Guided Tissue Regeneration Associated With Orthodontic Therapy

Peter R. Diedrich

Guided tissue regeneration (GTR) to enhance genuine new periodontal attachment may improve preorthodontic conditions for moving teeth into infrabony defects or for vertical movements of teeth with reduced bone support. The possible benefits of GTR for combined periodontal/orthodontic therapy are discussed and substantiated with preliminary experimental findings. (Semin Orthod 1996;2:39-45.) ***Copyright © 1996 by W.B. Saunders Company***

In the orthodontic treatment of patients with advanced periodontitis, the crucial issue is often to what extent the osseous topography can be favorably influenced by the orthodontic tooth movement. The preorthodontic periodontal conditions can be characterized by infrabony defects, furcation involvement, interproximal craters, inconsistent margins, and hard and soft tissue dehiscences. Questions may be posed as to what effects on the epithelial and connective tissue attachment result from:

1. Bodily or tipping tooth movement into an intraosseous defect. Can the bony lesion be reduced or eliminated?
2. Moving teeth away from an intraosseous defect (extrusion or uprighting of inclined teeth with one- or two-wall bony pockets)?
3. Intruding migrated elongated teeth back into the reduced alveolar bone?

The answer to these questions is closely correlated with the type of attachment created by the preorthodontic periodontal therapy. In the past, efforts were made to induce periodontal regeneration with traditional methods (open or closed curettage, mechanical or chemical root preparation, filling of intraosseus defects with grafts).

This periodontal therapeutic objective has remained elusive. No matter which technique was used, the healing of a periodontal lesion resulted not in regeneration but in various repair mechanisms such as the formation of a long epithelial attachment, ankylosis or root resorption.[1-5] This data confirms the results of previously published experimental and clinical studies dealing with the orthodontic movement of periodontally affected teeth.

Polson et al[6] studied periodontal response after bodily tooth movement into infrabony defects. Radiological examination of the alveolar bone adjacent to the moved teeth no longer showed angular defect morphology on the pressure side, but histological examination showed a thin epithelial layer interposed between the root surface and the bone, with epithelium terminating at the apical limit of root instrumentation. On the tension side the crest of bone was located apical to the level of the root planning. It was concluded that orthodontic tooth movement into infrabony periodontal defects had no favorable effects on the level of connective tissue attachment. However, it is possible to move teeth with reduced healthy periodontium without attachment loss. This finding was confirmed by the experimental data published by Ericsson et al.[7] Wennström et al[8] reported from an animal study that bodily orthodontic movement of plaque-infected teeth with infrabony pockets enhances the rate of destruction of connective tissue attachment. It was concluded that moving teeth into and through inflamed intraosseous lesions creates a high risk of additional attachment loss.

Orthodontic extrusion of teeth with in-

From the Department of Orthodontics, Medical Faculty of the University Aachen, Germany.

Address correspondence to Peter R. Diedrich, DM, DMD, Head of the Department of Orthodontics, Pauwelstr. 30, D-52057 Aachen, Germany.

1073-8746/96/0201-0006$5.00/0

frabony pockets is unproblematic. Ingber[9] recommends this technique with one-wall and two-wall bony pockets that are difficult to treat by means of conventional periodontal therapy alone. The extrusive tooth movement leads to a coronal positioning of intact connective tissue attachment and the bony defect is shallowed out. The orthodontically induced improvement of the crestal bone structure has been reported in clinical trials[10-13] and animal experiments.[14]

The extrusive component is also the key factor in uprighting tipped molars with a mesial angular bony lesion. In these situations, moving the tooth away from the osseous defect in a disto-occlusal direction shallows out the slanted contour of the alveolar crest.[15-21] Because of the tension of collagen fibers in the periodontal ligament, the alveolar bone follows the moving tooth on the mesial side, with the level of connective tissue attachment remaining unchanged. If the tipped molars have furcation involvement before orthodontic uprighting, simultaneous extrusion may increase the severity of the furcation defects, especially in the presence of periodontal inflammation.[22,23]

The intrusion of teeth with horizontal bone loss or infrabony pockets raises the question of whether attachment gain is possible. Although the positive periodontal effects of orthodontic extrusion can be predicted with certainty, the benefits of intrusive movement is controversial. The intrusion of plaque-contaminated teeth leads to the formation of angular bony defects and increased loss of attachment Ericsson et al[24,25] confirmed that tipping of a plaque-infected tooth may shift supragingivally located plaque into a subgingival position, resulting in periodontal tissue breakdown. Even in a healthy periodontal environment the question remains as to whether the orthodontic tooth movement intrudes a long epithelial attachment beneath the margin of the alveolar bone or whether the alveolar crest is continuously resorbed in front of the intruding tooth. Vanarsdall[19] recommends extrusion as more predictable in solving periodontal problems. Clinical and histological studies by Melsen[26-29] contradict this concept. In an animal experiment[28] periodontal tissue breakdown was induced on the test teeth and intrusion was initiated following conventional flap surgery. In addition to the orthodontic effects, the influence of marginal inflammation was evaluated. Histological analysis showed new cementum formation and connective tissue attachment on the intruded teeth if a healthy gingival environment was provided throughout the tooth movement. In the event of intrusion in the presence of periodontal inflammation, histological results varied from moderate new attachment to aggravation of periodontal bone loss.

A subsequent clinical investigation[29] considered the preorthodontic and postorthodontic periodontal status following the intrusion of extruded and spaced incisors in patients who had severe periodontal disease. Judging by the clinical (probing depth) and radiological level there was a pronounced beneficial effect on the periodontal status. The clinical crown length was reduced and the marginal bone level approached the cemento-enamel junction. The total amount of alveolar bone support was unaltered or increased.

All reported clinical and histological findings relating to a combined orthodontic-periodontal approach must be assessed from the viewpoint that conventional periodontal treatment does not succeed in regeneration, but in the repair of lost periodontal tissue. The new techniques of guided tissue regeneration (GTR) have virtually revolutionized periodontal therapy. Melcher's[30] vision of extensive restoration of lost attachment seems to be within grasp.

In light of these possibilities, orthodontic techniques in the periodontally diseased dentitions must also be reconsidered.

Guided Tissue Regeneration: Altered Preorthodontic Conditions

The fundamental change in the healing of periodontal lesions is the use of a barrier membrane to prevent cells of the gingival connective tissue and the epithelium from colonizing the decontaminated root surface. Cells of the residual periodontal ligament and from bone marrow spaces are allowed to initiate genuine periodontal regeneration,[3,4,30-34] and a new cementum layer with inserting collagen fibres and new alveolar bone. There is still controversy as to the role of additional bone grafting materials to support the formation of the new attachment.[35]

Long-term studies by Schallhorn and McClain[36] compared the treatment of severe periodontal defects (furcation defects degree II and

III, bone dehiscences, wide infrabony defects, horizontal bone loss) with membrane material alone and with composite osseus grafting and citric acid root conditioning. The combined membrane-grafting technique produced better clinical results with complete furcation fill found in 72% of the defects. Partial apposition of alveolar bone was noted in dehiscences and horizontal defects with combined therapy, but not at membrane-only sites.

The milipore filter of regenerative membrane material was e-PTFE (GoreTex, Gore Company), which had the drawback of having to be removed at a later time in a second intervention. More satisfactory results have recently been obtained with polymer-based biodegradable membranes (Vicryl, Guidor). It is conceivable that complete periodontal regeneration, even in cases of one-wall infrabony pockets or horizontal bone loss, may be achieved in the future with the additional use of bone morphogenetic proteins,[37] cultured alveolar bone or periodontal ligament (PDL) cells.[38]

The advancement of the techniques of GTR provides a distinct improvement in orthodontic therapy in the periodontally compromised patient. New supracrestal and PDL collagen fibers are gained on the tension side which can transfer the orthodontic force stimulus to the alveolar bone. This might be advantageous for extrusion or intrusion of teeth with intraosseous defects and for the uprighting of tipped molars with mesial angular lesions. It is also conceivable that an over erupted molar tooth with severe furcation involvement might be intruded, thus solving the furcation problem. The findings published by Polson[36] should also be reconsidered in the light of the GTR technique. If preorthodontic measures can prevent the epithelium from proliferating apically, a bodily tooth movement into or through an intraosseus defect might be promising.

A variant of GTR is the restoration of lost bone around implants or in constricted bone areas in preparation for implantation.[39-41] This guided bone regeneration (GBR) might also be useful for an orthodontic movement of teeth into a long-standing atrophied alveolar process. Experimental reports[42,43] and clinical studies[44] have shown that a reduction in vertical bone height is not a contraindication for orthodontic tooth movement and that bone height is recreated ahead of the moving tooth. Computer tomography analysis[45] and human histological findings[46] indicate that buccal or lingual bone dehiscences may be provoked by tooth movement into an area of reduced bone width. These defects are not revealed by conventional radiological diagnosis.

Preorthodontic GBR of markedly constricted alveolar ridges also has the advantage that tooth movement through cancellous bone is easier and less detrimental and the formation of interfering gingival invaginations is avoided. The GTR-technique with its wide-ranging new perspectives provides a fruitful field for further orthodontic investigations.

Experimental Pilot Study

So far, relatively little has been known about the benefits of the GTR technique in orthodontic therapy. Diedrich et al[47] published a preliminary pilot experiment on dogs in which the intrusive effects on periodontally affected teeth following conventional flap surgery and GTR were compared. A group of teeth with flap surgery served as a control. Polyglactin 910 (Vicryl) a bioresorbable polymer, was used as membrane material (Fig 1). During flap surgery the level of the initial osseous crest was marked by a reference notch in the root surface. In addition, nonstained undecalcified sections were examined by fluorescence microscopy to reveal the remodelling bone dynamics and formation of new cementum. Stains with calcium affinity had been administered intravitally at different time intervals using the polychromatic sequential marking technique.

Within the flap operation plus intrusion group, apical positioning of a long epithelial attachment was the rule. Only 6 of 24 evaluated root areas showed an attachment gain and bone support, ie, new cementum and functionally oriented PDL fibres extending beneath the alveolar crest. In areas with pronounced gingival inflammation the bone level was generally located apical of the marking notch, ie, continous reduction of the alveolar bone margin occurred parallel to the intrusive tooth movement (Fig 2). This correlation between gingival inflammation and nonformation of new attachment was also evident in the GTR plus intrusion group.

However, in all root sections with minimal or

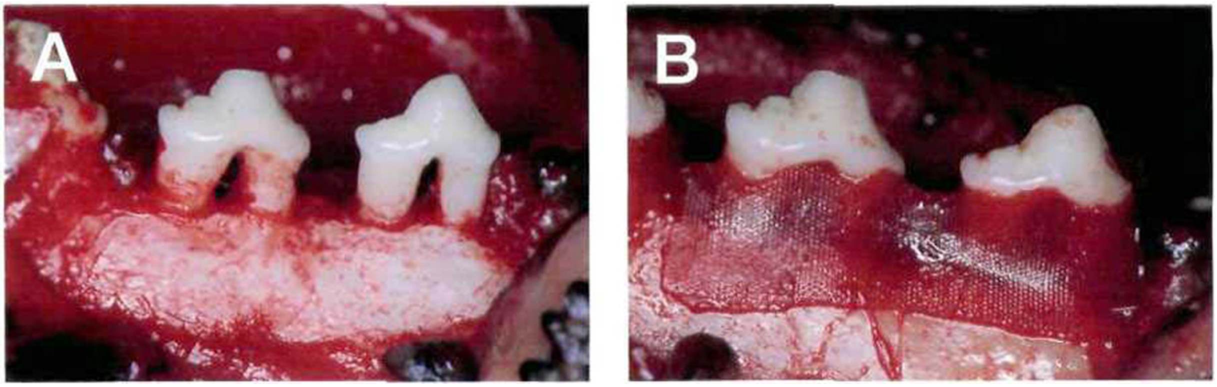

Figure 1. (A) Open debridement after experimentally induced periodontitis, approximal and interradicular bone loss. (B) Before flap repositioning the curetted root surfaces are covered with a Vicryl membrane. After initial wound healing (6 weeks) intrusion was initiated.

no signs of round cell infiltration the marking notch was located beneath the alveolar margin and new attachment was formed. The potential of the intrusive/regenerative mechanism was most impressive within the interradicular area. Fifty percent of the test teeth displayed complete reconstruction of bone septum and periodontal ligament. The polychromatic bone labeling tech-

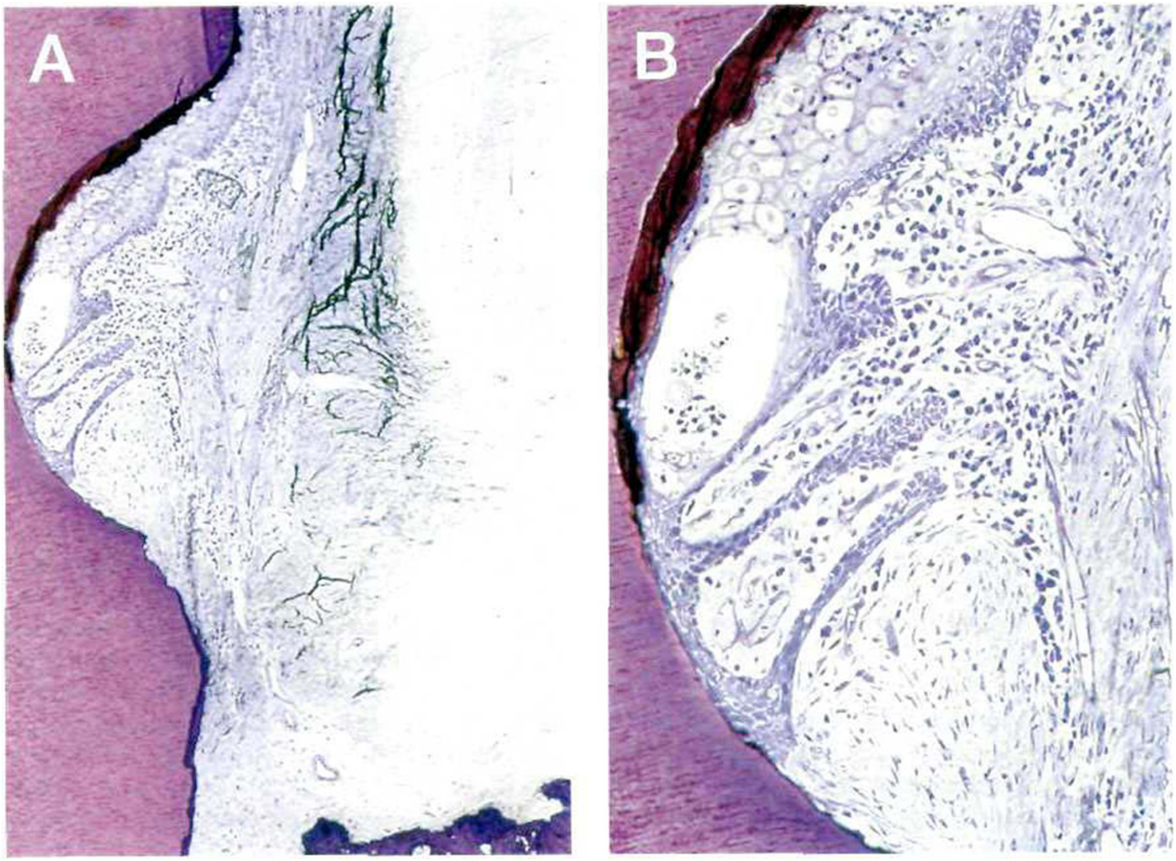

Figure 2. (A) Intruded approximal root area with pronounced round cell infiltration in the gingival connective tissue. Despite intrusion, the marking notch is located above the bone level caused by progressive bone destruction (toluidin blue staining; original magnification ×250). (B) Higher magnification: proliferating junctional epithelium within the notch area.

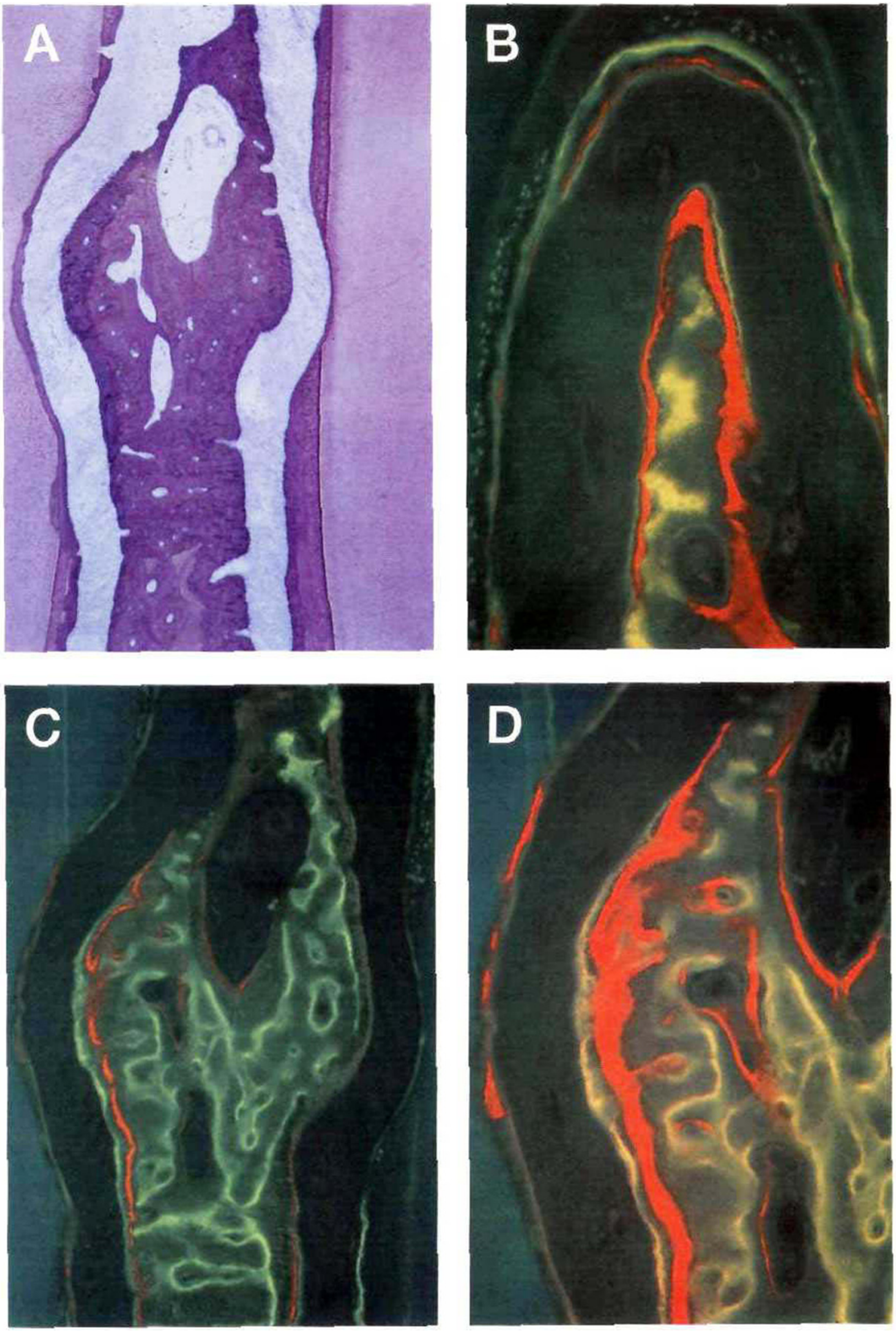

Figure 3. (A) Interradicular finding after GTR and intrusion: completely reconstructed bone in the notch area up to the fornix of the furcation (toluidin blue staining; original magnification ×200). (B) The polychromatic calcium-labeling technique shows pronounced new formation of bone and cementum in the fornix of furcation (fluorescence microscopy; original magnification ×400). (C) Extensive bone remodeling processes in the septum, notch area (fluorescence microscopy; original magnification ×320). (D) Higher magnification of the periodontal ligament and interradicular bone in the notch area: new cementum layer and appositional bone formation (fluorescence microscopy; original magnification ×400).

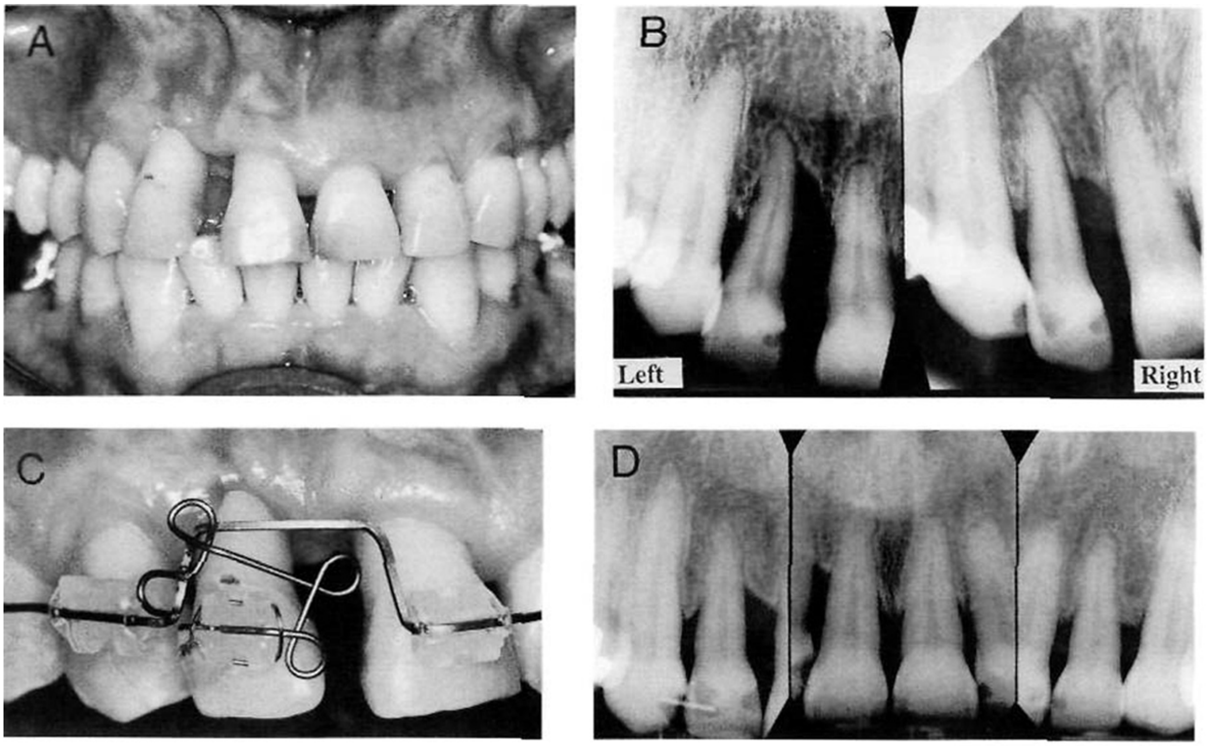

Figure 4. (A) Pathological tooth migration as a result of an advanced periodontal lesion (female patient aged 56 years). (B) Severe intraosseus defect between the right central and lateral incisors (left radiograph). Three months after GTR-treatment (GoreTex membrane) partial reossification is evident, possibly new attachement (right radiograph). (C) A calculated force system from a TMA sectional wire controls space closure and intrusion of the lateral incisor. (D) Retention period after 6 months of orthodontic tooth movement indicating no root resorption and a consolidated alveolar crest. For permanent stabilization a titanium pin was inserted from canine to incisor.

nique proved that the functionally oriented PDL collagen fibers inserted into the newly formed cementum (Fig 3). These positive findings should be substantiated by further studies dealing with other three-dimensional tooth movement. Initial clinical observations (Fig 4) already indicate that GTR enriches the therapeutic spectrum not only in periodontics but also in a combined periodontal/orthodontic approach.

References

1. Caton J, Nymann S. Histrometic evaluation of periodontal surgery. I. The modified widman flap procedure. J Clin Periodontol 1980;7:212-217.
2. Karring T, Nyman S, Linde J. Healing of implantation of periodontitis affected roots into bone tissue. J Clin Periodontol 1980;7:96-105.
3. Karring T, Nyman S, Linde J, et al. Potentials for root resorption during periodontal healing. J Clin Periodontol 1984;11:41-42.
4. Nymann S, Karring T, Linde J, et al. Healing following implantation of periodontitis-affected roots into gingival connective tissue. J Clin Periodontol 1980;7:394-401.
5. Yukma RA. A clinical and histologic study of healing following the excisional new attachment procedure in rhesus monkeys. J Periodontol 1976;47:701-709.
6. Polson A, Caton J, Polson AP, et al. Periodontal response after tooth movement into intrabony defects. J Periodontol 1984;55:197-202.
7. Ericsson I, Thilander B, Linde J. Periodontal conditions after orthodontic tooth movements in the dog. Angle Orthod 1978;48:210-218.
8. Wennström JL, Lindskog-Stokland B, Nyman S, et al. Periodontal tissue response to orthodontic movement of teeth with infrabony pockets. Am J Orthod Dentofacial Orthop 1993;103:313-319.
9. Ingber J. Forced eruption. Part I. A method of treating isolated one and two-wall intrabony osseus defect—rationale and case report. J Periodontol 1974;45:199-206.
10. Diedrich P. Die Unterstützung der Parodontalbehandlung durch kieferorthopädische Maßnahmen im Erwachsenengebiß. Dtsch Zahnärztl Z 1984;39:570-580.
11. Vanarsdall RL. Uprighting the inclined mandibular molar in preparation for restorative treatment. Contin Dent Educ, vol 1, no 2, University of Pennsylvania Press, Philadelphia, 1977
12. Wagenberg BD, Eskow RN, Langner B. Orthodontic procedures that improve periodontal prognosis. J Am Dent Assoc 1980;100:370-373.

13. Wagenberg BD, Eskow RN, Langner B. Orthodontics: A solution for the advanced periodontal or restorative problem. Int J Periodont Res Dent 1986;6:37-45.
14. Van Venrooy JR, Yukna RM. Orthodontic extrusion of single-rooted teeth affected with advanced periodontal disease. Am J Orthod 1985;87:67-74.
15. Brown IS. The effect of orthodontic therapy on certain types of periodontal defects. J Periodontol 1973;44:742-754.
16. Diedrich P. Die Aufrichtung gekippter Molaren als präprothetische und parodontitis prophylaktische Maβnahme. Dtsch Zahnärztl Z 1986;41:159-163.
17. Marks MH. Tooth movement in periodontal therapy. In: Goldman H, Cohen DW, editors. Periodontal therapy (ed 6). St Louis, MO: Mosby 1980:564-627.
18. Marks MH, Rosen PS. Adult Orthodontics: Periodontics and cosmetic enhancements. Compend Contin Educ Dent 1991;12:584-593.
19. Vanarsdall RL. La réaction de tissus parodontaux aux movements orthodontiques. Orthodont Francaise 1986; 57:421-433.
20. Wehrbein H, Diedrich P. Parodontalbefunde bei der Aufrichtung gekippter Molaren Dtsch Zahnärztl Z 1992; 47:326-329.
21. Wise RJ, Kramer GM. Über die Bewertung und Einschätzung von Knochenveränderungen durch gezielte Sondierung nach Aufrichtung gekippter Molaren. Int J Parodont Restaurat Zahnheil 1983;1:69-81.
22. Burch JG, Bagci B, Sabulski D, et al. Periodontal changes in furcations resulting from orthodontic uprighting of mandibular molars. Quintessence Int 1992;23:509-513.
23. Roberts WW, Chacker FM, Burstone CJ. A segmental approach to mandibular molars uprighting. Am J Orthod 1982;81:177-184.
24. Ericsson I, Thilander B, Linde J, et al. The effect of orthodontic tilting movements on the periodontal tissues of infected and non-infected dentitions in dogs. J Clin Periodontol 1977;4:278-293.
25. Ericsson I. The combined effects of plaque and physical stress on the periodontal tissues. J Clin Periodontol 1986;13:918-922.
26. Melsen B. Tissue reaction following application of extrusive and intrusive forces on teeth in adult monkeys. Am J Orthod 1986;89:469-475.
27. Melsen B. Traitement orthodontique de patients présontant de lésions parodontales. J Parodontol 1987;6:285-296.
28. Melsen B, Agerbæk N, Eriksen J, et al. New attachment through periodontal treatment and orthodontic intrusion. Am J Orthod 1988;94:104-116.
29. Melsen B, Agerbæk N, Makenstam G. Intrusion of incisors in adult patients with marginal bone loss. Am J Orthod Dentofacial Orthop 1989;96:232-241.
30. Melcher AH. On the repair potential of periodontal tissues. J Periodontol 1976;47:256-260.
31. Becker W, Becker BE, Prichard JF, et al. Root isolation for new attachment procedures-surgical and suturing method: Three case reports. J Periodontol 1987;58:819-826.
32. Gotlow J, Nyman S, Karring T, et al. New attachment formation as the result of controlled tissue regeneration. J Clin Periodontol 1984;11:494-503.
33. Gotlow J, Nyman S, Linde J, et al. New attachment formation in the human periodontium by guided tissue regeneration. J Clin Periodontol 1986;13:604-616.
34. Nyman S, Gottlow J, Linde J, et al. New attachment formation by guided tissue regeneration. J Periodont Res 1987;22:252-254.
35. Bowers GM, Chadrof B, Carnevale R, et al. Histologic evoluation of new attachment aparatus formation in humans Part I, II, III. J Periodontol 1989;60:664-693.
36. Schallhorn RG, McClain PK. Combined osseus composit grafting, root conditioning and guided tissue regeneration. Int J Periodont Rest Dent 1988;4:9-13.
37. Bowers G, Felton F, Middleton C, et al. Histologic comparison of regeneration in human infrabony defects when osteogenin is combined with demineralized freeze-dried bone allograft and with purified bovine collagen. J Periodontol 1991;62:690-702.
38. Lang H. Parodontale Regeneration nach Implantation kultivierter Zellen. Dtsch Zahnärztl Z 1995;50:190-199.
39. Buser D, Dula K, Belser U, et al. Localized ridge augmentation using guided bone regeneration. Int J Periodont Rest Dent 1993;13:29-45.
40. Caudill RF, Meffert RM. Histologic analysis of the osseointegration of endosseous implants in simulated extraction sockets with and without e-PTFE barriers. Part I. Preliminary findings. Int J Periodont Rest Dent 1991;11: 207-215.
41. Nevins M, Mellonig JT. Enhancement of the damaged edentulous ridge to receive dental implants: Combination of allograft and the gore-tex membrane. Int J Periodont Rest Dent 1992;12:97-111.
42. Lindskog-Stokland B, Wennström JL, Nymann S, et al. Orthodontic tooth movement into edentulous areas with reduced bone height. An experimental study in the dog. Eur J Orthod 1993;15:89-96.
43. Wehrbein H, Rieβ H, Mayer R, et al. Körperliche Zahnbewegung in atrophierte Kieferabschnitte. Dtsch Zahnärztl Z 1990;45:168-171.
44. Hom BM, Turley PK. The effects of space closure of the mandibular first molar area in adults. Am J Orthodont 1984;85:457-469.
45. Fuhrmann RAW, Bücker A, Diedrich PR. Assessment of alveolar bone loss with high resolution computed tomography. J Periodont Res 1995;30:258-263.
46. Wehrbein H, Fuhrmann RAW, Diedrich PR. Human histologic tissue response after long-term orthodontic tooth movement. Am J Orthod Dentofacial Orthop 1995;107:360-371.
47. Diedrich P, Wehrbein H, Schneider B. Zur Problematik der orthodontischen Intrusion parodontal erkrankter Zähne. Parodontologie 1992;2:87-102.