

Perioesthetic Approach to the Diagnosis and Treatment of Carious and Noncarious Cervical Lesions: Part I

DOUGLAS A. TERRY, DDS*
MICHAEL K. MCGUIRE, DDS†
EDWARD McLAREN, DDS‡
RUDOLPH FULTON, DDS§
EDWARD J. SWIFT JR, DMD, MS||

ABSTRACT

Several factors can contribute to the development of noncarious cervical lesions. Therefore, these lesions can be described and classified according to their primary etiology. Traditionally, most dentists have treated noncarious cervical lesions only with restorative methods, for example, composite resin restorations. However, in many cases, a periodontal or a combined restorative/periodontal approach provides a better esthetic and functional result. In part I of this two-part report, we provide a review of noncarious cervical lesions and a series of clinical case reports showing surgical techniques used and the importance of the periodontal aspect of lesion management.

CLINICAL SIGNIFICANCE

For best esthetic results, the periodontal aspect of noncarious cervical lesions must be considered in treatment planning.

(*J Esthet Restor Dent* 15:217–232, 2003)

Erosive dental lesions, including noncarious cervical lesions (Figure 1), have been described in the literature for many years, and theories concerning their etiology have abounded for almost 150 years. As early as 1862, G.V. Black stated that the etiology was unexplainable and requested that his colleagues share facts on the subject to accumulate

data for a possible explanation in the future.^{1–3} Some of the theories proposed for causation included a disease inherent in the tooth or in the composition of saliva and the friction of the lips; mechanical agents or mechanical agents in the presence of alkalis or acids; friction of folds of the mucous membranes; exfoliation; acids or acids in combi-

nation with mechanical agents; electrolytic action; defective development; and resorption.^{3,4} Erosion was compared by Edwin Darby to the uric acid theory of gout: “Erosion, like gout, is a disease of advanced civilization.”³

In 1907 W.D. Miller⁴ suggested that erosive lesions had “a multiplicity

*Private practice and adjunct assistant professor, Department of Restorative Dentistry and Biomaterials, University of Texas Health Science Center at Houston, Houston, TX, USA

†Private practice and adjunct assistant professor, Department of Periodontics, University of Texas Health Science Center at Houston, Houston, TX, USA

‡Adjunct assistant professor, Biomaterials and Advanced Prosthodontics Department, School of Dentistry, University of California at Los Angeles, Los Angeles, CA, USA

§Associate professor and chairman, General Dentistry Department, University of Texas Health Science Center at Houston, Houston, TX, USA

||Professor and chair, Department of Operative Dentistry, University of North Carolina, Chapel Hill, NC, USA



Figure 1. The etiology of the cervical lesion has been the subject of controversy for over a century.

of names no one of which is fitted to all of the conditions and phenomena present.” The collective term for wearing away of tooth substance was “wasting.” Specific categories of wear included the slow and gradual loss of tooth tissue by friction (“abrasion”), the rubbing of teeth against each other during mastication (“attrition”), the effects of chemical agents (“erosion”), and the effects of mechanical and chemical agents combined (“chemico-abrasion”).⁴

In 1931 W.I. Ferrier described dental erosion as a gradual disintegration of enamel without the caries. He stated, “Its etiology seems to be shrouded in mystery.”^{1,2} In 1932 Kornfeld described his observation of wear facets on the articulating surfaces of teeth involved with cervical erosion.⁵ However, it was not until 1982 that McCoy first reported the breaking or chipping of tooth

substance as a result of occlusal forces and that these lesions occurred in both dentin and enamel and could result in tooth fracture.⁶

The current scientific classification categorizes the forms of tooth substance loss according to Miller⁴ as *attrition*, *abrasion*, and *erosion*. An additional category, initially reported by McCoy and defined by Grippo, was named *abfraction*, derived from the Latin roots that translate as *away* and *breaking*.⁶

CLASSIFICATION OF NONCARIOUS LESIONS

The four recognized categories of noncarious cervical lesions are defined and described as follows:

- Erosion is a chemically induced loss of tooth substance from intrinsic or extrinsic origin occurring mainly from acid dissolution. The intrinsic form of erosion can

be caused by regurgitation of gastric acids, as occurs with habitual vomiting associated with bulimia, anorexia nervosa, hiatal hernia, and pregnancy morning sickness.⁶⁻¹⁵ The external form can be caused by diet (eg, carbonated soft drinks, candies that contain phosphoric or citric acid, citrus fruits or juices, and “baby bottle syndrome”), airborne acids such as industrial chemicals, and chlorinated swimming pool water.⁶⁻¹⁸

- Attrition is the physiologic wear of tooth substance caused by normal tooth-to-tooth contact (ie, incisal, occlusal, and interproximal wear from mastication^{5,19}).
- Abrasion is the pathologic wear of tooth substance caused by abnormal mechanical forces (eg, excessive and improper toothbrushing and improper oral habits such as biting fingernails, bruxism, biting a pipe stem, holding nails between the teeth, and opening hair pins^{6,19-21}).
- Abfraction is the pathologic wear of tooth substance by biomechanical loading forces, primarily at the cervical regions of the dentition. However, it can be manifested also as occlusal invaginations resulting from excessive eccentric loading from parafunctional habits such as clenching and bruxism.^{6,22,23}

Although this classification allows for a better understanding of the causes and treatment of the lesions, several concomitant effects (bio-

chemical, biomechanical, and bioelectric processes) may be responsible for the development of noncarious cervical lesions.^{7,21,22,24} Because a particular lesion can result from one or more of these etiologic factors, from a clinical perspective the criteria used for a differential diagnosis must be based on direct clinical examination, a comprehensive review of the patient's medical and dental history, an inspection of the patient's occlusion for symptoms and clinical signs of trauma, and the morphologic characteristics of the lesion.^{20,24}

MORPHOLOGIC CHARACTERISTICS OF NONCARIOUS CERVICAL LESIONS

The extrinsic form of erosive lesions caused by ingestion of acidic foods, beverages, and medications is generally U shaped or disk shaped, broad, and shallow. These lesions often have poorly defined margins, and the adjacent enamel is smooth, shiny, and free of developmental ridges. The extrinsic form of erosion results from exogenous acids such as dietary acids, fruit juices, and ascorbic acid in sport drinks and candies; this erosion is generally located on the facial surfaces of the anterior teeth (Figure 2).^{7,8,20} The intrinsic form of erosive lesions, caused by reflux of gastric contents¹ or regurgitation, is generally located on the lingual and incisal surfaces of maxillary anterior teeth and appears as flattened wear. Erosive lesions usually are

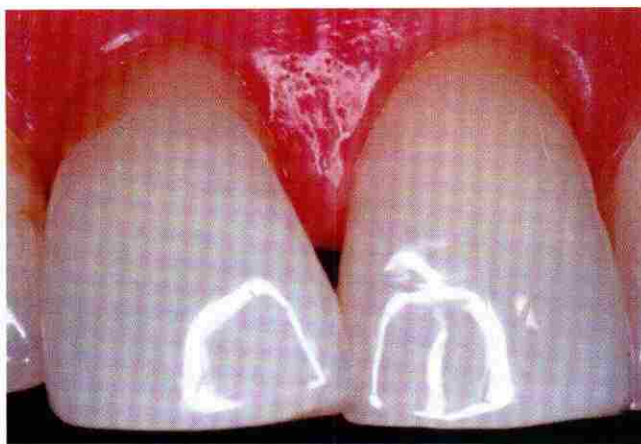


Figure 2. The extrinsic form of the erosive lesion appears as U-shaped or disk-shaped, broad, and shallow smooth-edge depressions that are found on the facial surfaces of anterior teeth.

free from plaque accumulation unless sensitivity prevents adequate oral hygiene.^{16,25}

Attrition lesions usually occur on the occlusal surfaces, incisal edges, and lingual surfaces of maxillary anterior teeth and labial surfaces of mandibular anterior teeth (Figure 3A and B). The teeth are worn in flat facets that can be attributed to the functional movements of the dentition. Also, attrition can occur on proximal surfaces as a result of the anterior component of force, where small horizontal and vertical movements of teeth occur during function, thus causing frictional wear.^{6,7,16,20,21,26-28}

The morphologic characteristics of cervical lesions produced by abrasive forces generally have sharply defined margins and a hard smooth

surface that may exhibit scratching (Figure 4). The cervical abrasion lesion is commonly produced by improper toothbrushing techniques, and the interproximal lesion is caused by friction from objects such as toothpicks. These abrasive lesions are usually free of plaque and are not discolored.²¹

Abfraction lesions typically are irregular V- or wedge-shaped cervical lesions (Figure 5A). The shape of the lesion depends on the relative areas of compression and tension exerted by occlusal forces. If the cusp is put into a state of tension, the resultant cervical defect is wedge shaped; conversely, if the cervical region is subjected to compressive stresses, the defect is more concave or saucer shaped. Circular occlusal lesions also can develop in the enamel and dentin to form

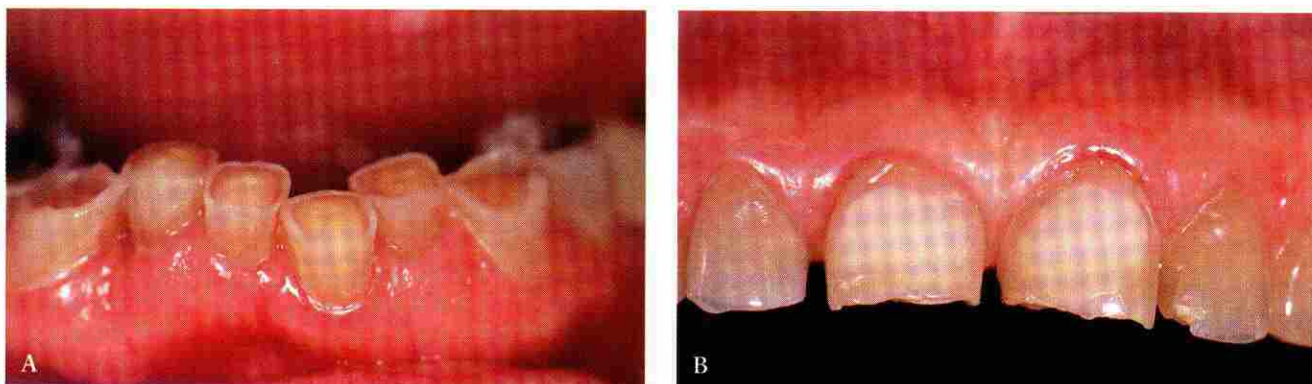


Figure 3. A and B, The attrition lesion appears on teeth as worn flat facets that can be attributed to the functional movement of the dentition as indicated on the incisal edges of these mandibular anterior teeth and maxillary anterior teeth.

occlusal cusp tip invaginations (Figure 5B).^{6,7,19,23,29-33}

After considering all factors related to tooth substance loss from erosion, attrition, abrasion, abfraction, or a combination of these processes, a differential diagnosis should be developed. This differential diagnosis provides information for determining etiology and can require additional information such as age,

diet, oral hygiene routine, medical and dental factors, abnormal oral habits, and occlusal idiosyncrasies.^{19,24,29,30,33} The information acquired during the differential diagnosis allows for a methodical approach for preventive and restorative therapy.

A review of the literature indicates that restorative therapy mainly includes operative procedures for

reconstructing hard tissue, without much consideration of final overall esthetic result.^{19,24,29,30,33-51} Optimal functional and esthetic results may require periodontal as well as operative procedures, or perhaps a combination of the two. This two-part article provides the clinician with a perioesthetic approach for diagnosing and treating carious and noncarious cervical lesions.

TREATMENT CONSIDERATIONS

Cox asserts, "Most Class V lesions are not due to dental caries, and their treatment often strains the limits of technology and esthetics."⁵² A few of the reasons for periodontal and/or operative restorative therapy of carious and/or noncarious lesions include the following:

- Facilitation of self-cleansing and hygiene procedures^{34,38}
- Reduction of cervical dentin sensitivity⁵³



Figure 4. The abrasive lesion is characterized as having sharply defined margins and a hard smooth surface that may exhibit scratching.



Figure 5. A and B, The abfraction lesion appears irregular in shape, typically as a V- or wedge-shaped cervical lesion and as occlusal circular lesions on the enamel and dentin to form occlusal cusp tip invaginations.



- Improvement of esthetics⁵³
- Restoration of normal anatomic contours⁵⁴
- Improvement of gingival health⁵⁴ and symmetry^{33,55}
- Reduction of plaque retention^{34,55}
- Reduction of irritation to surrounding soft tissue
- Prevention of root caries⁵⁵
- Strengthening of the tooth³³
- Prevention of pulpal involvement³³
- Provision of a moderator to the effects of the piezoelectric phenomenon³³
- Diminishment of the progress of the lesion, tooth flexure, and stress concentrations³³
- Prevention of root fracture³³
- Re-creation of appropriate coronal tooth length
- Maintenance of the gingival contour³⁴

Preoperative considerations and procedures may include preventive measures such as fluoride therapy, iontophoresis, brushing with desensitizing dentrifices, professional application of potassium oxalate or other tubule-occluding agents, application of dentin adhesives, occlusal adjustments, dietary instruction, toothbrushing and oral hygiene instruction, discontinuation of poor oral habits, and occlusal guard fabrication.^{19,24,29,56-58}

The restorative therapy for the reconstruction of these cervical lesions may require an interdisciplinary diagnosis and treatment plan that includes the input of other members of the restorative team, including the general dentist, periodontist, orthodontist, and ceramist. Treatment may involve periodontal

plastic surgery, orthodontic measures, and operative procedures. The periodontal procedures include free autogenous mucosal grafts, subepithelial connective tissue grafts, the coronally advanced flap technique, guided periodontal tissue regeneration, and enamel matrix derivative grafts.⁵⁹⁻⁶⁴ Restorative methods can involve the use of conventional glass ionomers, resin-modified glass ionomers, compomers, flowable composites, hybrid composites, microfill composites, laboratory-processed composite and porcelain veneers, laboratory-processed inlays and crowns, all-ceramic inlays and crowns, and porcelain-fused-to-metal crowns and bridges.^{24,29,30,40,41} Orthodontic therapy can involve intrusion, rotation, uprighting, extrusion,

space closure, and restoration of a functional occlusion.⁶⁵

After the diagnostic phase and any appropriate preventive strategies are in place, the concern focuses on the direction of the restorative treatment; this requires determination of the treatment sequence. The treatment depends on the amount of gingival recession, the location and the size of the carious or noncarious lesion,²⁴ and the lesion's relationship to the cemento-enamel junction (CEJ). A periodontal reconstructive approach should be considered when there is root exposure, when the carious or noncarious lesion is apical to the CEJ, and when it is possible to remove the

caries or existing restoration and achieve a relatively flat root surface without endangering the pulp (Figure 6A and B). The carious lesion or recurrent decay on an existing restoration coronal to the CEJ should be removed and restored before surgical treatment. Restorations below the CEJ should be removed because the presence of restorative materials on the root surface precludes the ability to perform root coverage procedures.⁶⁶ In addition, a restorative-only approach should be considered if the carious or noncarious lesion is coronal to the CEJ without gingival recession (Figure 7). The remainder of this article describes the reconstructive periodontal plastic surgery proce-

dures for the carious and noncarious lesion with gingival recession.

In 1985, in order to identify, recognize, and categorize gingival recession in relation to the amount of root coverage anticipated, Miller described four categories for recession-type defects⁶⁷:

- Class I. Marginal tissue recession that has not extended to the mucogingival junction. There is no loss of interdental bone or soft tissue, and complete root coverage can be achieved (Figure 8).
- Class II. Marginal tissue recession that extends to or beyond the mucogingival junction. There is no loss of interdental bone or soft

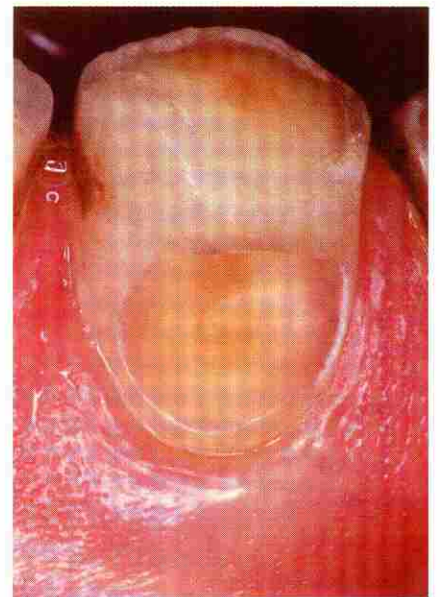
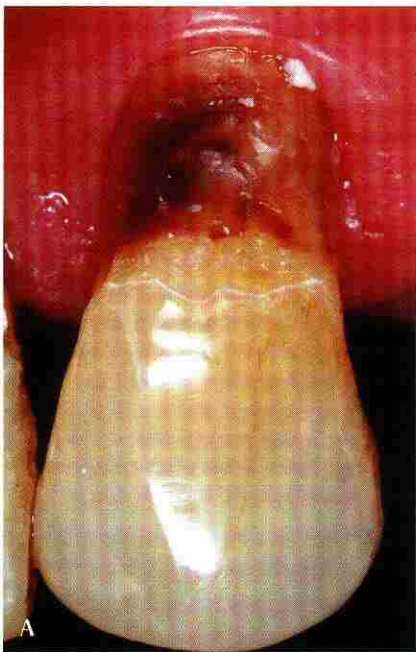


Figure 7. The carious or noncarious lesion that is supragingival to the CEJ without gingival recession requires the removal of the caries and/or a restoration.

Figure 6. A and B, The carious or noncarious lesion that is subgingival to the CEJ requires the removal of the caries and/or restoration to achieve a relatively flat root surface without endangering the pulp.



Figure 8. Class I gingival recession-type defect.



Figure 9. Class II gingival recession-type defect.

tissue, and complete root coverage can be achieved (Figure 9).

- Class III. Marginal tissue recession that extends to or beyond the mucogingival junction, and there is loss of interdental bone. The interdental soft tissue is apical to the CEJ but remains coronal to the apical extent of the marginal tissue recession. The teeth may be malpositioned. Only partial root coverage can be achieved to the height of the contour of the interproximal tissue (Figure 10).

- Class IV. Marginal tissue recession that extends beyond the mucogingival junction. There is loss of interdental bone and soft tissue to a level corresponding to the apical extent of the marginal tissue recession or severe malpositioning of the teeth. Root coverage is unpredictable and requires adjunctive (orthodontic) treatment (Figure 11).

Periodontal plastic surgery procedures should be part of the clinician's recipe for restoring the den-

togingival complex. Traditionally, restorative therapy of teeth with gingival recession and carious or noncarious lesions has been achieved through operative procedures with little attention to the overall esthetic picture. In contrast, the perioesthetic approach considers the harmonious integration and interrelationship of the gingiva and tooth complex.

The periodontal plastic surgery procedures available for the treatment and correction of gingival recession



Figure 10. Class III gingival recession-type defect.



Figure 11. Class IV gingival recession-type defect.

include free gingival autografts, subepithelial connective tissue grafts, coronally positioned flaps, guided tissue regeneration, and enamel matrix derivative grafts. These soft tissue grafts are indicated for the restoration of noncarious and carious cervical radicular lesions and for previously restored class V restorations associated with gingival recession.

PERIODONTAL PLASTIC SURGERY PROCEDURES

Periodontal plastic surgery can be divided into two clinical subdivisions that can be identified according to the presence of an adequate or inadequate zone of keratinized attached gingiva. Although infrequent, clinical situations occur in which an adequate zone of keratinized attached tissue exists and gingival recession is present. This requires only a coronal repositioning of the existing gingival tissue with a coronally positioned or a semilunar flap. However, most clinical situations that involve gingival recession also involve a deficiency of keratinized attached gingiva, which requires mucogingival surgery. The first technique was the thick free autogenous gingival graft, presented by Miller, which allowed predictable root coverage around teeth and an increase in attached gingiva and vestibular depth.⁶⁸ The color variation in the earlier technique resulted in the use of thinner grafts, which improved the color and reduced shrinkage of the graft.

The subepithelial connective tissue graft technique, popularized in the 1980s by Langer and Calagna to correct ridge concavities, was modified and combined with a coronally positioned flap to treat gingival recession.⁶⁹⁻⁷¹ This procedure uses collateral blood supply from the mucogingival flap and the periosteum of the recipient bed and produces a better color match as a result of the thinner connective tissue graft and the overlying native mucogingival flap. Although numerous modifications have been made to this technique over the years, it remains the gold standard for root coverage.

For obvious reasons, limited human histologic evidence exists regarding the type of attachment that is achieved when denuded roots are covered by grafts. No one particular technique seems to predictably provide regeneration (new bone, cementum, and insertion of periodontal ligament fibers) more frequently than repair (a soft tissue

adaptation to the root surface). If the etiology is controlled, root coverage grafts tend to remain stable with minimal probing depth regardless of the type of root surface attachment achieved. True regeneration remains the goal, and biomimetics and tissue engineering hold great promise in allowing us to achieve stability on a more predictable basis. These new therapies of the future may require clinicians to “rewrite the rules of the game.”

Case Studies

Surgical Procedure 1. A 23-year-old woman presented in the mid-1980s with a chief complaint of root surface sensitivity on her maxillary canine. She had been advised previously that a class V composite restoration would be the most effective treatment alternative to correct her problem. Oral examination revealed, among other things, that significant parafunctional habits had contributed to the recession, which was classified as Miller’s class I (Figure 12). The



Figure 12. Case study 1. Facial view of the maxillary left cuspid with a preexisting defective composite restoration and a class I gingival recession-type defect.

patient reviewed the benefits and risks of a root coverage graft and consented to treatment. After the construction of a hard acrylic bite guard, the root was scaled and planed under local anesthetic, and citric acid was used to remove the smear layer. A partial-thickness bed was made extending approximately half a tooth's width mesial and distal to the area of recession. The width of the bed on either side of the recession was necessary to provide vascularity to the graft

over the denuded avascular root surface. A thick free autogenous graft was harvested from the palate (Figure 13) and sutured to the bed (Figure 14).

The patient's schedule prevented her from having the contralateral side grafted, and she was not seen at the practice for 5 years. When she returned it was evident that the first treatment had been successful; the denuded root surface was completely covered with the thick free

autogenous graft, and there was no evidence of further recession (Figure 15). The other maxillary canine and lateral incisor had continued to experience gingival recession, and an abfraction-type lesion was noted on the facial aspect of the canine below the CEJ (Figure 16). The patient had not worn her bite guard for the previous 3 years. She was reminded of the importance of wearing the appliance on a consistent basis, and the bite guard was adjusted.

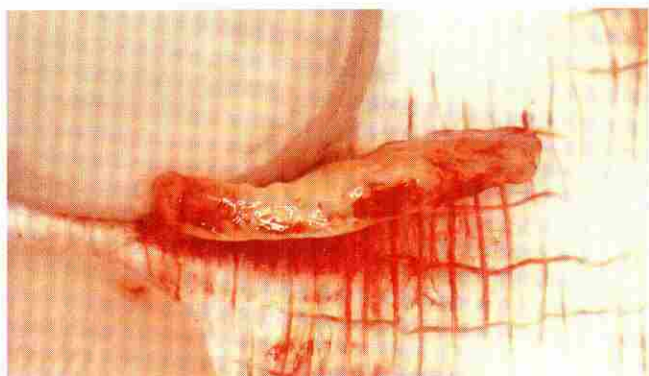


Figure 13. Case study 1. Thick free autogenous graft harvested from the palate.

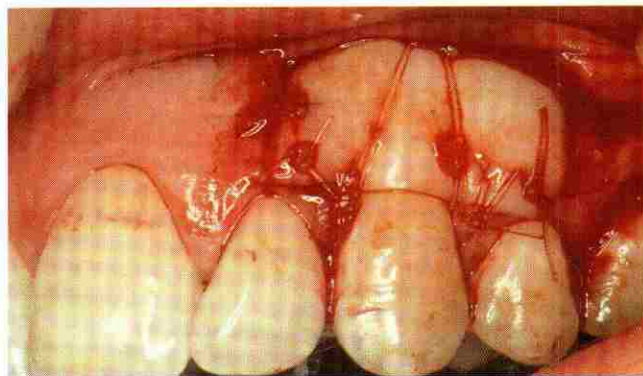


Figure 14. Case study 1. The free autogenous graft is sutured to the recipient bed.



Figure 15. Case study 1. A 5-year postoperative result of the thick autogenous graft with no evidence of further recession.



Figure 16. Case study 1. Facial view of the maxillary right cuspid with an abfraction lesion and a class II gingival recession-type defect.

The root surface of the contralateral maxillary canine was scaled and planed under local anesthetic. The root surface on the lateral incisor was not sensitive and, because of financial constraints, the patient chose only to graft the canine. Newer grafting techniques allowed for a smaller bed limited just to the tooth being grafted. This more conservative bed size was made possible by the development of a connective tissue grafting tech-

nique that preserved the mucogingival flap, which increased blood supply to the graft. Note how much thinner the connective tissue is in this technique (Figure 17) when compared with the free autogenous graft in the older technique (see Figure 13). The connective tissue was sutured over the bed (Figure 18), and the mucogingival flap was coronally advanced over the connective tissue (Figure 19). At 6 months postoperatively, one can

see complete root coverage and a more esthetic result (Figure 20) than in the graft performed on the other maxillary canine 5 years earlier (see Figure 15).

Surgical Procedure 2. A 30-year-old man presented to the office after being told that the class V restoration on his maxillary canine needed to be replaced (Figure 21). As this was the second time in 3 years that the restoration had



Figure 17. Case study 1. Thin connective tissue graft harvested from the palate.

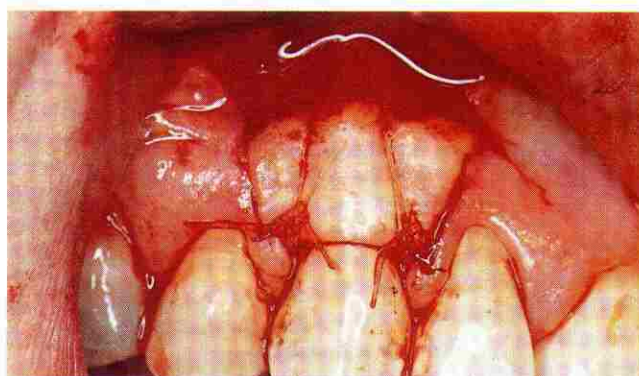


Figure 18. Case study 1. The thin connective tissue graft is sutured over the smaller recipient bed, which is limited to the tooth being grafted.



Figure 19. Case study 1. The mucogingival flap is coronally advanced over the connective tissue and sutured in place.



Figure 20. Case study 1. A 6-month postoperative facial view reveals complete root coverage with a harmonious dento-gingival complex.

required replacement, the patient was seeking an alternative solution. Upon examination the recession was classified as Miller's class II; therefore, one could expect complete root coverage on an unrestored root. The patient was advised of the risks and benefits of the procedure, which included the possibility that a graft might not be possible if it was found that the restoration extended too far axially. That scenario would create a "dead space" too large to be bridged by the graft. The patient accepted the risks and agreed to treatment.

The restoration was removed under local anesthetic, and the class V restorative preparation was eliminated with vigorous root planing using hand instruments and high-speed finishing burs. The area was treated using the same grafting technique as presented in the previous case (see Figures 16 through 20). A 2-year postoperative photograph (Figure 22) shows no further recession and no visual signs of inflammation. Note the improved esthetic outcome with more appropriate tooth length and gingival contours.

Surgical Procedure 3. This 26-year-old male patient presented with Miller's class I recession and caries below the CEJ (Figure 23). After obtaining informed consent, the caries was removed under local anesthetic, and the root surface was scaled and planed, removing any significant dead spaces. Citric acid



Figure 21. Case study 2. Facial view of the maxillary left cuspid with a preexisting defective composite restoration and a class II gingival recession-type defect.



Figure 22. Case study 2. A 2-year postoperative result reveals no further recession and a harmonious soft and hard tissue integration without the use of restorative materials.



Figure 23. Case study 3. Facial view of the maxillary left cuspid with caries apical to the CEJ and a class I gingival recession-type defect.



Figure 24. Case study 3. A 6-month postoperative facial view reveals soft and hard tissue integration without the use of restorative materials.

was burnished onto the root surface, removing the bacterial smear layer, and a connective tissue graft, as previously described, was placed over the root surface. A 6-month postoperative photograph (Figure 24) shows no significant probing depth, and the result is superior both functionally and esthetically to one that could have been achieved with a restoration.

Surgical Procedure 4. A 57-year-old female patient presented to the dental office with concerns of sensitivity on the maxillary left canine. The tooth was found to have Miller's class I recession and cervical abrasion below the CEJ (Figure 25). After restorative examination and consultation, the dentist and patient decided to include evaluation by the periodontist, who felt

that a periodontal approach would restore the balance of the dento-gingival unit. In addition, the restorative team determined that, because of the occlusal wear and history of parafunctional habits, an occlusal guard should be constructed before periodontal surgery ensued. A dual-laminate acrylic occlusal guard (Figure 26) was designed and fabricated with a flat plane of occlusion, so all teeth would touch evenly in all excursions without anterior disclusion.

Upon reviewing the benefits and risks of a root coverage graft, the patient agreed to treatment. Following administration of local anesthetic, a partial-thickness flap was elevated (Figure 27). After scaling and root planing to smooth the abraded root surface, the root was modified with PrefGel™ (Biora AB, Malmö, Sweden), and an enamel matrix derivative, Emdogain® Gel



Figure 25. Case study 4. Facial view of the maxillary left cuspid with cervical abrasion apical to the CEJ and a class I gingival recession-type defect.



Figure 26. Case study 4. A dual-laminate acrylic occlusal guard allows a flat plane of occlusion so that all teeth touch evenly in all excursions without anterior disclusion.

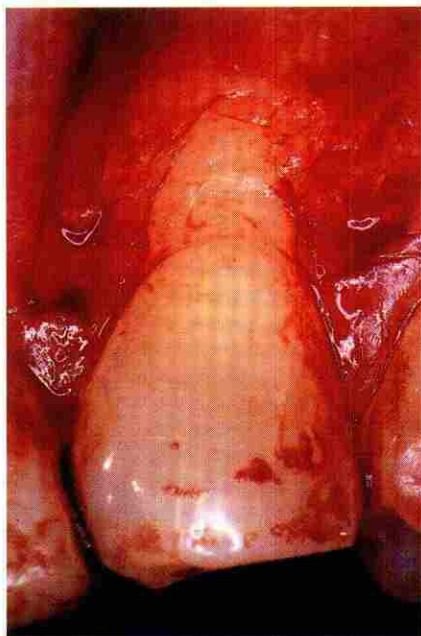


Figure 27. Case study 4. Elevation of a partial-thickness flap.

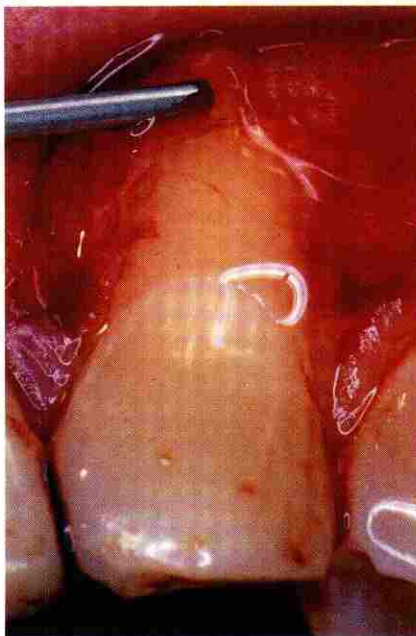


Figure 28. Case study 4. The abraded root surface was biomodified with PrefGel.

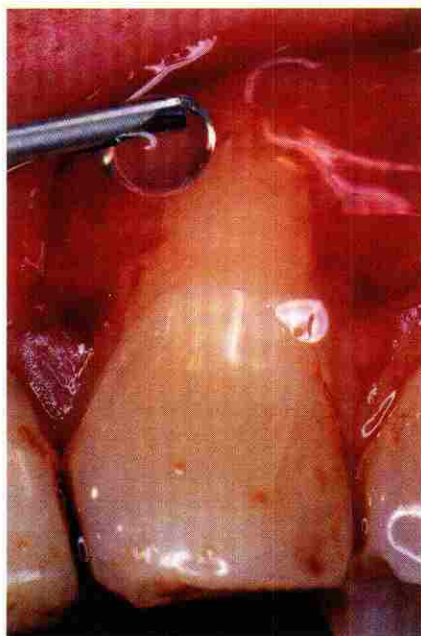


Figure 29. Case study 4. Enamel matrix derivative (Emdogain Gel) was applied to the root surface in an effort to facilitate regeneration.

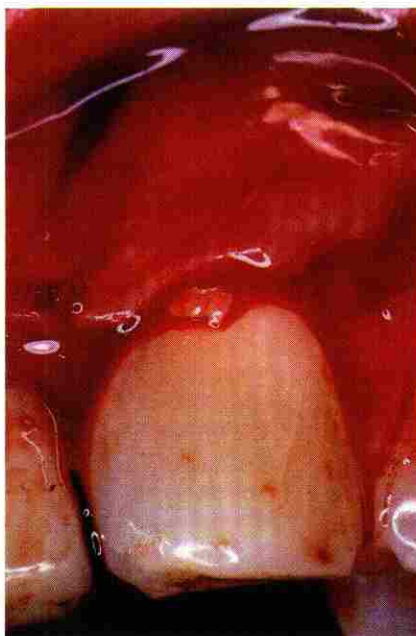


Figure 30. Case study 4. The mucogingival flap was coronally advanced over the denuded root surface.

(Biora AB), was applied to the root surface (Figures 28 and 29) in an effort to facilitate regeneration. PrefGel consists of 24% ethylenediaminetetraacetic acid and removes the smear layer from the root surface. The mucogingival flap was coronally advanced over the denuded root surface (Figure 30) and sutured laterally and interproximally (Figure 31A and B). A 1-year postoperative photograph (Figure 32A and B) demonstrates complete root coverage. The probing depth was < 2 mm. This procedure achieved root coverage without the need for a secondary surgical site to harvest the donor tissue.

CONCLUSIONS

Although management of any clinical situation begins with prevention, knowledge of the etiology of cervical lesions and recession and an understanding of the various therapeutic methods provide the clinician with alternative solutions for the perioesthetic dilemma. As this article illustrates through clinical presentations, often a periodontal rather than an operative approach should be considered when gingival recession is associated with a cervical lesion.

Part II of this article will discuss the operative procedures and restorative materials available for restorative therapy of carious and noncarious cervical lesions.

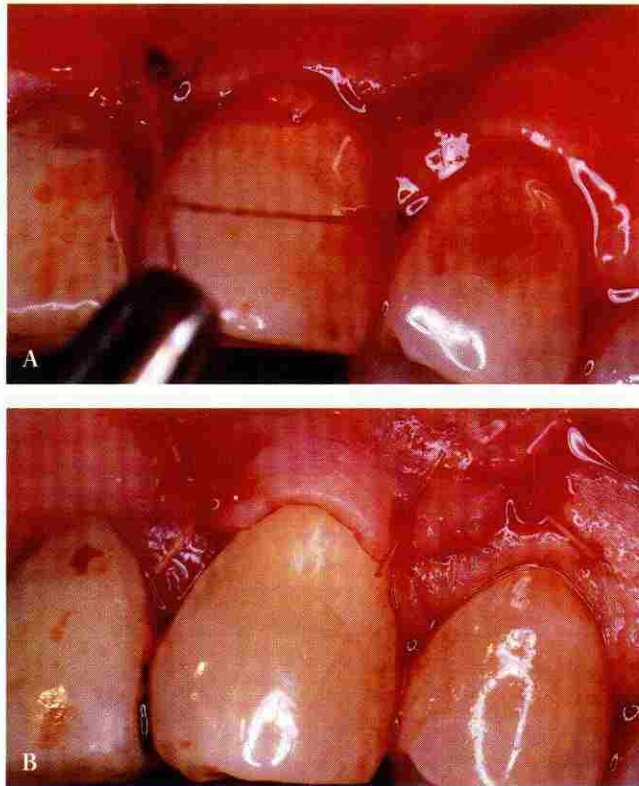


Figure 31. Case study 4. A and B, Suturing of the mucogingival flap laterally and interproximally.

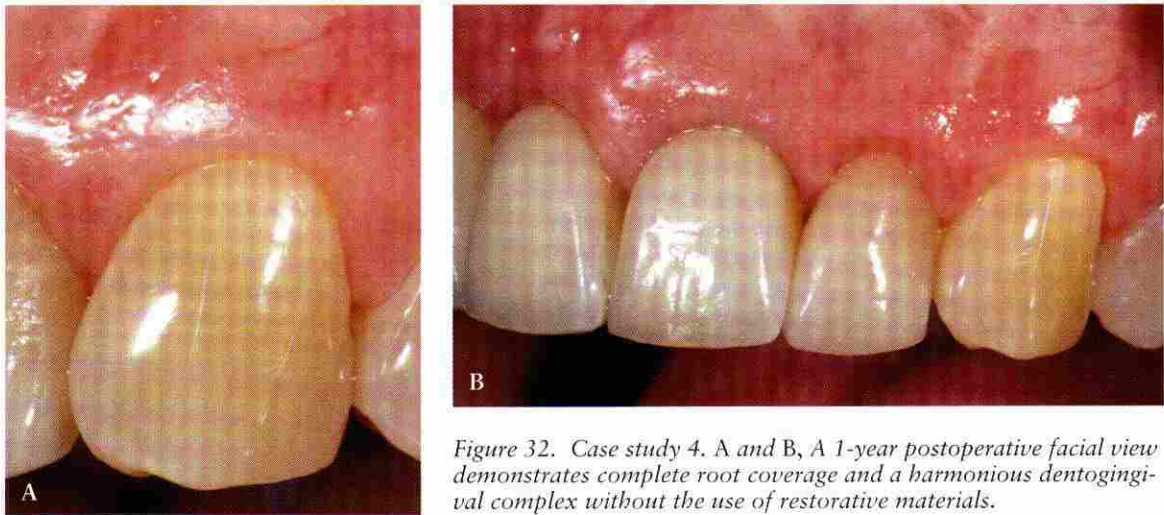


Figure 32. Case study 4. A and B, A 1-year postoperative facial view demonstrates complete root coverage and a harmonious dentogingival complex without the use of restorative materials.

DISCLOSURE

The authors have no financial interest in any of the companies or products mentioned in this article.

REFERENCES

1. Ferrier WL. Clinical observations on erosions and their restoration. *J Calif Dent Assoc* 1931; 7:187-196.
2. McCoy G. On the longevity of teeth. *Oral Implantol* 1983; 11:248-267.
3. Darby ET. Dental erosion and the gouty diathesis: are they usually associated? *Dent Cosmos* 1892; 34:629-640.
4. Miller WD. Experiments and observation on the wasting of tooth tissue variously designated as erosion, abrasion, chemical abrasion, denudation, etc. *Dent Cosmos* 1907; 49:1-23.
5. Kornfeld B. Preliminary report of clinical observations of cervical erosions, a suggested analysis of the cause and treatment for its relief. *Dent Items Int* 1932; 54:905-909.
6. Grippo JO. Abfractions: a new classification of hard tissue lesions of teeth. *J Esthet Dent* 1991; 3:14-19.
7. Imfeld T. Dental erosion. Definition, classification and links. *Eur J Oral Sci* 1996; 104:151-155.
8. Filler SJ, Lazarchik DA. Tooth erosion: an unusual case. *Gen Dent* 1994; 42:568-569.
9. Allan DN. Dental erosion and vomiting: a case report. *Br Dent J* 1969; 126:311-312.
10. Milosevic A, Slade PD. The orodental status of anorexics and bulimics. *Br Dent J* 1989; 167:66-70.
11. White DK, Hayes RC, Benjamin RN. Loss of tooth structure associated with chronic regurgitation and vomiting. *J Am Dent Assoc* 1978; 97:833-835.
12. Kleier DJ, Aragon SB, Averbach RF. Dental management of the chronic vomiting patient. *J Am Dent Assoc* 1984; 108:618-621.
13. Rosenthal P, Rosenthal R. Tooth enamel erosion from vomiting treated with an acrylic sealant. *Clin Pediatr* 1983; 22:818.
14. Simmons NS, Thompson DC. Dental erosion secondary to ethanol-induced emesis. *Oral Surg Oral Med Oral Pathol* 1987; 64:731-733.
15. Howden GF. Erosion as the presenting symptom in hiatus hernia. *Br Dent J* 1971; 131:455-456.
16. Eccles JD, Jenkins WG. Dental erosion and diet. *J Dent* 1974; 2:153-156.
17. Lynch MA, Brightman VJ, Greenburg MS. *Burket's oral medicine*. 8th Ed. Philadelphia: JB Lippincott Co, 1984:568.
18. Holloway PF, Mellanby M, Stewart RJC. Fruit drinks and tooth erosion. *Br Dent J* 1958; 104:305-309.
19. Eccles JD. Tooth surface loss from abrasion, attrition, and erosion. *Dent Update* 1982; 9:373-381.
20. Gallien GS, Kaplan I, Owens BM. A review of noncarious dental cervical lesions. *Compend Contin Educ Dent* 1994; 15:1366-1372.
21. Kaidonis JA, Townsend GC, Richards LC. Abrasion: an evolutionary and clinical view. *Aust Prosthodont J* 1992; 6:9-16.
22. Lee WC, Eakle WS. Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. *J Prosthet Dent* 1984; 52:374-380.
23. McCoy G. The etiology of gingival erosion. *J Oral Implantol* 1982; 10:361-362.
24. Lambrechts P, Van Meerbeek B, Perdigão J, et al. Restorative therapy for erosive lesions. *Eur J Oral Sci* 1996; 104:229-240.
25. Zipkin I, McClure FJ. Salivary citrate and dental erosion. *J Dent Res* 1949; 28:613-626.
26. Shafer WG, Hine MK, Levy BM. Regressive alterations of the teeth. In: *A textbook of oral pathology*. 4th Ed. Philadelphia: WB Saunders Co, 1983:318-339.
27. Shugars DA. Patient assessment, examination and diagnosis, and treatment planning. In: *Sturdevant CM, Barton RE, Sockwell CL, et al, eds. The art and science of operative dentistry*. 2nd Ed. St. Louis: CV Mosby Co, 1985:73-74.
28. Charbeneau GT. Examination, diagnosis, and treatment planning. In: *Principles and practice of operative dentistry*. 3rd Ed. Philadelphia: Lea and Febiger, 1988:25-27.
29. Leinfelder KF. Restoration of abraded lesions. *Compend Contin Educ Dent* 1994; 15:1396-1400.
30. Tyas MJ. The class V lesion—etiology and restoration. *Aust Dent J* 1995; 40:167-170.
31. Braem M, Lambrechts P, Vanherle G. Stress-induced cervical lesions. *J Prosthet Dent* 1992; 67:718-722.
32. Heymann HO, Bayne SC. Current concepts in dentin bonding: focusing on dental adhesion factors. *J Am Dent Assoc* 1993; 124:27-36.
33. Grippo JO. Noncarious cervical lesions: the decision to ignore or restore. *J Esthet Dent* 1992; 4:55-64.
34. McVane TP, Ettinger RL. Periodontal margin in the older adult: considerations for position, placement, and support. *J Esthet Dent* 1991; 3:209-216.
35. Tjan AH, Tan DE. Microleakage at gingival margins of class V composite resin restorations rebonded with various low-viscosity resin systems. *Quintessence Int* 1991; 22:565-573.
36. Toledano M, Perdigão J, Osorio R, Osorio E. Effect of dentin deproteinization on microleakage of class V composite restorations. *Oper Dent* 2000; 25:497-504.
37. Estafan D, Pines MS, Erakin C, et al. Microleakage of class V restorations using two different compomer systems: an in vitro study. *J Clin Dent* 1999; 10:124-126.
38. Powell LV, Gordon GE, Johnson GH. Sensitivity restored of class V abrasion/erosion lesions. *J Am Dent Assoc* 1990; 121:694-696.
39. Folwaczny M, Loher C, Mehl A, et al. Class V lesions restored with four different tooth-colored materials—3 year results. *Clin Oral Investig* 2001; 5:31-39.
40. Miller MB. Restoring class V lesion. Part 1: carious lesions. *Pract Periodontics Aesthet Dent* 1997; 9:441-442.

41. Estafan D, Dusserschleger FL, Miuo LE, et al. Class V lesions restored with flowable composite and added surface sealing resin. *Gen Dent* 2000; 48:78–80.
42. van Dijken JWV. Clinical evaluation of three adhesive systems in class V non-carious lesions. *Dent Mater* 2000; 16:285–291.
43. Ramos RP, Chimello DT, Chinelatti MA, et al. Effect of three surface sealants on marginal sealing of class V composite resin restorations. *Oper Dent* 2000; 25:448–453.
44. Capel-Cardoso PE, Placido E, Francci CE, et al. Microleakage of class V resin-based composite restorations using five simplified adhesive systems. *Am J Dent* 1999; 12:291–294.
45. Hakimeh S, Vaidyanathan J, Houpt M, et al. Microleakage of compomer class V restorations: effect of load cycling, thermal cycling, and cavity shape differences. *J Prosthet Dent* 2000; 83:194–203.
46. Phillips RW. The restoration of eroded cervical areas. *CDS Rev* 1980; 73(4):31–34.
47. Kemp-Scholte CM, Davidson CL. Complete marginal seal of class V resin composite restorations effected by increased flexibility. *J Dent Res* 1990; 69:1240–1243.
48. Krejci I, Lutz F. Marginal adaptation of class V restorations using different restorative techniques. *J Dent* 1991; 19:24–32.
49. Kemp-Scholte CM, Davidson CL. Marginal sealing of curing contraction gaps in class V composite resin restorations. *J Dent Res* 1988; 67:841–845.
50. McCoy RB, Anderson MH, Lepe X, et al. Clinical success of class V composite resin restorations without mechanical retention. *J Am Dent Assoc* 1998; 129:593–599.
51. Mount GJ. Restorations of eroded areas. *J Am Dent Assoc* 1990; 120:31–35.
52. Cox CF. Etiology and treatment of root hypersensitivity. *Am J Dent* 1994; 7:266–270.
53. Salette D, Pini Prato G, Pagliaro U, et al. Coronally advanced flap procedure: in the interdental papilla a prognostic factor for root coverage. *J Periodontol* 2001; 72:760–766.
54. Cvitko E, Denehy GE. Utilization of composite resins and direct bonding following periodontal treatment. *Pract Periodontics Aesthet Dent* 1993; 5(4):33–38.
55. Tugnait A, Clerehugh V. Gingival recession—its significance and management. *J Dent* 2001; 29:381–394.
56. Gillam DG, Newman HN, Bulman JS, et al. Dentifrice abrasivity and cervical dentinal hypersensitivity. Results 12 weeks following cessation of 8 weeks' supervised use. *J Periodontol* 1992; 63:7–12.
57. Xhonga FA, Sognaes RF. Dental erosion: progress of erosion measured clinically after various fluoride applications. *J Am Dent Assoc* 1973; 87:1223–1228.
58. Markowitz K. Tooth sensitivity: mechanisms and managements. *Compend Contin Educ Dent* 1993; 14:1032–1044.
59. Camargo PM, Lagos RA, Lekovic V, et al. Soft tissue root coverage as treatment for cervical abrasion and caries. *Gen Dent* 2001; 49:299–304.
60. Karsten RH, Roeters FJM, Spanauf AJ, et al. Use of combined periodontal and restorative procedures in the comprehensive esthetic treatment of anterior teeth. Report of a case. *Quintessence Int* 1988; 19:149–155.
61. Bruno JF. A subepithelial connective tissue graft procedure for optimum root coverage. *Atlas Oral Maxillofac Surg Clin North Am* 1999; 7(2):11–28.
62. Modica F, Del Pizzo M, Rocuzzo M, et al. Coronally advanced flap for the treatment of buccal gingival recessions with and without enamel matrix derivative. A split-mouth study. *J Periodontol* 2000; 71:1693–1698.
63. Quinones CR. Treatment of gingival recession using guided periodontal tissue regeneration. *Pract Periodontics Aesthet Dent* 1997; 9:145–153.
64. Oringer RJ. Biologic mediators for periodontal and bone regeneration. *Compend Contin Educ Dent* 2002; 23:501–516.
65. Bednar JR, Wise RJ. Interaction of periodontal and orthodontic treatment. In: Nevins M, Mellonig JT, eds. *Periodontal therapy: clinical approaches and evidence of success*. Carol Stream, IL: Quintessence Publishing, 1998:149–164.
66. Pini Prato G, Tinti C, Cortellini P, et al. Periodontal regenerative therapy with coverage of previously restored root surfaces: case reports. *Int J Periodontal Res Dent* 1992; 12:451–461.
67. Miller PD Jr. A classification of marginal tissue recession. *Int J Periodontal Res Dent* 1985; 5(2):9–13.
68. Miller PD. Root coverage using a free soft tissue autograft following citric acid application. Part III. A successful and predictable procedure in areas of deep wide recession. *Int J Periodontal Res Dent* 1985; 5(2):15–37.
69. Langer B, Calagna L. Subepithelial graft to correct ridge concavities. *J Prosthet Dent* 1980; 44:363–367.
70. Langer B, Langer L. Subepithelial connective tissue graft technique for root coverage. *J Periodontol* 1985; 56:715–720.
71. Langer B, Calagna LJ. The subepithelial connective tissue graft: a new approach to the enhancement of anterior cosmetics. *Int J Periodontal Res Dent* 1982; 2(2):23–34.

Reprint requests: Douglas A. Terry, DDS, 12050 Beamer Street, Houston, TX, USA 77089; e-mail: dterry@dentalinstitute.com
©2003 BC Decker Inc

