggressive periodontitis presents with a diverse clinical manifestation. It is associated with various bacterial, viral, immunological, and systemic cofactors. Therefore, treatment outcome is very much dependent on the manifestation of disease, the patient's age, as well as functional and esthetic demands. To address a patient's treatment, the clinician needs to employ an approach tailored to each individual's clinical manifestation and historical background. When choosing a treatment modality, a "dynamic therapeutic approach" will provide flexibility in adapting decision-making to the outcome of every diagnostic evaluation and treatment modality during each phase of therapy. Ultimately, interdisciplinary communication among different providers is the key element in successful management of patients affected by aggressive periodontitis. ■■■■

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CDA JOURNAL, VOL 39, Nº6

Peri-Implant Management of Patients With Aggressive Periodontitis

WINSTON CHEE, DDS, FACP

ABSTRACT Peri -implant mucositis has been a common clinical finding around implant-supported restorations.[1] More recently, several publications have documented progressive bone loss around implants.[4-8] A peri-implant tissue maintenance protocol seems essential to maintain health postimplant therapy among patients affected with aggressive periodontitis.

AUTHOR

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As recently as 2009, recognized experts have described peri-implantitis as a manufactured disease or a local nuisance event and not a disease process.[2,3] However, there is a growing body of evidence that indicates that peri-implantitis is a disease entity and that patients with chronic periodontitis or aggressive periodontitis at the time of implant placement are more prone to exhibiting these lesions.[4-10]

In 1991, Slots and Rosenberg evaluated the microflora associated with osseointegrated implants.[11] Failed implants were retrieved from both partially dentate and fully edentulous patients. Arbitrarily etiology of failure was attributed to infection if there was bleeding, suppuration, high plaque and gingival indices, and granulomatous tissue upon surgical removal. In absence of these findings, failures were considered traumatic failures. In their publication, they found distinct differences in the microbiota of the two different types of failures with infectious failures having a preponderance of spriochetes and motile rods. **FIGURES 1 AND 2** illustrate typical clinical presentations of the two types of failures. **FIGURE 1** illustrates clinical findings of swelling and inflammation, and when the implant is removed it is covered with granulomatous tissue. **FIGURE 2** illustrates "traumatic" failure where intraoral tissues appear relatively healthy and implants removed appear "clean."

In 2003, Karoussis et al. reported on long-term implant prognosis in patients with and without a history of chronic periodontitis.[9] This was a 10-year prospective study of the ITI Dental Implant system. They found that the survival rate for patients with a history of chronic periodontitis was 90.5 percent, and without a history of

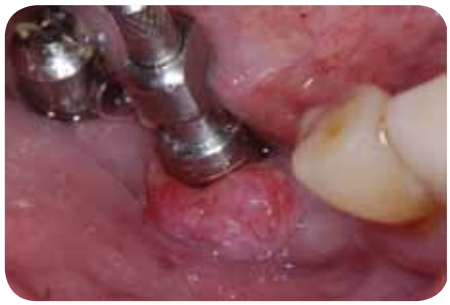

FIGURE 1A. Intraoral image of peri-implant soft tissue of implant with bone loss exhibiting swelling and discoloration.

FIGURE 1B. Implant in **FIGURE 1A** removed. Note the granulation tissue on implant.

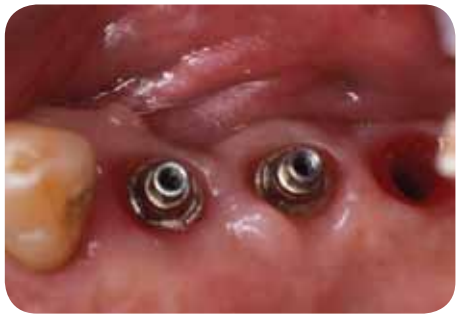

FIGURE 2A. Intraoral image of socket of failed implant. Note that tissues appear normal compared to implants that have successfully integrated.

FIGURE 2B. Implant in from wound in **FIGURE 2A.** Note the relatively clean surface of the implant.

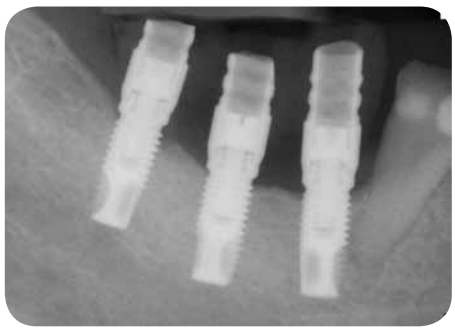

FIGURE 3A. Radiograph of implants evaluating the fit of the provisional restoration.

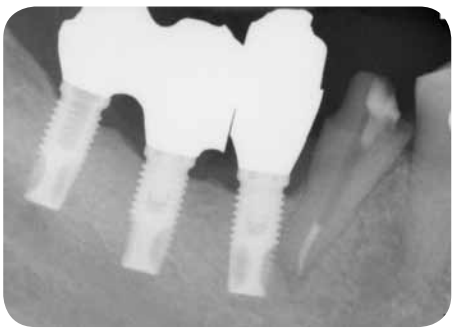

FIGURE 3B. Radiograph of implants in **FIGURE 3A** during framework try-in with anterior implant sectioned for solder.

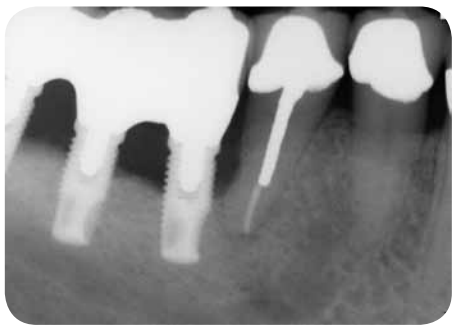

FIGURE 3C. Completed restorations with some evidence of bone loss.

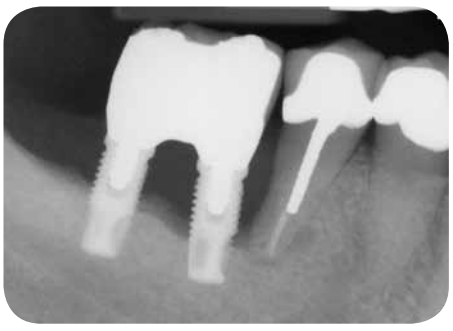

FIGURE 3D. Distal implant has been sectioned and removed due to peri-implant bone loss. Note the bone loss on anterior implants has progressed.

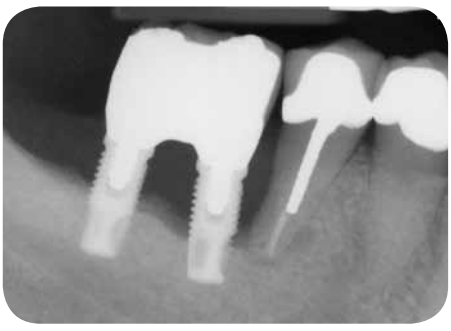

FIGURE 3E. Bone loss has continued and the anterior most implant has complete bone loss to the apex.

chronic periodontitis was 96.5 percent. Additionally, more biological problems such as bleeding on probing and more peri-implant bone loss were demonstrated on patients with a history of chronic periodontitis.

More recently, Roos-Jansaker, in a retrospective nine to 14 years postrestoration examination of 218 patients with 1,057 implants where Brånemark implants were used, found a significant relationship between implant loss and periodontal bone loss of remaining teeth at time of implant placement.[4-6] Early implant failures were attributed to causes other than peri-implantitis and later failures (after delivery of definitive prostheses) were considered losses due to peri-implantitis. As an example, **FIGURE 3** illustrates progressive loss of bone surrounding implants over a three-year time period.

Patients with aggressive periodontitis suffer severe attachment loss of affected teeth. When these teeth are replaced, often the treatment of choice will be implant-supported restorations. Implant-supported restorations can replace missing teeth without incorporating the remaining dentition. Moreover, implant-supported restorations can provide occlusal support to reduce occlusal forces on the remaining dentition.

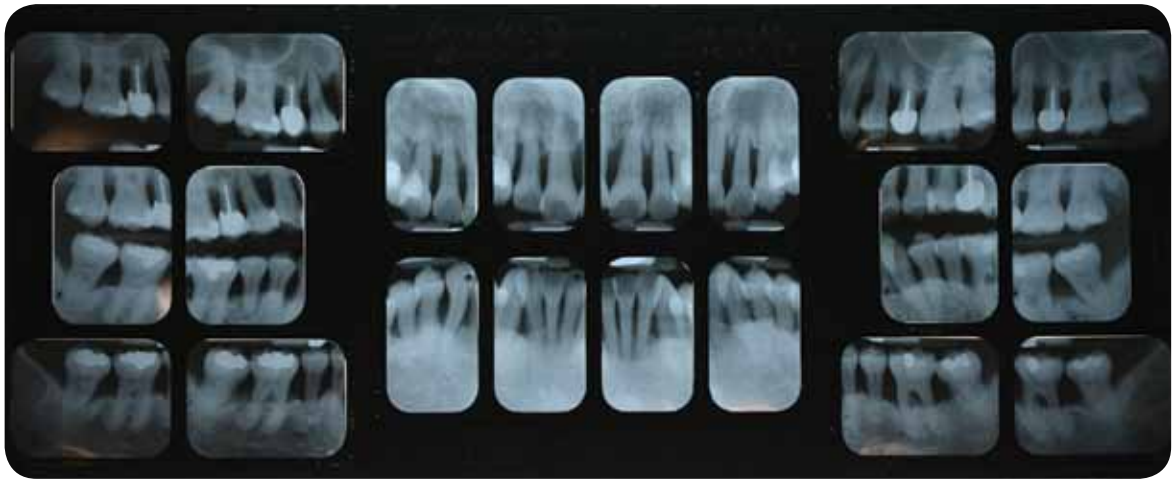

FIGURE 4A. Typical full-mouth radiographs of patient with aggressive periodontitis of long standing.

Frequently, when patients were restored with dental implants, clinicians would mostly focus on mechanical issues such as screw loosening, mechanical breakage of restorations, or breakage of implant components.[12,13] It was commonly thought that with no calcified tissues to decay and no periodontium to infect that implant-supported restorations were immune to the bacterial insults in the oral environment. In fact, this is the sentiment expressed by some leaders in the field of implant dentistry.[2,3]

Currently this thinking may only apply to patients who have lost teeth due to trauma or decay. Therefore, dental implant maintenance of patients, who lost teeth primarily due to caries or trauma, can be less stringent than those who lost teeth due to periodontal disease particularly to aggressive periodontitis.

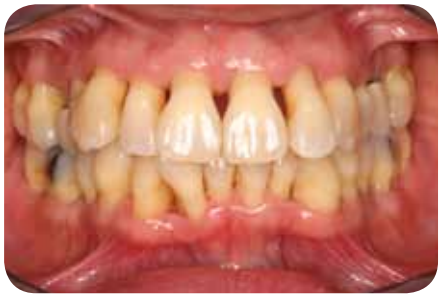

FIGURE 4B. Clinical presentation of patient in **FIGURE 4A.**

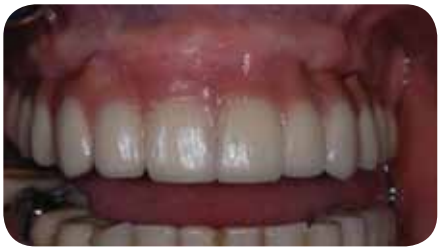

FIGURE 5. Intraoral view of implant-supported restoration with soft-tissue analog; this can compromise oral hygiene procedures.

Amongst individuals with generalized aggressive periodontitis and advanced attachment loss, it is not uncommon to assume a poor prognosis for all or most of the teeth. When treatment planning for these individuals, most clinicians will proceed with periodontal infection control including scaling and root planing concomitant with bacterial culture, systemic antibiotics therapy together with intraoral use of chlorhexidine, sodium hypochlorite, and iodine. After this initial stage there is usually a re-evaluation of periodontal infection control and a reassessment of prognosis.

Often times a decision may be made that due to functional and esthetic demands the dentition is nonmaintainable.[14] Hence, two restorative options are available: either removable prosthesis

FIGURE 6A. Intraoral view of implants placed to support tissue bar.

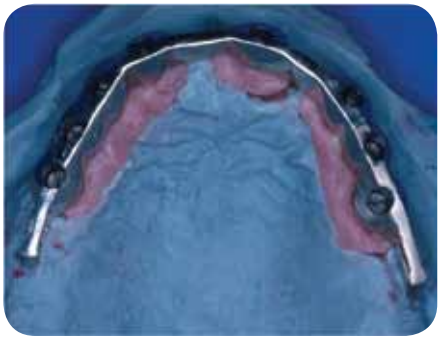

FIGURE 6B. Framework to support overdenture prostheses fitting over implants in **FIGURE 6A.**

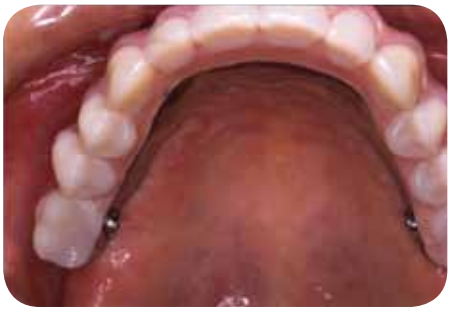

FIGURE 6C. Intraoral view of implant-supported overdenture that can compromise oral hygiene procedures around implants.

FIGURE 7. Intraoral view of implants after resective therapy due to peri-implant bone loss; poor esthetics is evident.

(occasionally with some type of implant-supported overdenture) or implant-supported fixed restoration. Either option will provide a challenge for oral hygiene maintenance. Not only the contours of the replaced teeth but also often the analogs used to improve esthetics of lost hard and soft tissues will hinder oral hygiene access and maintenance (**FIGURES 4-7**).

Once these restorations are delivered, the patient must undergo a strict oral hygiene regimen at home and maintain a rigid three-month interval maintenance program. Any signs of increased probing depth, bleeding on probing, and peri-implant mucositis must be actively investigated. A radiograph should be taken to evaluate for any peri-implant bone loss. When bleeding on probing and mucositis are detected, home care must be re-enforced, submucosal mechanical plaque control and submucosal application of local antiseptic agents such as iodine, sodium hypochlorite, and chlorhexidine or local delivery antibiotics should be initiated and a re-evaluation should be carried out after a one- to two-week period.[15] If those steps fail to control mucosal inflammation, bacterial sampling, and systemic antibiotic therapy may be indicated when putative periodontal pathogens and/or enteric rods are detected, especially if peri-implant bone loss may be evident. Surgical mucosal contouring and submucosal debridement may also be considered to manage persistent peri-implant mucositis.

The treatment of peri-implant bone loss has not reached consensus but common sense will dictate that a conservative approach should be undertaken initially and resective therapy only performed if conservative approaches fail. Resective procedures to eliminate soft-tissue pockets can be disfiguring and lead to compromised esthetics (**FIGURE 8**). Conservative approaches that have been proposed range from irrigation with saline to use of lasers, some are supported by animal studies and some are reported in case series or anecdotally.[16-21] If no treatment is performed, the peri-implant bone loss can proceed causing loss of the implant (**FIGURE 3**).

Conclusion

Dental restoration of patients suffering from aggressive periodontitis can be predictably completed with implant-supported or implant-assisted prostheses. However, there is a higher incidence of peri-implant diseases and less favorable long-term survival rate among this population when compared to patients without a history of aggressive periodontitis. In view of current information, any patient with classification of aggressive periodontitis (or severe forms of chronic periodontitis) who is subsequently restored with dental implants should be considered high risk for peri-implant diseases compared to patients who have lost teeth due to caries or trauma. A rigid three-month maintenance protocol should be implemented to intervene with a conservative treatment when signs of mucositis are detected. ■■■■

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Continuing Education Courses

Listed are C.E. courses offered by California's dental schools, local dental societies, ethnic dental societies and specialty organizations, from June through December 2011. For more information, please contact the course provider.

TOPIC	DATE	LECTURER(S)	LOCATION	COST	UNITS
ARTHUR A. DUGONI SCHOOL OF DENTISTRY					**dental.pacific.edu/ce1**
Certification in Radiation Safety for Allied Dental Professionals	July 16; Aug. 13	Gurminder Sidhu, BDS, MS, DDS	San Francisco	$625	32
Hospital Dentistry	July 23-24	Paul Glassman, DDS,MA, MBA; Allen Wong, DDS, EdD, FACD, FICD	San Francisco	$335 Dentist/ $295 Auxiliary	13.5
Culture and Dental Ethics	Aug. 12	Bruce Peltier, PhD, MBA	San Francisco	$75	3
Certification in Radiation Safety for Allied Dental Professionals	Oct. 15, 29	Gurminder Sidhu, BDS, MS, DDS	San Francisco	$625	32
Digital Photography Workshop	Oct. 22	Marc J. Geissberger, DDS, MA	San Francisco	$395 Dentist/ $195 Auxiliary	6
Infection Control and the Dental Practice Act	Nov. 5	Eve Cuny, BA, MS; Bruce Peltier, PhD, MBA	San Francisco	$125	4
CALIFORNIA ACADEMY OF GENERAL DENTISTRY					**caagd.org**
Laser Dentistry Workshop	Aug. 19	Robert Convissar, DDS	San Jose	$195 Member/ $245 Non-member	8
Esthetic Restorative Dentistry and Innovative Color Control Workshop	Sept. 18	Roger Garrett, DDS; Uri Yaroevesky, CDT	Garden Grove	$195 Member/ $245-$275 Non-member	8
Atraumatic and Efficient Exodontia: New Concepts and Technology	Nov. 19	Karl R. Koerner, DDS	Anaheim	$85	8
Orthodontics by GP's for Children and Adults	Dec. 4	Rob Veis, DDS	Garden Grove	$195 Member/ $245-$275 Non-member	8
CENTRAL COAST DENTAL SOCIETY					**centralcoastds.org**
OSHA, Infection Control and the Dental Practice Act	Sept. 16	LaDonna Drury-Klein, RDH	Embassy Suites Hotel, San Luis Obispo	$240 Member/ $75 Hygienist/ $60 Staff/ $480 Non-member/ $150 Non-member Hygienist/$120 Non-member Staff	6
Oral Pathology	Oct. 21	Bill Carpenter, DDS; Sol Silverman, DDS	Embassy Suites Hotel, San Luis Obispo	$240 Member/ $75 Hygienist/ $60 Staff/ $480 Non-member/ $150 Non-member Hygienist/$120 Non-member Staff	7

TOPIC	DATE	LECTURER(S)	LOCATION	COST	UNITS
FRESNO-MADERA DENTAL FOUNDATION					**fmdentalfoundation.org**
The Art of Endodontics and the Use of the Microscope in Surgical Endodontics	July 8	Anthony Tran, DDS	Fresno	$140 Member/ $170 Non-member/ $90 Auxiliary	7
Cone Beam Computerized Tomography	Sept. 9	Gurminder Sidhu, DDS	Fresno	$150 Member/ $190 Non-member/ $100 Auxiliary	7
Implant Prosthodontics	Oct. 7	Baldwin Marchack, DDS	Fresno	$140 Member/ $170 Non-member/ $90 Auxiliary	7
Update on Human Nutrition and the Oral Systemic Connection	Nov. 11	William Carpenter, DDS, MS; Charles Carpenter, MD	Fresno	$140 Member/ $170 Non-member/ $90 Auxiliary	7
HARBOR DENTAL SOCIETY					**harbordentalsociety.org**
Interactive Treatment Planning	Sept. 15	Ravipan Smith, DDS; Simona Arcan, DMD, MD	Lakewood	$175 Member/ $60 Hygienist/ $40 Auxiliary	3
Everything is Marketing	Oct. 21	Fred Joyal	Lakewood	$175 Member /$60 Hygienist/ $40 Auxiliary	7
OSHA, Infections Control, California Dental Practice Act	Nov. 18	Marcella Oster, RDA	Lakewood	$175 Member/ $60 Hygienist/ $40 Auxiliary	7
HERMAN OSTROW SCHOOL OF DENTISTRY OF USC CONTINUES ON NEXT PAGE					**uscdentalce.org**
Clinical Intravenous Sedation	July 7-10, 15-17	Stanley Malamed, DDS; Faculty	Los Angeles	$11,450	49
Avoiding and Managing Complications Associated with Implant Therapy: Lecture and Impact Panel	July 16	Bach Le, DDS, MD, FICD; Faculty	Los Angeles	$375	8
Esthetic Full-Mouth Implant Reconstruction: Advanced Prosthodontic Techniques for Challenging Patients (Module I, II and III)	July 22-24	Harel Simon, DMD; Faculty	Los Angeles	$1,945 Dentist/ $1,595 Auxiliary	21
Esthetic Full-Mouth Implant Reconstruction: Advanced Prosthodontic Techniques for Challenging Patients (Module I)	July 22	Harel Simon, DMD; Faculty	Los Angeles	$275	7
Esthetic Full-Mouth Implant Reconstruction: Advanced Prosthodontic Techniques for Challenging Patients (Module II)	July 23	Harel Simon, DMD; Faculty	Los Angeles	$275	7
Esthetic Full-Mouth Implant Reconstruction: Advanced Prosthodontic Techniques for Challenging Patients (Module III)	July 24	Harel Simon, DMD; Faculty	Los Angeles	$1,795	7
Simplifying Anterior Restorations: Problem Solving in the Esthetic Zone (Part I - Lecture)	July 29	Abdi Sameni, DDS; Faculty	Los Angeles	295	7
Simplifying Anterior Restorations: Problem Solving in the Esthetic Zone (Parts I and II — Lecture and Hands-On Workshop)	July 29-31	Abdi Sameni, DDS; Faculty	Los Angeles	$1,995 Dentist	21
37th Annual Review of Continuing Education in Dentistry	Aug. 1-4	Avishai Sadan, DMD; Sillas Duarte, DDS	Maui, Hawaii	$595	16
Clinical Intravenous Sedation	Aug. 4-7, 12-14	Stanley Malamed, DDS; Faculty	Los Angeles	$11,450 Dentist	49

TOPIC	DATE	LECTURER(S)	LOCATION	COST	UNITS
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New Approaches for Antimicrobial Treatment of Periodontal Disease	Aug. 26	Jørgen Slots, DDS, DMD, PhD, MS, MBA	Los Angeles	$255 Dentist/ $175 Auxiliary	7
The Artistic Dentist/ Excellence in Direct Anterior and Posterior Composites	Aug. 19-20	Jose-Luis Ruiz, DDS, FAGD; Faculty	Los Angeles	$1,295 Dentist/ $495 Auxiliary	7
Mastering Bone Grafting for Implant Site Development (Module I)	Aug. 27	Bach Le, DDS, MD, FICD; Faculty	Los Angeles	$1,195 Dentist/ $595 Auxiliary	8
Mastering Bone Grafting for Implant Site Development (Module II - Cadaver Workshop)	Aug. 28	Bach Le, DDS, MD, FICD; Faculty	Los Angeles	$1,765 Dentist/ $995 Auxiliary	7
Fundamentals of Implant Surgery and Restoration	Sept. 9-11, Oct. 1-2, Nov. 5-6	Homayoun H. Zadeh, DDS, PhD; Faculty	Los Angeles	$4,395 Dentist/ $2,195 Auxiliary	56
Team Driven Diagnosis, Treatment Planning and Acceptance for a Successful Esthetic Practice	Sept. 23-24	Jose-Luis Ruiz, DDS, FAGD; Faculty	Los Angeles	$595 Dentist/ $325 Auxiliary	14
Pain Medications Update for Dentists Treating Chronic Pain and TMD	Sept. 30-Oct. 1	Glenn Clark, DDS, MS; Faculty	Los Angeles	$495Dentist/ $315 Auxiliary	14
The USC Fourth International Restorative Dentistry Symposium	Sept. 30-Oct. 1	Abdi Sameni, DDS; Faculty	Los Angeles	$495 Dentist/ $325 Auxiliary	14
The USC Fourth Geriatric Dentistry Symposium	Oct. 14-15	Roseann Mulligan, BA, DDS, MS, FADPD, DABSCD; Faculty	Los Angeles	$425 Dentist/ $265 Auxiliary	14
Esthetic Periodontal Surgery for the General Practitioner (Module I - Lecture)	Oct. 21	Ziv Simon, DMD, MSc	Los Angeles	$295 Dentist/ $175 Auxiliary	7
Esthetic Periodontal Surgery for the General Practitioner (Module I and II - Lecture and Hands-On Workshop)	Oct. 21-23	Ziv Simon, DMD, MSc	Los Angeles	$1,795 Dentist	21
Emerging Diseases, Infection Control and California Dental Practice Act	Oct. 22	Joyce Galligan, RN, DDS; Patricia Galligan, JD	Los Angeles	$190 Dentist/ $145 Auxiliary	6
Supra-Gingival Dentistry Workshop: Easy, Predictable Porcelain Veneer Onlays and Full Crowns	Oct. 28-29	Jose-Luis Ruiz, DDS, FAGD; Faculty	Los Angeles	$1,850 Dentist	14
Esthetic Full-Mouth Implant Reconstruction: CAD/CAM Restorations and Computer Guided Technology (Module I, II and III)	Nov. 11-13	Harel Simon, DMD; Faculty	Los Angeles	$1,945 Dentist/ $1,595 Auxiliary	21
Esthetic Full-Mouth Implant Reconstruction: CAD/CAM Restorations and Computer Guided Technology (Module I)	Nov. 11	Harel Simon, DMD; Faculty	Los Angeles	$275 Dentist/ $175 Auxiliary	7
Esthetic Full-Mouth Implant Reconstruction: CAD/CAM Restorations and Computer Guided Technology (Module II)	Nov. 12	Harel Simon, DMD; Faculty	Los Angeles	$275 Dentist/ $175 Auxiliary	7
Esthetic Full-Mouth Implant Reconstruction: CAD/CAM Restorations and Computer Guided Technology (Module III)	Nov. 13	Harel Simon, DMD; Faculty	Los Angeles	$1,795 Dentist	7
The USC Tenth International Endodontic Symposium	Nov. 18-19	Ilan Rotstein, DDS; Faculty; Guest Speakers	Los Angeles	$495 Dentist/ $315 Auxiliary	14
A Contemporary Approach to Diagnosis, Treatment Planning and Therapy in Periodontics	Dec. 9	Ziv Simon, DMD, MSc; Faculty	Los Angeles	$255 Dentist/ $185 Auxiliary	7
Implant Therapy for Edentulous Patients	Dec. 10-11	Homayoun H. Zadeh, DDS, PhD; Faculty	Los Angeles	$1,495 Dentist/ $795 Auxiliary	16

TOPIC	DATE	LECTURER(S)	LOCATION	COST	UNITS
HERMAN OSTROW SCHOOL OF DENTISTRY OF USC CONTINUED					uscdentalce.org
Focus on the Maxillary Sinus: Lecture and Hands-On Workshop	Dec. 10	Bach Le, DDS, MD, FICD	Los Angeles	$995 Dentist/ $695 Auxiliary	7
Pediatric Oral Sedation Certification Program	Dec. 14-16	Stanley Malamed, DDS; Faculty	Los Angeles	$2,895 Dentist/ $345 Auxiliary	21
Pediatric Advanced Life Support (PALS)	Dec. 17-18	Stanley Malamed, DDS; Faculty	Los Angeles	$195	14
KERN COUNTY DENTAL SOCIETY					kerncountyds.org
Infection Control, Dental Practice Act, OSHA Compliance	July 22	Leslie Canham	Bakersfield	$200 Member/ $300 Non-member/ $75 Auxiliary	6
Bisphosphonates and Oral Pathology	Sept. 16	Parish Sedghizadeh, DDS	Bakersfield	$200 Member/ $300 Non-member/ $75 Auxiliary	6
Mini Implants for the General Dentist	Oct. 21	Raymond Choi, DDS	Bakersfield	$200 Member/ $300 Non-member/ $75 Auxiliary	6
MARIN COUNTY DENTAL SOCIETY					mcdsweb.org
Wellness	Sept. 20	Marc Geissberger, DDS	McInnis Park Club Restaurant, San Rafael	$45 Member/ $90 Non-member	2
Health Care Provider CPR Class	Sept. 29, Oct. 27, Nov.17	TBD	Muir Woods Room, San Rafael	$75 Member/ $150 Non-member	3.5
Sleep Apnea	Oct. 18	Janice S. Lee DDS, MD, MS	McInnis Park Club Restaurant, San Rafael	$45 Member/ $90 Non-member	2
MID-PENINSULA DENTAL SOCIETY					mpds.org
Why Are Women So Strange and Men So Weird? Joint Meeting with San Mateo County Dental Society	Oct. 20	Bruce Christopher	Hiller Aviation Museum, San Carlos	$60	2
MONTEREY BAY DENTAL SOCIETY					mbdsdentist.com
New Products: When do I Make the Switch?	Sep. 16	Greg Gillespie, DDS	Hyatt Regency, Monterey	$280 Member/ $130 Auxiliary	7
Street Drugs Update 2011	Oct. 7	Harold Crossley, DDS	Hyatt Regency, Monterey	$280 Member/ $130 Auxiliary	7
NAPA-SOLANO DENTAL SOCIETY					707-428-3894
Dental Compounding	Sept. 15	Chuck Snipes	Fairfield	$60	2
High Tech Power Dental Office	Oct. 21	Lawrence Emmott, DDS	Fairfield	$285	7
Dental Law, Infection Control, OSHA	Nov. 17	TBD	Fairfield	$125	6
NORTHERN CALIFORNIA DENTAL SOCIETY CONTINUES ON NEXT PAGE					ncdsonline.org
Emergency Medicine and Post Operative Complications	Sept. 9	James Baribaldi, BS, DDS, MA	Community Center, Red Bluff	$125 Member/ $225 Non-member/ $55 Auxiliary/$15 late registration fee	7

TOPIC	DATE	LECTURER(S)	LOCATION	COST	UNITS
NORTHERN CALIFORNIA DENTAL SOCIETY CONTINUED					**ncdsonline.org**
Biomimetic Dentistry — Four Fundamentals of Advanced Adhesion (Biomimetic) Dentistry	Oct. 14	David S. Alleman, DDS	Community Center, Red Bluff	$125 Member/ $225 Non-member/ $55 Auxiliary/$15 late registration fee	7
Esthetic Dentistry	Nov. 4	James R. Dunn, DDS	Community Center, Red Bluff	$125 Member/$225 Non-member/$55 Auxiliary/$15 late registration fee	7
ORANGE COUNTY DENTAL SOCIETY					**ocds.org**
It's a Snap...Implant Assisted Removable Prosthetics	Sept. 13	Tony Daher, DDS, MSEd, FACP	Irvine	$69	2.5
BLS for the Healthcare Provider	Oct. 5	Helen McCracken, RDH, MS	Orange	$69	3
Clear the Fears, Drug the Bugs: Pharmacological Management of the Surgical Patient	Oct. 11	John Yagiela, DDS, PhD	Irvine	$69	2.5
The New Age "Digital Dental Team": The Synergy of Man, Materials and Machines	Nov. 8	Edward McLaren, DDS, MDC	Irvine	$69	2.5
SACRAMENTO DISTRICT DENTAL SOCIETY					**sdds.org**
CPR - Basic Life Support for the Dental Professional (full course)	June 25	SDDS Instructors	Sacramento	$70	5
CPR - Basic Life Support for the Dental Professional (renewal course)	Aug. 6; Nov. 12	SDDS Instructors	Sacramento	$55	4
Hard and Soft Tissue Dental Lasers	Sept. 9	Douglas Young, DDS, MS, MBA	Sacramento	$187	5
Treating Trauma Without Drama	Sept. 13	Kenneth Tittle, DDS	Sacramento	$57	2
Licensure in a Day — OSHA Review Dental Practice Act and Infection Control	Sept. 30	Marcella Oster, RDA	Sacramento	$165	6
Caries Management By Risk Assessment — The Caries Balance	Oct. 11	John D.B. Featherstone, MSc, PhD	Sacramento	$57	2
Smile Design: Something Old, Something New, Something Borrowed and Something RED?	Nov. 4	Douglas Lambert, DDS, FACD, FASDA, ABAD	Sacramento	$187	5
Generational Dynamics in Communication	Nov. 8	Gordon Fowler, President, 3Fold Communications	Sacramento	$57	2
SAN DIEGO COUNTY DENTAL SOCIETY					**sdcds.org**
Periodontics — Prosthetics	July 16	Jim Grisdale	Lawrence Family Jewish Community Center, San Diego	$50-$125	6
CPR Renewal	Aug. 18	Amerimed	San Diego	$20-$40	3
Hot Topics in Aesthetic and Restorative Dentistry	Sept. 15	David S. Hornbrook, DDS	San Diego	$99	3
OSHA Infection Control and CA Dental Practice Act	Oct. 7	Eve Cuny, BA, MS; Luis Dominicus	San Diego	$50-$125	4
CPR Renewal	Oct. 20	Amerimed	San Diego	$20-$40	3

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SAN FERNANDO VALLEY DENTAL SOCIETY					**sfvds.org**
Occlusion	Sept. 14	J. Luis Ruiz, DDS, FAGD	Van Nuys	$175 Member/ $300 Non-member	7
Dental Materials	Oct. 19	Brian Novy, DDS	Van Nuys	$175 Member/ $300 Non-member	7
SAN GABRIEL VALLEY DENTAL SOCIETY					**sgvds.org**
Practice Management	Sept. 20	TBD	Almansor Court, Alhambra	$65 Member/ $100 Non-member	3
Perioperative Patient Management	Oct. 18	Alan L. Felsenfeld, DDS	Almansor Court, Alhambra	$65 Member/ $100 Non-member	3
21st Century Caries Control	Nov. 15	Brian Novy, DDS	Almansor Court, Alhambra	$65 Member/ $100 Non-member	3
SAN JOAQUIN DENTAL SOCIETY					**sjds.org**
Occlusion and Restorative Dentistry — Truth vs. Myth	Sept. 15	Thomas Basta, DDS	Stockton	TBA	4
The Modern Restorative Practice — All Ceramics, Digital Impressions vs. Conventional D-CAM Restorations	Nov. 17	Michael DiTolla, DDS	Lodi	TBA	7
SAN MATEO COUNTY DENTAL SOCIETY					**smcds.com**
Employment Law and Trends in Malpractice	Sept. 15	Ron Goldman, ESQ	Crowne Plaza Hotel, Foster City	$45 Member/ $55 Non-member	3
AHA CPR — BLS Renewal Course	Sept. 27	Stephen R. John, DDS	Redwood City	$45 Member/ $60 Non-member	4
Legal Aspects of Asset Protection	Sept. 29	Lori Adesiewicz, ESQ	Redwood City	$10 Member/ $25 Non-member	0
AHA CPR — BLS Renewal Course	Oct. 11	Richard A. Fagin, DDS	Redwood City	$45 Member/ $60 Non-member	4
Why Are Women So Strange and Men So Weird? Joint Meeting with Mid-Peninsula Dental Society	Oct. 21	Bruce Christopher, Psychologist and Humorist	San Carlos	$50 Member/ $60 Non-member	3
Dental Board of California Requirements	Nov. 4	Julian Goduci, CHMM	Redwood City	$60 Member/ $70 Non-member	4
Cal-OSHA and Regulatory Requirements	Nov. 4	Julian Goduci, CHMM	Redwood City	$70 Member/ $80 Non-member	4
AHA CPR — BLS Renewal Course	Nov. 15	Stephen R. John, DDS	Redwood City	$45 Member/ $60 Non-member	4
All-Ceramic Update 2011	Nov. 17	David Hornbrook, DDS, FAACD	Crowne Plaza Hotel, Foster City	$45 Member/ $55 Non-member	3
AHA CPR - BLS Renewal Course	Dec. 12	Richard A. Fagin, DDS	Redwood City	$45 Member/ $60 Non-member	4

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SANTA BARBARA-VENTURA COUNTY DENTAL SOCIETY					**805-656-3166**
Dental Diagnostics with 3D Imaging	Sept. 14	Bruno Azevedo, DDS, MS	Westlake Village	$99	3
Implant Dentistry — Commercial Implications vs. Reality	Oct. 7	Hessam Nowzari, DDS	Oxnard	$185	7
Occlusion for Dummies	Nov. 18	Donald N. Reid, DDS	Oxnard	$185	6
Infection Control and Dental Practice Act	Dec. 9	Noel Kelsch, RDH; Jason Wood	Oxnard	$150	4
SANTA CLARA COUNTY DENTAL SOCIETY					**sccds.org**
TBA	Sept. 8	Edmond Bedrossian	Villa Ragusa, Campbell	$35 Non-member	2
Sleep Apnea/TMD	Oct. 13	Dennis Bailey	Villa Ragusa, Campbell	$35 Non-member	2
TBA	Nov. 10	Arun Sharma	Villa Ragusa, Campbell	$35 Non-member	2
Radiography/TBA	Dec. 8	Lawrence Chan	Villa Ragusa, Campbell	$35 Non-member	2
TRI-COUNTY DENTAL SOCIETY					**tcds.org**
What You Need to Know About Dental Trauma	Sept. 15	Leif Bakland, DDS	Colton	$40	2
Dental Trauma and Planning Successful Outcomes for Dental Implant Surgery	Sept. 15	Leif Bakland, DDS; Jaime Lozada, DDS	Colton	$40	2
Oral Pathology, Clinical Applications of Salivary Diagnostics, Dental Erosion and Acid Reflux Disease	Nov. 17	Elizabeth Andews, DDS; Bradley Henson, DDS; David Lazarchik, DDS	Colton	$40	2
TULARE-KINGS DENTAL SOCIETY					**tkdentalsociety.com**
Medical Emergencies in the Dental Office	Aug. 19	James Garibaldi, DDS, MA	Lamp Liter Inn, Visalia	TBD	7
California Dental Practice Act and Infection Control	Oct. 21	Marcella Oster, RDA	Lamp Liter Inn, Visalia	TBD	4
UNIVERSITY OF CALIFORNIA LOS ANGELES SCHOOL OF DENTISTRY CONTINUES ON NEXT PAGE					**dentistry.ucla.edu**
680 Pre-Conference Esthetics: Hawaii 2011	July 2	Jeff Morley, DDS	Maui, Hawaii	$198	4
681 Esthetic Dentistry and Periodontics: Hawaii 2011	July 4-8	Jeff Brucia, DDS; Naser Barghi, DDS	Maui, Hawaii	$798	30
480 UCLA Aesthetic Continuum	July 21-24, Aug. 18-21, Sept. 15-18	Jeff Morley, DDS; Jimmy Eubank, DDS; Brian P. LeSage, DDS	Los Angeles	$6,995	90
Sleep Medicine Mini-Residency	Aug. 12-13, Sept. 9-10, Oct. 7-8	Dennis R. Bailey, DDS; Robert L. Merrill, DDS, MS	Los Angeles	$5,995	60
Dentoalveolar Surgery	Aug. 13	Earl G. Freymiller, DMD, MD; Alan L. Felsenfeld, DDS	Los Angeles	$198 Dentist/ $98 Auxiliary	7

TOPIC	DATE	LECTURER(S)	LOCATION	COST	UNITS
UNIVERSITY OF CALIFORNIA LOS ANGELES SCHOOL OF DENTISTRY CONTINUED					dentistry.ucla.edu
RDA Required Course: Pit and Fissure Sealants	Aug. 27-28	Cara Batson, RDA; Charlene Flowers-Taylor, RDA	Los Angeles	$575	16
RDAEF Expanded Duties (Module II)	Aug. 20-21	Richard G. Stevenson III, DDS; Joseph Cooney, BDS, MS	Los Angeles	$4,995	128
Advanced Dental Sleep Medicine Seminar	Oct. 14-15	Dennis R. Bailey, DDS; Robert L. Merrill, DDS, MS	Los Angeles	$998	16
Dental Ethics for a Changing Profession	Oct. 15	Gary Herman, DDS	Los Angeles	$198 Dentist or Auxiliary	7
Advanced Implant Therapy	Oct. 17-21	Henry H. Takei, DDS, MS; Sascha A. Jovanovic, DDS, MS	Los Angeles	$3,995	40
Practical Occlusion for Esthetics and Function	Oct. 21-23	Jimmy Eubank, DDS	Los Angeles	$4,495	20
Recent Advances in Detection and Management of Pre-Cancer Lesions	Oct. 22	Diana V. Messadi, DDS, MMSc, DMSc	Los Angeles	$135 Dentist or Auxiliary	4
RDA Required Course: Infection Control	Oct. 22	Cara Batson, RDA; Charlene Flowers-Taylor, RDA	Los Angeles	$250	8
Certification in Pediatric Oral Sedation	Oct. 27-29	John A. Yagiela, DDS, PhD; Christine Quinn, DDS, MS	Los Angeles	$2995 Dentist/ $225 Auxiliary	26
Re-Certification in Pediatric Oral Sedation	Oct. 29	John A. Yagiela, DDS, PhD; Christine Quinn, DDS, MS	Los Angeles	$295	8
Complete Dentures: Back to the Future	Oct. 29	Eleni Roumanas, DDS; Kumar Shah, BDS	Los Angeles	$198 Dentist or Auxiliary	7
7th Annual Endodontic Distinguished Lecture	Oct. 29	Mo Kang, DDS, PhD; Nadia Chugal, DDS, MS, MPH	Los Angeles	$250	7
HIV Infection: An Update on Management and Emerging Issues	Oct. 29	Fariba S. Younai, DDS	Los Angeles	$198 Dentist/ $98 Auxiliary	7
Medical Emergencies for the Dental Team: Protecting Your Patient; Protecting Yourself	Nov. 5	John A. Yagiela, DDS, PhD; Fred Dennis, MD, MBA, FACEP	Los Angeles	$198 Dentist/ $98 Auxiliary	7
California Dental Practice Act and Infection Control	Nov. 5	Andy Wong, DDS	Los Angeles	$135 Dentist/ $95 Auxiliary	4
RDA Required Course: Pit and Fissure Sealants	Nov. 5-6	Cara Batson, RDA; Charlene Flowers-Taylor, RDA	Los Angeles	$575	16
Diagnostic Box: Esthetics, Occlusion Comprehensive Care	Nov. 11-13	Jimmy Eubank, DDS	Los Angeles	$3995 Dentist/ $395 Staff	24
Interpersonal Skills and Clinical Psychology	Nov. 19	Craig D. Woods, DDS	Los Angeles	$198	7
Your Patient's Medical History: What You Don't Know Can Hurt You	Nov. 19	Alan L. Felsenfeld, DDS; Earl G. Freymiller, DMD, MD	Los Angeles	$198 Dentist/ $98 Auxiliary	7
Guidelines to Implementing Practice-based Research Into Your Dental Practice	Dec. 3	Francesco Chiappelli, PhD	Los Angeles	$198	7

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UNIVERSITY OF CALIFORNIA SAN FRANCISCO SCHOOL OF DENTISTRY					**dentistry.ucsf.edu/CDE**
Periodontitis and Peri-Implantitis... What You Need To Know	Sept. 23-25	Richard Nathan, DMD, MS	Yosemite	TBA	12
Roundtable Seminars Study Group	Oct.-May 2012	Craig Pettengill, DDS	San Francisco	TBA	28
Drug Interactions	Oct. 8	MA Pogrel, MD, DDS; Mehran Hossaini, DMD	San Francisco	TBA	7
All Ceramics Lecture and Workshop	Oct. 14-15	A. Raigioski, DDS	San Francisco	TBA	14
Pediatric Oral Conscious Sedation — Renewal	Oct. 28	Richard Sobel, Coordinator	San Francisco	TBA	7
Top 50 Drugs	Dec. 2	Harold L. Crossley, DDS	San Francisco	TBA	7
Implant Therapy	Dec. 10	Thomas McGarry, DDS	San Francisco	TBA	7
UNIVERSITY OF CALIFORNIA SAN FRANCISCO SCHOOL OF DENTISTRY-FRESNO					**fresno.ucsf.edu/continuing_ed**
Geriatrics and Dentistry — Ultrasonics	July 8	Karen Hays, RDH	UCSF Fresno Campus, Fresno	$155 Dentist/ $129 RDH/ $109 RDA/$129 CDT	7
Clinical Applications of Short Implants: A Paradigm Change in Implant Dentistry	Aug. 19	Drauseo Speratti, DDS	UCSF Fresno Campus, Fresno	$155 Dentist/ $129 RDH/ $109 RDA/$129 CDT	7
Prevention for the Future — Innovative Dental Products	Oct. 21	3M ESPE Speaker	UCSF Fresno Campus, Fresno	$155 Dentist/ $129 RDH/ $109 RDA/$129 CDT	7
Infection Control and OSHA Review	Nov. 18	Morgan Lawson	UCSF Fresno Campus, Fresno	$112 Dentist/ $107 RDH/ $97 RDA/$107 CDT	4
Oral Pathology	Dec. 9	Nasser Said-Al-Naief, DDS	UCSF Fresno Campus, Fresno	$112 Dentist/ $107 RDH/ $97 RDA/$107 CDT	4
WESTERN LOS ANGELES DENTAL SOCIETY					**westernlads.org**
Oral Surgery	Sept. 13	Fred Freymiller, DDS	Culver City	$75 Member/ $120 Non-member/ $60 Auxiliary	3
Research on New Endodontic Technology	Oct. 11	Sergio Kuttler, DDS	Culver City	$75 Member/ $120 Non-member/ $60 Auxiliary	3
Advanced Restorative and Practice Branding	Dec. 3	Baldwin Marchack, DDS	TBA	TBA	7

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LOS ANGELES COUNTY

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ENCINO - Leasehold & Equip Only! Located on 2nd fl of multi story prof bldg w/ 2 eq. ops & 1 plmbd not eq. Low sales price.ID #4005.
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LOS ANGELES - Over 20 yrs of goodwill this Turn Key practice is located in a 7 story Med/Dent building. Low sale price! ID#3791.
LOS ANGELES - Long established practice located in a shopping center w/ heavy traffic flow. NET $58K. ID #2771.
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UPLAND / CLAREMONT Ortho - Long established practice located a med/dent building with low rent. ID #3681.
WEST HILLS - Long established state-of-the-art practice w/ 6 computerized eq. ops., in a 3 story Med Prof. bldg. Fee for service. #4013.
WHITTIER - Fee for serv pract w/ 59 yrs of gdwll. Located in a 1,450 sf single standing bldg w/ private parking. Bldg for Sale. ID#3931.
WOODLAND HILLS - Well equipped Pedo office with 3 chairs in open bay area. 31 years of goodwill. NET OF $237K. ID #3661.

ORANGE COUNTY

ALISO VIEJO - Beautiful spacious practice with 4 fully eq. ops. Great opportunity for GP or Spec. Leasehold & Equip Only! #3831.
ALISO VIEJO - PRICE REDUCED!! Modern design Turn Key practice with great views and beautiful decor. ID #3301.
ALISO VIEJO - Modern design office loc 2nd fl of a busy mall. Fee for service practice. Open 5 days/wk w/ 3 days/wk of hygn. ID#3981
GARDEN GROVE - Turnkey practice w/ over 20 years of gdwll located in one story free standing building w/ ample parking. ID #3988.
IRVINE - Price Reduction!! Leasehold & equip. only. 5 eq. ops., 1,450 sq. ft suite located in busy Ralph's shopping center. ID #3401.
IRVINE GP - Established in 1987, located in 3 story med/dent bldg. Next to a busy shopping center. NET OF $74K. ID #3901.
IRVINE - Leasehold & Equip Only! Great opportunity for a Spec.! Beautiful décor office w/ 4 ops, located in a Med / Dent bldg.ID#3986.
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RIVERSIDE / SAN BERNARDINO COUNTIES

HEMET - Established 30 yrs ago this beautiful practice consist of 3 eq. ops & 1 plmbd not eq. located in a busy shopping center # 3851.
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RANCHO CUCAMONGA - Leasehold & equip. only! 6 eq. ops., 1,800 sq. ft. ste located in 2 story med/ dent building. ID #3191.
UPLAND - Leasehold & Equip Only! 3 eq ops office located busy single story shopping center with great mountain views. ID#3982.
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BAKERSFIELD - State of the art office. Fee for service. Grossed approx. $1.9M for 2010. NET OF $405K. Gorgeous office. ID #4017.
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Licensed agents and brokers may not place classified ads. For information on display advertising, please contact Corey Gerhard at 916-554-5304 or Corey.Gerhard@cda.org.

CDA reserves the right to edit copy and does not assume liability for contents of classified advertising.

CLASSIFIEDS, CONTINUED FROM 431

OFFICE SPACE FOR LEASE IN ROHNERT PARK — Blank slate. Build your dream office. 3,100 sq. ft. in a dental/medical area. Email brushflosssmileinfo@gmail.com.

SACRAMENTO FOOTHILLS OFFICE FOR LEASE OR SALE — Beautiful, turn-key office in high-end retail center available for lease or purchase located in Sacramento Foothills. Owner finance okay. Contact 916-390-5993.

SANTA BARBARA OFFICE CONDO FOR LEASE/SALE — Prime location. Contact 310-546-3683 or e-mail rmemsic@earthlink.net.

SANTA CLARA OFFICE FOR RENT OR LEASE — Fully equipped, six operatories, ample parking, free standing one story building, approximately 1,800 sq. ft. Close to Santana Row. Option to buy. Call 619-644-2906.

SPACE AVAILABLE FOR RENT — SF Bay Area North. 750-5000 sq. ft. of existing dental office. Very reasonable. Inquiries 707-994-1218.

TWO OPERATORIES FOR SUBLEASING IN BEAUTIFUL VICTORIAN SUITE — Close to main thoroughfare, across from the Alameda City Hall. Ideal for general practitioner considering restorative or dental hygiene. Call 510-865-2450 for details.

WOODLAND HILLS OFFICE FOR SUBLEASE — Very desirable Ventura Blvd. location with lush landscaping and waterfalls. Floor to ceiling windows in all operatories. Very reasonable terms and free patient parking. Perfect for relocation, second office, recent grad or specialist. Contact 818-884-9420.

CONTINUES ON 436

WESTERN PRACTICE SALES

John M. Cahill Associates

BAY AREA

A-8941 SAN FRANCISCO– Ready to Move In. Fully Equipped. 2 ops. Plumbed for 1 add'l **$75k** **A-970 SF – FACILITY 450 Sutter never looked so good!!!** Corner suite w/ 7 large windows! Stunning city views! 1,000 sf w/4 fully equipped ops **$125k**

B-9541 BRENTWOOD - Facility Only Centrally located in a highly visible shopping complex w/ ample parking in a well-established neighborhood. 2,203sf & 6 ops **$230k**

B-956 BERKELEY- Professionally planned, efficiently laid out for ease of workload and patient flow overlooking Berkeley Hills. 792 sf w/3 ops **$220k**

C-7811 SOLANO CO - 2,997 sf w/6 ops + 2 Hyg ops + 1 add'l op! Buy the whole practice for $1.3m or only 50% for $650k. **Call for Info!**

C-8901 SANTA ROSA– Residential area. 40+ new pats/mo. Highly Visible! 1291sf & 3 + 1 op. **$475k**

C-9501 MARIN COUNTY–Remarkable oppty awaits you! Near HWY 101.~ 800 sf w/3 ops. **$300k**

D-842 PLEASANTON –1,488sf w/ 2 ops **$295k**

D-877 LOS ALTOS -Pristine Professional plaza. Office is ~ 2,400sf - 6 ops **2009 Collections - $819k!! Reduced to $350k to offset rent amount**

D-9091 ATHERTON -Turnkey operation 969 sf & 3 ops ***Call for Details!***

D-925 SANTA CLARA - Retail Center in the heart of the Silicon Valley. 1,500 sf & 3 ops **$499k**

D-9331 SARATOGA– FACILITY– GD & Specialists! State of the Art Equip. 1,187sf w/3 ops **$98k**

D-960 *Facility only* SAN JOSE - Establish primary or satellite office with minimal start-up costs. This would cost more than our asking price to duplicate! *Reasonable rent and great lease* with **opportunity to purchase condo suite also!** 1,158sf w/3 fully equipped ops **$118k**

D-965 WATSONVILLE - Location and a large stable patient base! Office ~ 2,393sf, w/ 4 equipped ops + plumbed for 4 add'l ops. **$420k**

D-967 SAN JOSE – FACILITY—A fully equipped facility in the Silicon Valley! Like new, this beautiful scratch-start office is conveniently located. The suite ~1,600+ sf w/ 4 ops **$195k**

D-968 MILPITAS— FFS practice is situated on the corner of two major thoroughfares. Office is ~ 1,950sf w/2ops. **$175k**

NORTHERN CALIFORNIA

E-729 AUBURN - Busy retail shp ctr w/excellent signage & good traffic flow. 1750sf, 4ops. Plumbed for 2 add'l ops **$250k**

E-915 ELK GROVE—Averages 8 patients w/ approx 5-6 new pats/month. Located in an attractive professional building. 1,200sf / 4 ops. **$650k**

E-8641 SACRAMENTO-FACILITY Single Story office near county buildings. 2,100+ sf w/ 3 ops & plumbed for 1 add'l **$50k**

E-955 ELK GROVE First-floor suite in desirable commercial corridor. Giant foyer/spacious office w/ spectacular décor. 3 ops. Plumbed for 2 add'l **$375k**

E-961 SACRAMENTO -Great opportunity! Doctor averages 12-15 patients .Office is located near two major thoroughfares and has 5ops**. $325K**

E-948 WOODLAND - Customized service in warm and caring atmosphere. 5 ½ hygiene days/w. Office ~ 1,400 sf w/ 4 ops. **$465K**

E-969 FAIR OAKS Everyday will be a joy to come to work. Averages 10-15 patients per Office is ~ 600sf w/2 ops. **$250k**

G-751 WILLOWS- Complete remodel ~5 yrs ago. FFS GP. 2350sf /4 ops. Plumbed for 2 add'l. **Practice $50k / Real Estate $185k**

G-875 YUBA CITY–Estab. 30 + years, GP, FFS, 3575sf /9 ops, great location. **$1.63m w/Cerec**

G-883 CHICO VICINITY – Quality FFS GP. Attractive Prof Plaza. 1,990 sf w/ 5 ops **$535k**

G-952 PARADISE- Well-established fee-for-service practice. Ample parking w/ remote controlled, privately locked garage. 1,138 sf w/3 ops **$185k**

H-856 SOUTH LAKE TAHOE Over 50 new patients/ mo Respected & Growing! 1568 sf & 4 ops **$325k**

CENTRAL VALLEY

I-889 MERCED- Heart of town, bustling with activity & foot traffic. 3 ops **$265k**

I-923 MODESTO—1495sf/ 4op+1, Newer, All digital. **$250K**

CENTRAL VALLEY CONTINUED

I-945 TRACY - Young, growing, family-oriented practice. Highly motivated patient base. 1,300 sf & 4 ops **$350k**

I-951 MODESTO- Street-level suite. Dental Professional building. 886sf w/ 3 ops **$265k**

I-966 MODESTO - Facility The practice newly renovated, w/ professional décor and floor plan. Sparkling, immaculate Office ~ 700sf w/2 ops, **$89k**

J-928 ATWATER - Well-established & respected for gentle treatment. Prof Bldg in desirable area. 1,313 sf w/3 spacious ops **$230k**

J-943 CLOVIS FACILITY ONLY—This would cost more to duplicate! Located in a highly visible shopping center. Office is ~2,098sf w/ 6 ops **$80k**

SOUTHERN CALIFORNIA

K-887 ESCONDIDO-Beautifully landscaped dental prof bldg 1,705 sf w/5 ops **$175k**

SPECIALTY PRACTICES

I-7861 CTRL VLY ORTHO- 2,000sf, open bay w/8 chairs. Garden View. 45 years Goodwill. FFS. 60-70 patients/day. Prof Plaza. **$370k**

D-892 MORGAN HILL ORTHO- Remarkable Oppty! Floor to Ceiling windows—wooded courtyard. 1900sf & 6 chairs in open bay. **$275k**

H-913 SIERRA FOOTHILLS ORTHO– Strong, loyal base referral base. Practice averages 30 – 60+ pats/day. Pristine, remodeled building w/ ample parking. 2,600 sf w/ 5 chairs/bays **$500k**

K-929 SANTA MARIA – PROSTHODONTICS - Where "the patient comes first". Restorative/Implant Practice, FFS, 3 ops 1400 sf **$450k**

I-9461 CENTRAL VALLEY/ORTHO - Seller has strong referral base and happy patients! Well-respected for excellent, quality service in this family-oriented community. 1,650 sf w/5 chairs/bays plus (2) additional plumbed. **$140k**

Timothy G. Giroux, DDS

Jon B. Noble, MBA

Mona Chang, DDS

John M. Cahill, MBA

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3041 SOUTH BAY GP

Well est. & successful practice in gorgeous state-of-the-art facility located in a most desirable area. Modern equipment updated in 2007 and near paperless office. Equipment includes Gendex digital x-rays, Panorex, Cerec & Dexis. 1,653 sq. ft. facility w/6 fully-equipped ops. Avg. GR for past 5 years 1.6M w/59% overhead. 2010 GR as of Aug. on track for 1.5M+. Quality staff. Long term lease available. This is an outstanding opportunity for the experienced dentist looking for a high quality practice. Asking $1.3M.

3049 SAN JOSE GP

Well-located, across from O'Connor Hospital, general practice in 2,118 sq. ft.state-of-the-art facility w/ 3 fully-equipped ops. 2 pvt. offices (1 can be plumbed for 4th op.). This office is beautifully designed and is stunning. In addition to his general practice, owner treats sleep apnea patients. He is selling just the general operative portion of the practice and is willing to help for a smooth transition. Ideal for an experienced dentists looking to merge an existing practice.

3048 SAN JOSE GP

Owner retiring from a small well-est. practice with great upside potential. 900 sq. ft. office with 3 ops. near medical center. 3 Dr. days/week. Owner willing to help for a smooth transition. Asking $95K.

SOLD

3050 EAST SAN JOSE FACILITY

Exceptional opportunity for a beautiful state-of-the-art, first class facility with 8 large ops. & 2 pvt. rooms, in a well traveled area. 1 level shopping center almost fully-equipped office with high visibility signs near E. Capital Expressway and 101. If you want exposure, this is the place to be. Asking $190K.

3045 VACAVILLE GP

Turn-key, traditional dental practice with loyal staff and sense of community. Well maintained 900 sq. ft. tastefully decorated office with 2 fully-equipped ops. 2009 GR 224K+, 2010 projected GR as of Aug. $270K+ with 50% avg. overhead. Owner retiring and willing to help for a smooth transition. Asking $172K.

3006 MONTEREY COUNTY ORTHO

Est. Ortho practice in 2,668 sq. ft. office with 5 open bay chairs in a professional dental complex. Panorex and Cephlometric X-ray machines. Stable and loyal referral base. Annualized GR as of Oct 2009 are $335K+. Owner retiring and willing to help for a smooth transition. Asking 227K.

3028 NAPA-SOLANO COUNTY GP

Owner retiring from well-est. practice in 1,400 sq. ft. facility with 5 ops. All fee-for-service pts. with great word-of-mouth reputation. 2009 GR $731K+, June 2010 FY on schedule for $771K+ with just 4/doctor-days. Asking $518K.

3047 WEST SAN JOSE GP

Owner retiring from well-established practice in professional dental building with 3 ops in 950 sq. ft. office. Ideal location near O'Connor Hospital, Town & Country Village and Valley Fair Shopping Center. Avg. GR $169K+ w/60% overhead. Asking $95K.

SOLD

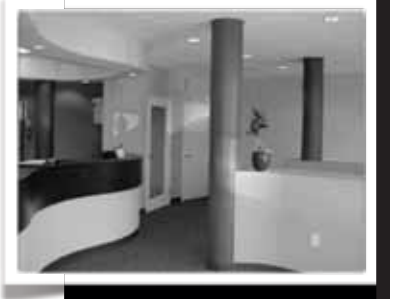
3037 PLACER COUNTY GP

Well est. Placer County General & Cosmetic Practice. 6 fully-equipped state-of-the-art ops., in single story 2,500 sq. ft. stand alone professional building. Avg. GR for past 4 years $1.4M+ with 61% overhead and just 4 doctor-days/week. Asking $1,134,000.

SOLD

Upcoming:

Santa Cruz County GP & Sonoma County GP



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- **BARSTOW:** *For Sale*-General Dentistry Practice. Gross Receipts $395K with an adjusted net income of $193K. Office consists of 1,100 sq. ft. 4 operatories. Intra-Oral camera, Dentisoft. There are 3-hygiene days per week. Practice has been in its present location for the past 25 1/2 years.
- **BIG BEAR CITY:** *For Sale*-General Dentistry Practice. 26 years at current location. Gross Receipts $428K. 3-equipped operatories. Doctor owns the building. New lease available or option to purchase. #14345
- **FOLSOM:** *For Sale*-General Dentistry Practice. Gross Receipts in excess of 1.5M the past three years. Adjusted Net of $550K. 2,700 sq. ft. office with 7 ops, Digital, Dentrix, Intra-Oral Camera, Laser, 5+year old equipment, 8 days hygiene. Beautiful office, great location. Owner retiring. #14336
- **FRESNO:** *For Sale*-General Dentistry IV Sedation Practice. (MERGER OPPORTUNITY) Owner would like to merge his practice into another high quality general dentistry or IV sedation practice. The merger would be into Buyers office. Seller would like to continue to work as either a partner or associate after the merger. 2010 collections were $993K with a $422K adjusted net income. There are 7 days of hygiene.
- **GRASS VALLEY:** *For Sale*-General Dentistry Practice. 2009 GR of $307,590 (3 days/wk) with adjusted net income of $105K. 3 Ops. refers out most/all Ortho. Perio, Endo, Surgery. Intra-oral camera, Diagnodent, EZ Dental Software. Good Location. Owner retiring. #14337
- **GREATER CHICO:** *For Sale*-General Dentistry Practice. Gross receipts in 2010 were $584K, with an adjusted net income of $152K. Approx 1,100 active patients. 4 operatories, Pano, Intra-Oral Camera. Easy dental software. Leased office 1,200 sq. ft. Owner is retiring.
- **GREATER FAIR OAKS-SUNRISE AREA:** *For Sale*-Gross Receipts in excess of $1.1 Million dollars for the past three years. Adjusted net $450K. 2,400 sq ft office-5 ops. Hygiene days-6, Owner works 32 hours per week. Eagle Soft, Laser, Pano Intra-Oral camera, fiber optics. Owner retiring. #14343
- **GREATER SACRAMENTO:** *For Sale*-Pediatric Practice. 2010 GR of $1,095,914, with a 45% overhead. Prevention oriented practice with 2,600 sq. ft. Digital office with Dentrix. Equipment is nine years old. Delta Premier is only insurance. Owner retiring. #14349
- **IRVINE & COSTA MESA:** *For Sale*-General Dentistry practice combined. Gross receipts combined $781K with adjusted net of $396K. Both office spaces are leased with 4-5 ops in each. Both are 1,600 sq. ft. Irvine is equipped with Intra-Oral Camera, Pano & Dentrix. Costa Mesa is equipped with Laser, Intra-Oral Camera, Pano and Dentrix
- **LAGUNA NIGUEL:** *For Sale*-General Dentistry Practice. 2010 gross receipts were $503k. 4 operatories, Pan, computerized with EZ dental software. 1,500 sq. ft. lease. 10 years in present location. Owner retiring. #14352
- **LAKE COUNTY:** *For Sale*-General Dentistry Practice. Gross Receipts 904K with adjusted net $302K. Practice has been in same location for past 23 yrs, and 25 yrs in previous location. 2,600 sq ft with 8 equipped treatment rooms. Intral-Oral camera, Pano, and Data Con software. Owner to retire. #14338
- **LIVERMORE:** *For Sale*-General Dentistry Practice. 2009 Collections were $688K with an adjusted net income of $287K. There are 4 ops in this nicely updated 1,082 sq. ft. office space. Dentrix software, 6-days/wk hygiene. Owner has been in same location for 36 years with long-term employees. Owner is retiring. #14326 SOLD
- **LOS ANGELES:** *For Sale*-General Dentistry Practice.1,200 sq ft 4ops, 29 yrs in present location. Gross Receipts $274K with adjusted net income of $89K. Owner to retire. #14348
- **LOS ANGELES:** *For Sale*-General Dentistry Practice: This practice 80% Dentical and has approximately 2000 active patients. Owner has operated in same location for 31 years. 2009 receipts were $709,000. 6 equipped tx rms, laser, Intra-Oral camera Pano and Ceph. Call for details. #14319 SOLD
- **MODESTO:** *For Sale*-General Dentistry Practice. 5 operatories, 32-years in practice. Gross Receipts $884K w/adjusted net income of $346. Dentrix, Cerec, and Intra-Oral camera. Owner to retire. #14308
- **NAPA:** *For Sale*-General Dentistry Practice. Gross Receipts $800K, with adjusted net income of $300K. Fee for Service. 1300 sq ft 4 ops 6 hygiene days. 38 yrs in present location, 30 yrs in previous location. Owner to retire. SOLD
- **NEWPORT BEACH:** *For Sale*-General Dentistry Practice. Practice has operated at its present location since 1986. Located in a highly affluent Newport Beach community. Three (3) hygiene days per week. Leased office space with 4 ops. in 1,450 sq. ft. Pano & Practice Works software.
- **NORTHERN CALIFORNIA:** *For Sale*-Pediatric practice. Owner has operated in same location for 32 years. Approx. 1,760 active patients, 1,160 sq. ft., Panoramic X-ray, Dexis Digital and Dentrix software in this 5-chair office. 2010 Gross Receipts $610K. Owner retiring. Call for details. #14322. SOLD
- **NORTHERN FRESNO:** *For Sale*-General Dentistry Practice. This is a perfect starter or satellite practice. Excellent location in North Fresno. Gross Receipts in 2010 were $173K. Approximately 450 active patients. 3 operatories. Dentrix software. Leased office 1,200 sq. ft. Owner has been accepted to an Endodontic Residency after starting practice 1 1/2 years ago.
- **OCEANSIDE:** *For Sale*-Modern looking office. 4 op, office space and equipment only. Belmont chairs. Gendex x-ray system, intraoral camera, approx 1200 sq ft. Low overhead-Rent is $1,900/month, and it's a 5 year lease. Staff is available for rehire-front desk $15/hr, assistant 13/hr. Update all the computer systems after purchasing the office in 07. Computers and monitors in every room. #14346
- **PALM SPRINGS:** *For Sale*-General Dentistry Practice. Fee for Service. Gross Receipts $282K with adjusted net income of $157K. 1,280 sq. ft., 3 equipped operatories. Intra-Oral camera, Pano, Practice-NEB software. Doctor willing to transition by working 1-2 days a week. #14332
- **PLUMAS COUNTY:** *For Sale*-3 equipped ops. Space available for 4th op. 1,245 sf office in good location. Gross Receipts $475K. Practice in present location over 50 years. Owner is retiring. #14318
- **REDDING:** *For Sale*-Owner looking for Assoc. trans. into Partnership w/Buy-Out. GR $1 Million dollars income $436K. 5.5 days hygiene, 2,200 sq. ft. #14293
- **RENO:** *For Sale*-General Dentistry Practice and Dental Building: 2009 Gross Receipts $517K with adjusted net income of $165K. 4 ½ hygiene days/week. 1, 800 sq. ft. with 6 equipped ops. (7 Avail). Dentrix software, Pano. Practice has been in its present location for 40 years. Owner retiring. SOLD
- **ROCKLIN:** *For Sale*-General Dentistry Practice. Gross Receipts $593K in 2010 with $240K adjusted net income. Office is 1,630 sq. ft., with 4 operatories equipped with fiber optics. Owner has been in present location for the past 13 years. 3 1/2 days hygiene. Intra-Oral camera, Dentrix software. Owner to retire.
- **ROSEVILLE:** *For Sale*-General Dentistry Practice. Great Location. 2009 GR $900K with adjusted net income of $300K. 1,975 sq. ft. with 4 ops, 8 days hygiene/wk. Digital, Intraoral camera, Dentrix, Trojan, fiber optics, P & C chairs - all less than 5 years old. Owner is retiring. #14327 SOLD
- **SACRAMENTO/ROSEVILLE:** *For Sale*-One of many partners is retiring in this highly successful General Dentistry Group Practice. Intra-Oral camera, Digital Pano-Dexis, electronic charts, owner Financing. Call for further information. #14334
- **SAN DIEGO:** *For Sale*-General Dentistry Practice. This office is plumbed for 4 ops. 3 ops. are equipped with Promo Equipment. Lease is $2,200 per month. 2009 receipts were $185,645. PPO and Fee for service practice. #14315 SOLD
- **SAN DIEGO:** For Sale-General Dentistry practice. Gross Receipts $414K. Practice has been operated by the same owner for the past 6 years. Leased 950 sq. ft. office with 3 equipped operatories. Dentix software, Intra-Oral camera, Panoramic X-Ray. Owner to relocate.
- **SAN DIEGO:** *For Sale*-General Dentistry Practice. 6 ops, Intra-Oral camera, Eagle Soft Software. Office square feet 2,300 with 3 years remaining on lease. 2009 Gross Receipts $1,448,520, with an adjusted net income of $545K. Doctor would like to phase out then retire. #14331
- **SAN DIEGO/CITY HEIGHTS:** For Sale-General Dentistry practice. Owner has operated in same location for 12 years. Approx. 1,000 active patients, Panoramic X-ray, Intra-Oral camera, in this 3-chair office. #14321
- **SANTA BARBARA:** *For Sale*-General Dentistry Practice. This excellent practice's 2009 gross Receipts $891K with steady increase every year. Practice has 6 days hygiene. 1,690 sq. ft., 5 ops, Laser, Intra-Oral camera, Schick Digital X-Ray, Datacon software. Doctor has been practice in same location for the past eleven years of his 31 years in Santa Barbara. Doctor is retiring. #14333 SOLD
- **SAN LUIS OBISPO:** For Sale -Two Doctor General Dentistry Practice. Gross receipts $1,537,142 for 2010 with an adjusted net income of $691K. The office has 2,331 sq. ft. with 8 equipped operatories. Pano, E4D, and Dentrix software. Practice started in 1990 and has been in its present location since 1998. Approx. 3000 active patients. Great location with nice views.
- **SANTA CRUZ:** For Sale-General Dentistry practice. Gross Receipts $300K with a 57% overhead. Office is 1,140 sq. ft. 3 equipped operatories. Intra-Oral Camera, Pano, Digital X-Rays, and Dentrix software. Practice has been in its present location since 1980. Owner retiring.
- **TORRANCE:** For Sale-General Dentistry Practice: Owner has operated in same location for 20 years. Approx. 1,000 active patients, 1,080 sq. ft., Brican System, and Camsight software in this 2 equipped, 3 available-chair office. Gross Receipts $434K with 38% overhead. Owner relocating. #14320
- **TURLOCK:** For Sale- General Dentistry Practice. 2009 Gross Receipts $2,728,319 with an adjusted net income of $925,251. 13 days of hygiene in this tastefully decorated 5,500 sq ft office space. Owner is retiring form clinical dentistry. SOLD
- **VISALIA:** For Sale- General Dentistry Practice. Gross Receipts $616K with an adjusted net income of $ 3[illegible]K. Office is 1,380 sq ft with 3 equipped operatories, Intra-Oral camera, Digital X-Rays, Mogo software, equipment & leaseholds look new. 5 years in present location. Owner to relocate. #14347 SOLD

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CLASSIFIEDS, CONTINUED FROM 432

OFFICES FOR SALE

MODERN FOUR OPERATORY DENTAL BUILDING IN DUNSMUIR FOR SALE OR LEASE — A beautiful mountain setting in Northern California with hunting, fishing and skiing. Building well maintained. Seller motivated, all terms negotiable. Seller would consider forgiving lease/sale payments for first year to help practitioner establish a practice. Present dentist moving out in order to reduce commute driving time. Contact seller at mollyruss@sbcglobal.net or Doris Moss Realty, Brett Waite, Broker, 530-926-3807 or brett@mtshastarealty.com.

OPPORTUNITIES AVAILABLE

DENTAL JOBS AVAILABLE — Aspen offers tremendous earning potential and a practice support model that empowers dentists. We eliminate obstacles for dentists to own their own practice. Call 866-745-5155 or visit aspendentaljobs.com. EOE

OPPORTUNITY AVAILABLE — A great place for a family to live? A practice with a great income and a future? Family, friends and a community? This is the place. Join our team and be "working together to be better." Try Missouri, you'll like it. Contact Sheila at 573-201-9298.

OPPORTUNITY AVAILABLE IN NORTHWESTERN WASHINGTON — Seeking experienced dentist for busy, established, rapidly growing, fee-for-service group dental practice. Excellent immediate income opportunity ($180K to $375K + per year) depending on productive ability and hours worked. Secure long-term position. You can concentrate on optimum patient treatment without practice management duties. Newly equipped, modern office with excellent staff and lab services provided. If you are bright, energetic with a desire to be productive, very personable, people oriented and have great general and specialty clinical skills, please fax resume to Otto J. Hanssen at 425-484-2110.

RELOCATION & SIGN ON BONUS OFFERED FOR ARIZONA — Immediate need for a FT Dentist willing to relocate to Glendale Arizona. The office has a steady patient flow, FT Hygienist and excellent earning potential. Doctor must have 3-5 years experience and be proficient in molar endo. Benefits package offered including Malpractice coverage. Please contact Kristin Armenta at 714-428-1305 or fax to 714-460-8564.

SEEKING MANAGING DENTISTS — If you're looking for a long-term commitment and desire to be productive the opportunity is yours! Seeking full-time, managing dentists to join large group practice in the following areas: Los Angeles, Orange County, Inland Empire, San Diego and doctors willing to relocate to Arizona. Steady patient flow in high volume HMO environment. Required: 3-5 yrs experience and proficient in molar endo. Benefits include: medical, dental, vision, 401K, malpractice coverage and competitive pay! For available positions please call: 714-428-1305, submit your resume to kristin.armenta@smilebrands.com or fax to 714-460-8564.

CONTINUES ON 438

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5999 **PLEASANTON** Adjacent to Hacienda Business Park. 2011 tracking $900,000. Strong Profits. Digital radiography with computers in Ops. Great visibility.

6000 **"SOLD" MILLBRAE** Completely digitized and computerized. 2010 collected $720,000. Available Profits topped $370,000.

6002 **SAN JOSE'S EVERGREEN VALLEY – FILIPINO PRACTICE** Near Highway 101 & East Capitol Expressway. Housed in new building and suite. Busy Hygiene schedule. 2011 tracking $800,000+. Strong Profits.

6003 **"SOLD" PINOLE'S APPIAN WAY** Collected $710,000 in 2010. Excellent Recall System, busy Hygiene Department.

6004 **"SOLD" SAN JOSE'S SANTA TERESA AREA** Beautiful 3-Op office. $200,000 invested here 10/09. Digital & paperless. Collected $277,000 in 2010.

6005 **FAIRFIELD - WEST OF INTERSTATE 80** Generates $500,000/year on Owner's 2.5 day week & 2-days of Hygiene. 2010's Available Profits totaled $249,000. Shall do better with more attention.

6006 **STOCKTON** Beautiful office near West Hammer & Lower Sacramento. Busy retail location. Ideal for nearby Dentist or someone with a Business Plan. 4 Ops, digital radiography, computer charting. No goodwill.

6007 **LIVERMORE** Centrally located. Housed in free-standing building. 2010 topped $400,000 in collections. 4-Ops, paperless charting and digital radiography.

6008 **MENDOCINO COAST** Located in desirable cultural haven. 4-days of hygiene. Owner works 3-day week. $675,000+ revenue performer. Computerized Ops, digital radiography and laser. Great lifestyle.

6010 **BERKELEY – ALTA BATES MEDICAL VILLAGE** Attractive revenues. Last 2-years Profits have averaged $225,000. 2011 doing better!

6011 **SAN JOSE – WEST OF I-280** Long established practice off Saratoga Avenue. Has averaged $400,000 per year in collections. 3-Ops with 4th available in 1,000 sq.ft. suite.

For complete details on any of these opportunities, go to www.PPSsellsDDS.com

Professional Practice Sales of The Great West

Ray and Edna Irving

(415) 899-8580 ~ (800) 422-2818

www.PPSsellsDDS.com

Thinking on selling your practice? Call "PPS of The Great West" today.
This shall be the best decision you make regarding this important change in your life!

"I listed with a competitor for 12 months. Had two people visit my practice. First weekend PPS had my listing, I had 3 people visit and an offer by the end of the first week. Thank you for allowing me to move on to the next step of my life."

"It was a pleasure to work with PPS. I had to sell because of health complications. Mr. Irving listed my practice on Jan 1st, we closed escrow on Feb 27th. It took him less than 60 days to complete the sale as promised."

"When I decided to sell my ortho practice, I sought the services of a large company. Over the 12-month contract, I had one buyer visit. Word was out. It had a devastating effect on my bottom line. Fortunately, I found Ray and Edna Irving! When I finally sold, I choose between two good offers. My regret was the time and money lost with the other guys."

"When I signed the Listing on June 1st, Ray stated he would have the practice sold by Labor Day. The sale was concluded on Sept 1st, two days before Labor Day. Wow!"

"I will always remember your statement when I questioned your contract being only four months. You stated: 'If I can't sell your practice in that time, you should get someone else.' Well, you did with time to spare!"

"Before I called Ray, I had a listing with another prominent Broker. After eleven months without a sale, I called Ray. He sold it in about a month! Would I recommend Ray? Yes!"

"In April, I asked Ray Irving to sell my practice. At the same time my friend decided to sell his practice. He employed another firm. My practice sold June 22. My friend's practice still hasn't sold and he was putting his dreams on hold."

PPS of The Great West's reputation is built upon grounded ethics and effectiveness. Our trademark "client services" include accurate assessments, impeccable marketing plans, complete transparency, generating quick responses, realizing multiple Offers, securing 100%+ financing in days, expert papering of our transactions and sound counsel. Everything is done to protect our Client and to effect a successful transfer. Our intent is simply to provide the best service imaginable for this very important engagement.

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CLASSIFIEDS, CONTINUED FROM 436

OPPORTUNITIES WANTED

IN HOUSE PERIODONTIST/IMPLANT SURGEON AVAILABLE FOR YOUR PRACTICE — In the Greater San Francisco Bay Area. Implant Surgeon/Bone Grafting/ Perio Surgery/3rd Molar Extractions. Contact bayareaperio@gmail.com or 617-869-1442.

PRACTICES FOR SALE

DENTAL PRACTICE FOR SALE — WHITE MOUNTAINS OF ARIZONA — Long established practice. Has weathered the economic storm of the last year near three years intact. I wish to retire. I am 68 years old. The office is 2,000 sq. ft., and has six operatories, fully plumbed and set up. I employ two dental assistants, one hygienist and two front office staff. We have a large inventory of patient records. The office has a reasonable amount of electronic data material and rather modern operatories. It is fully plumbed for nitrous oxide sedation; we use global microscope in endodontics. If interested, please contact me at mountaindental1@hotmail.com. I will respond immediately.

PEDIATRIC PRACTICE IN EUREKA, CALIFORNIA — Don't miss this opportunity! Perfect situation for one or two pediatric dentists looking for an exceptional life style, higher than average income/benefits working in a state-of-the-art facility with six bays, one quiet room and lots of extras! Revenues of $1.5M with low overhead and limited competition. Owner retiring and motivated. Check it out! Email growingsmilesdds@sbcglobal.net.

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LEE SKARIN AND ASSOCIATES

Dental Practice Sales

QUESTIONS MOST OFTEN ASKED BY . . .

. . . SELLERS

1. Can I get all cash for the sale of my practice?
2. If I decide to assist the Buyer with financing, how can I be guaranteed payment of the balance of the salesprice?
3. Can I sell my practice and continue to work on a part-time basis?
4. How can I most successfully transfer my patients to the new dentist?
5. What if I have some reservation about a prospective Buyer of my practice?
6. How can I be certain my Broker will demonstrate absolute discretion in handling the transaction in all aspects, including dealing with personnel and patients?
7. What are the tax and legal ramifications when a dental practice is sold?

. . . BUYERS

1. Can I afford to buy a dental practice?
2. Can I afford not to buy a dental practice?
3. What are ALL of the benefits of owning a practice?
4. What kinds of assets will help me qualify for financing the purchase of a practice?
5. Is it possible to purchase a practice without a personal cash investment?
6. What kinds of things should a Buyer consider when evaluating a practice?
7. What are the tax consequences for the Buyer when purchasing a practice?

Lee Skarin & Associates, Inc.
Offices
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(818) 991-6552

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LEE SKARIN & ASSOCIATES gives straightforward answers to these important questions as well as others you may have – by phone or in person – **without obligation!**

Lee Skarin & Associates have been successfully assisting Sellers and Buyers of Dental Practices for nearly 30 years in providing the answers to these and other questions that have been of concern to Dentists.

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DR. BOB, CONTINUED FROM 442

surprise nobody who has delved into the complexities of the business world where Standard Oil, for example, could easily be the parent company of Huggies diapers, Milk Duds, and Ginzu cutlery.

Mothers who have been soft-pedaling to their kids the fact that they were being served lime-flavored cow parts, can now take some measure of relief learning that "collagen" is the key word and that's all they need to know. Never mind divulging that nowadays most collagen comes from pig and cow skin and bones, all ground up and treated with acids or bases. Add a little whipped cream and the kids are happy as long as the final product jiggles.

In any event, the term "gel" has become so universal today that everything from self-tanning gels to almost anything that can be squeezed out of a tube is basically a gel except JELL-O.

Get ready, dentistry! We already are familiar with gels of various applications, but on the horizon looms something big, and, as far as I know, doesn't involve pig or cow formulas or belong to Kraft Foods.

Stop for a minute and recall what is widely touted as a startling revolution in the dental world at least once each year. Predictions that an anti-caries vaccine is coming soon. A new product that painlessly removes caries is now available … lasers, air abrasive units … all of this is focused on driving the manufacturers of dental handpieces to corporate suicide. No more drilling! A dentist who insists on postponing the acquisition of the new technologies might as well fold up his or her loupes and leave by the back door.

Listen to the riveting revelation of Nadia Benkirane-Jessel, a scientist at France's Institut National de la Sante et de la Recherche Medicale (INSERM) and a coauthor of a new study in ACS Nano, a peer-reviewed journal published by the American Chemical Society, "It's not like a toothpaste," reveals Benkirane-Jessel in case you were thinking Colgate or Crest would have it by Christmas. What it is is a gel containing a peptide, known as MSH (pronounced moosh), or melanocyte-stimulating hormone. Experiments reported in the *Proceedings of the National Academy of Sciences* showed that MSH encourages bone regeneration. Sympathetically based on the feeling that bones need all the encouragement they can get, French scientists reasoned that if the MSH were to be applied to carious teeth, it should help healing as well. After all, bones and teeth are very similar, like molars and mastoids, premolars, and phalanges.

Get ready, dentistry! We already are familiar with gels of various applications, but on the horizon looms something big, and, as far as I know, doesn't involve pig or cow formulas or belong to Kraft Foods.

The French applied a gel containing MSH to "cavity-filled" (meaning bombed-out, presumably) mice teeth that are almost exactly like ours to the extent that they have some, but are seldom treated because they have no dental insurance. After about one month, says Benkirane-Jessel, who is no relation to Georgie, the cavities had disappeared. Disappeared! She allowed that the application could result in a more pleasant trip to the dentist's office. Instead of a drill, she suggested, with the distaste nondentists display when forced to acknowledge that instrument, a quick dab of gel or a thin film against an infected tooth could heal teeth from within.

Really? Restorative material suppliers are properly skeptical but are already calculating the markup on "quick dabs" and working out how to match JELL-O's selection of flavors. As you can imagine, the mice were ecstatic and were issued little certificates of certified dental health. Not recorded, but presumably if collagen was involved and was supplied by the Franco Hog and Cattle Cartel, that group celebrated by eschewing the vin ordinaire in favor of some big-franc bubbly.

Yet, there would be a downside; there always is. In this case, numerous clinical trials over several years will have to be completed before the gels laced with MSH are available to treat caries in humans. Patent attorneys, the DEA, both houses of Congress, PETA, the ACLU and the Pig and Cow Anti-Discrimination League will all have to have a go at it. You guys at Kavo and Midwest: Don't quit your day jobs. ■■■■

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In Development: Jiggle and Moosh to Be Caries Foe

JELL-O was an instant flop, partially because the extensive advertising enthusiastically promoted it as 'The Best Combo Hoof and Horn Dessert You Ever Tasted.'

Robert E. Horseman, DDS

ILLUSTRATION BY DAN HUBIG

Who knew back in 1845 when industrialist Peter Cooper obtained the first patent for a gelatin dessert, that it would turn out to be a worldwide phenomenon? Not Peter — he never promoted the product — probably because he didn't see any future to something made out of cows' horns and hooves. Rib-eye steaks and top sirloins, yes, but recycling had not become in vogue yet, so the patent languished like the gas-powered buggy whip and hydrofluoric acid depilatory patents did previously.

It took a forward-looking cough syrup manufacturer from Le Roy, N.Y., named Pearle B. Wait to buy up Cooper's patent, turning it into a prepackaged commercial product to get the ball rolling. The ball just sat there until Wait's wife, May, renamed the product JELL-O.

JELL-O was an instant flop, partially because the extensive advertising enthusiastically promoted it as "The Best Combo Hoof and Horn Dessert You Ever Tasted."

It wasn't until a patent medicine entrepreneur named Orator Francis Woodward who had successfully promoted "Raccoon Corn Plasters" bought the rights from the Waits in 1899 for $450, figuring if he could sell corn plasters to raccoons, he could cash in on anything.

He was right. At his death in 1906, sales had reached more than $1 million. In 1923, his business was renamed the JELL-O Company, which, following the path of corporations everywhere, merged with Postum Cereal to become the General Foods Corporation, and finally merged with Kraft Foods. This should

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