Treatment of Juvenile Periodontitis Patients by Control of Infection and Inflammation

Four Case Reports

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FOUR PATIENTS, AGES 14–17, who demonstrated characteristics of juvenile periodontitis, underwent antibiotic and surgical therapy to control microbial etiologic factors. No occlusal equilibration or bone grafting techniques were employed. Subsequent to treatment, all four patients demonstrated decreased pocket depths and mobility on teeth associated with vertical osseous defects. Osseous repair was evident on postoperative radiographs. Reentry procedures, on one patient, confirmed that osseous repair had occurred in 2- to 3-wall, 3-wall, hemi-circumferential and furcal defects. If infection and inflammation are controlled, it appears that the potential for osseous repair in juvenile periodontitis patients is greater than has been thought.

Juvenile periodontitis, formerly referred to as diffuse alveolar atrophy,[1] paradontosis[2] and periodontosis,[3] affects a small percentage of the population. The *osis* ending was used to connote a degenerative disease, because the degree of osseous destruction was out of proportion to the amount of inflammation present.[2,3] In 1966, however, the World Workshop in Periodontics concluded that no clinical or histologic evidence existed to support the contention that this was a degenerative disease.[4] Subsequently, the term juvenile periodontitis (JP) was introduced into the literature by Butler to indicate advanced alveolar bone loss in young, healthy individuals.[5]

The precise etiology that would account for the distinctive characteristics of JP is unknown.[6] A familial tendency has been noted[5,7] and a genetic cause has been suggested by several authors.[5,8] A systemic basis has been discounted[9] and oral manifestations of systemic diseases should not be confused with idiopathic JP.[10–12] Despite a paucity of supragingival plaque and clinical signs of inflammation, Baer[6] and Waerhaug[13] consistently found subgingival plaque along the root surfaces of JP patients, supporting the concept that JP has a bacterial etiology. Current studies have demonstrated an association between certain Gram-negative microorganisms and JP.[14,15] Tanner et al.[16] have identified a Gram-negative anaerobic rod, *Actinobacillus actinomycetemcomitans* (*AA*) which has been correlated with rapid alveolar bone loss in young individuals. Additional evidence for the bacterial etiology of JP has been provided (1) by studies in gnotobiotic animals where extensive alveolar bone loss has been induced by *AA*[17] and (2) as a result of immunologic testing which has indicated elevated levels of AA antibody in patients susceptible to JP.[18,19]

Lack of clinical inflammation led early investigators to hypothesize that there was an occlusal component in the etiology of JP.[2,20] While it is generally accepted that occlusal trauma cannot initiate periodontal disease,[21–23] the interaction between inflammation and occlusal trauma is still a controversial issue in dentistry.[24] Inability of clinicians to clinically determine the relative influence of occlusal trauma or inflammation on hypermobile teeth results in confusion as to what extent occlusal therapy needs to be implemented.

Therapeutic management of JP has consisted of clinical procedures similar to those used in treating chronic periodontitis. Recent reports[25–27] of successful results in JP cases have involved various therapies: control of inflammation, bone grafting and occlusal management. The cases presented here involved treatment aimed at control of microbial etiologic factors only, without the use of bone grafting material and/or occlusal therapy.

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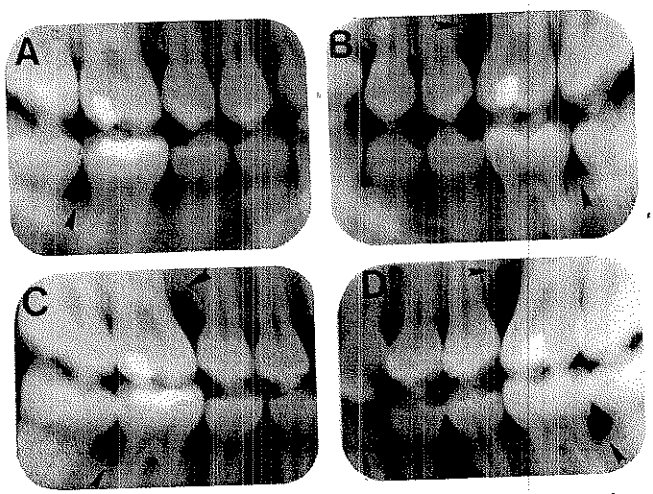

Figure 1. *Case #1, bitewing radiographs revealing progressive bone loss (arrows) on mesial of maxillary and distal of mandibular first molars.* **A,B.** *April 1974.* **C,D.** *June 1975.*

Case #1

In November 1976, a 14-year-old white male was evaluated after his pedodontist noticed progressive alveolar bone loss (Fig. 1, A–D). Medical history was unremarkable, with the exception of a sulfa allergy which had manifested as erythema multiforme. During the previous 7 years, dental history had consisted of routine operative care. Extraoral examination revealed no abnormalities. The gingiva was pink, with some areas of marginal edema and red granulomatous proliferations on the mesial of the maxillary first molars and right lateral incisor. Seven- to 10-mm pocket depths associated with these masses and 8- to 10-mm pockets on the distal and lingual of the mandibular first molars (Table 1) corresponded with radiographic osseous defects (Figs. 2, 3, A, C, E and 4A). Teeth adjacent to these defects were hypermobile (Table 2). The patient's age, location and depth of pockets, lack of clinically detectable calculus, plaque and inflammation all supported a diagnosis of juvenile periodontitis in this and subsequent cases presented.

Therapy involved an initial course of oral tetracycline, 250 mg q.i.d. for 28 days and oral hygiene instruction. This was followed by surgery in the affected sites. Full thickness buccal and partial thickness palatal flaps were dissected in the maxilla, and full thickness buccal and lingual flaps in the mandible were elevated to facilitate debridement of osseous defects and root planing. A class II furcation* involvement was noted on the mesial and a class I involvement was found on the buccal of the maxillary right first molar. On the mesial, a 2–3 wall osseous lesion measured 8 mm from the alveolar crest to the base of the defect (Fig. 5A). The right lateral incisor had a 5-mm deep, 3-wall defect on the mesial. The lesion on the mesiobuccal of the maxillary left first molar reached the apex of the root and extended interradicularly, involving all three furcations. The tooth had a hopeless prognosis and was extracted. In the mandibular arch, bilateral symmetrical moat-like defects were noted on the distobuccal of the first molars, which extended around to the lingual furcations (Fig. 6A). After debridement, flaps were sutured at the alveolar crest, intentionally leaving the osseous defects denuded.

Postoperatively, the patient received tetracycline (250 mg q.i.d.) for 2 weeks and an analgesic. One year postoperatively, reentry procedures were performed to complete pocket elimination.

Case #2

The 17-year-old brother of Case #1 was referred for evaluation in April 1981. Medical history revealed no abnormalities and dental history consisted of routine care which included orthodontics and extraction of the first bicuspids. Minimal calculus, plaque and clinical inflammation were observed. The maxillary right first molar was hypermobile (Table 2) and had a 10-mm pocket on the mesial (Table 1) which correlated with a radiographic osseous defect (Fig. 7A). All other crestal areas appeared normal. The area with the lesion was

Table 1
Pocket Depths (mm) Associated With Osseous Defects

Case	Tooth No.*	Surface	Pocket Depth	
			Preoperative	Postoperative
#1	3	MB	7	3
		ML	8	3
	7	MB	7	2
		ML	7	2
	14	MB	10	
		ML	10+	
	19	DB	10	2
		DL	10	3
		L	8	2
	30	DB	10	2
		DL	10	3
		L	8	2
#2	3	MB	9	2
		ML	10	3
#3	3	MB	9	3
		ML	10	4
	8	D	7	3
	14	MB	5	4
		ML	7	4
	19	MB	10	4
		ML	10	4
		L	6	2
	30	MB	10	3
		ML	10	4
		L	6	2
#4	3	MB	10	3
		ML	10	4
	14	MB	8	3
		ML	9	3

* Teeth No. 1–32 (universal numbering system).

* Furcations described according to Hamp et al.[29]

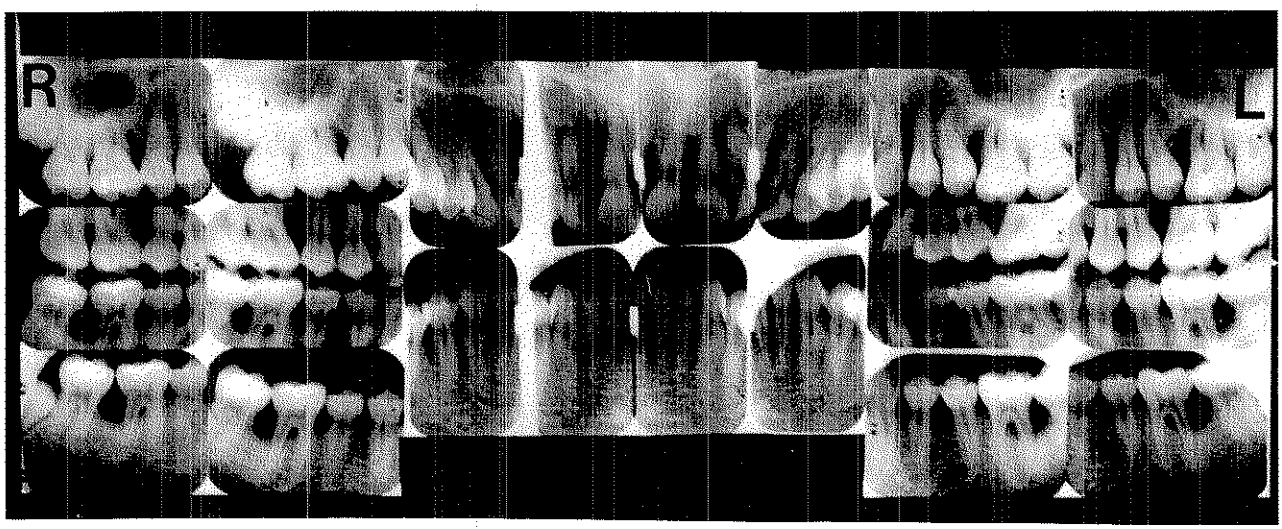

Figure 2. *Case #1, radiographs at initial visit, November 1976.* R = *right;* L = *left.*

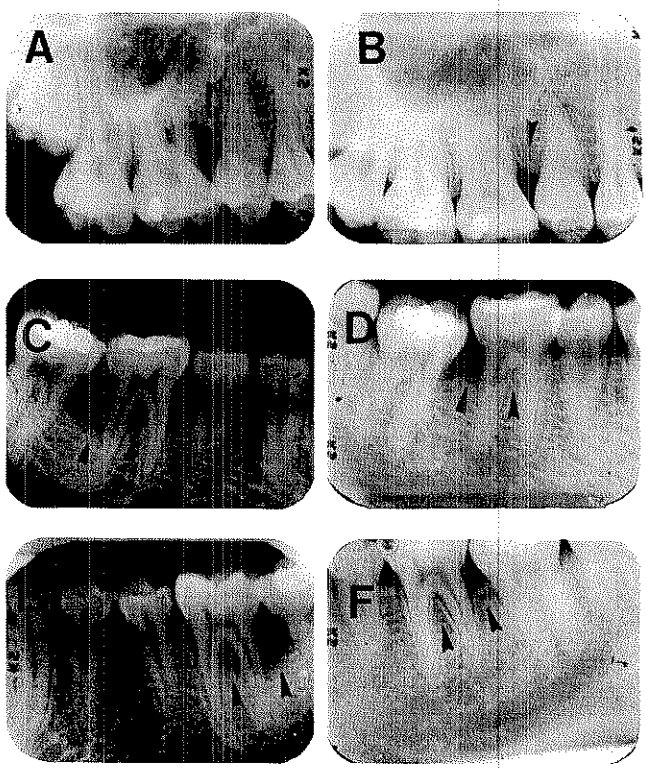

Figure 3. A,C,E. *Case #1, radiographs with vertical defects, November 1976.* **B,D,F.** *Case #1, radiographs demonstrating osseous repair, May 1981.*

treated in a manner similar to Case #1. The defect consisted of 2–3 walls, extended 7 mm intraosseously and involved the mesial furcation (class II).

Case #3

In March 1979, a 15-year-old black male had an evaluation. Medical history was noncontributory to his oral condition. Previous dental treatment had consisted

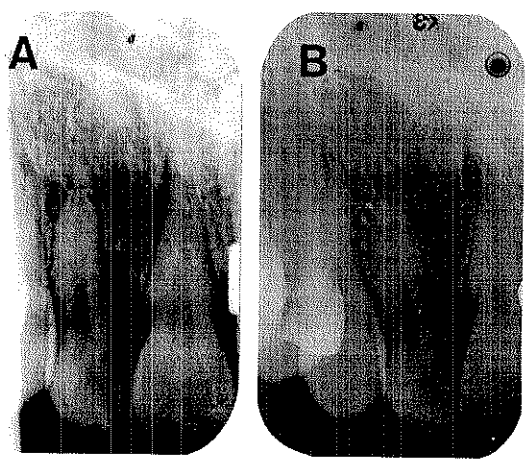

Figure 4. A. *Case #1, radiograph of maxillary right lateral incisor with mesial vertical defect, November 1976.* **B.** *Case #1, radiograph demonstrating osseous repair of maxillary right lateral incisor, May 1980.*

Table 2
Mobility Values

Case	Tooth No.*	Mobility[28]	
		Preoperative	Postoperative
#1	3	1.5	0
	7	1	0
	14	2.5	
	19	2.5	0
	30	1.5	0
#2	3	1	0
#3	3	1.5	0
	8	0.5	0
	14	0.5	0
	19	1	0
	30	1	0
#4	3	1.5	0
	14	1.5	0

* Teeth No. 1–32 (universal numbering system).

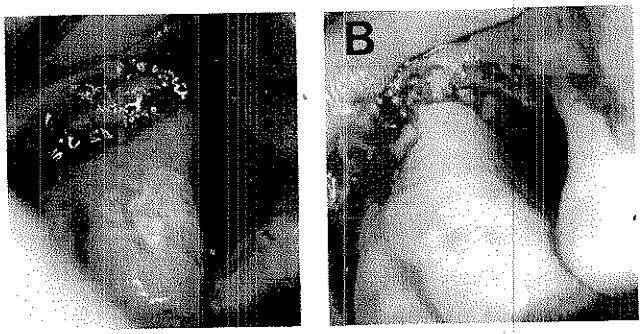

Figure 5. A. *Case #1, osseous defect on mesial of maxillary right first molar, December 1976.* **B.** *Case #1, maxillary right first molar reentry with osseous fill, December 1977.*

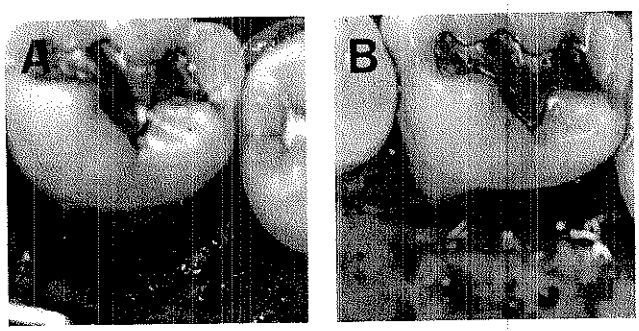

Figure 6. A. *Case #1, osseous defect on distal and lingual of mandibular right first molar, December 1976.* **B.** *Case #1, mandibular right first molar reentry, revealing osseous fill, December, 1977.*

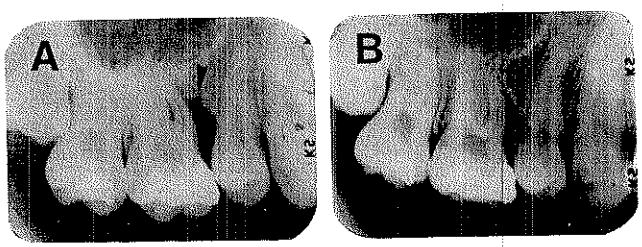

Figure 7. A. *Case #2, maxillary right first molar with vertical defect, April 1981.* **B.** *Case #2, maxillary right first molar with osseous repair, July 1982.*

of routine operative care. The gingiva was pink, with minimal plaque and calculus deposits on the lingual of the mandibular anterior teeth. Ten-mm pockets were found on the mesial of the maxillary and mandibular right first molars (Table 1). In addition, a 7-mm pocket was found on the mesial of the maxillary left first molar and on the distal of the maxillary right central (Table 1). Deep pockets were associated with radiographic osseous defects (Fig. 8, A, C, E and G) and hypermobility (Table 2). Initial therapy was the same as in Case #1. Surgical debridement revealed 6 to 8 mm deep, 2–3-walled intraosseous defects and mesial furcation involvement (class II) of the maxillary first molars. The patient's plaque control postsurgically was poor and there was concern that this would decrease the potential of osseous repair in the lesions.

Case #4

In May 1981, a 17-year-old black male was referred for evaluation by his orthodontist, who noticed on radiographs alveolar bone destruction on the mesial of the maxillary first molars (Fig. 9, A and C). Medical and dental history, which included orthodontic care, was unremarkable. Some plaque and calculus were present. Pocket depths of 8 and 10 mm (Table 1) corresponded with radiographic defects on the mesial of the right and left maxillary first molars, respectively. Treatment was as previously described. Both maxillary molars exhibited class II mesial furcation involvement. Intrabony defects on the mesial of both teeth were 6 to 8 mm deep and consisted of 2–3 walled combinations.

RESULTS

Clinical Findings

The oral hygiene performed by Cases #1 and #2 was exemplary, whereas Cases #3 and #4 showed a lack of

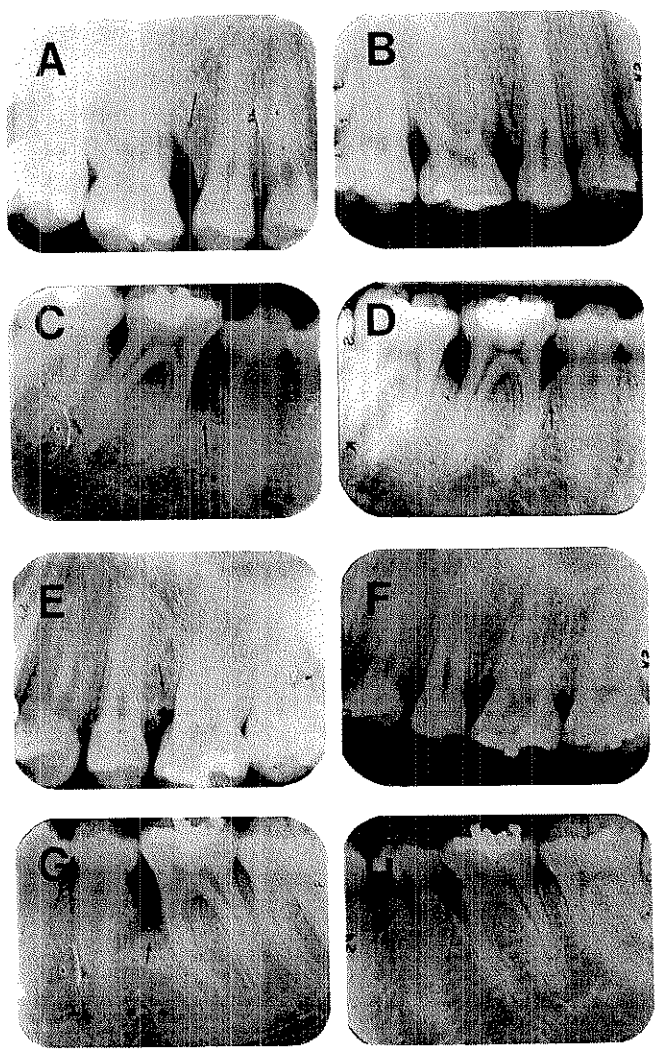

Figure 8. A,C,E,G. *Case #3, radiographs with associated vertical defects, January 1979.* **B,D,F,H.** *Case #3, radiographs with resulting bone fill, February 1983.*

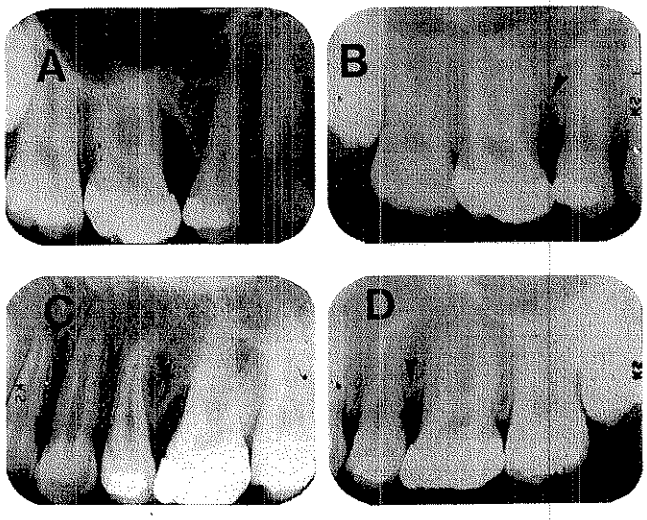

Figure 9. A,C. *Case #4, radiographs of maxillary molars with vertical defects, May 1981.* **B,D.** *Case #4, radiographs after treatment revealing osseous fill, February 1983.*

interest in proper brushing and flossing, resulting in some clinical inflammation. Pocket depths associated with osseous defects have been reduced and remained 4 mm or less (Table 1). Mobility of teeth associated with severe vertical osseous defects diminished after therapy (Table 2).

Osseous Repair

In all four patients, granulation tissue was apparent in the defects one week after surgery. Periodic radiographs indicated increasing radiodensity and osseous repair in all treated areas (Figs. 3, 4, 7, 8 and 9). In Case #1, where reentry was performed, confirmation of bone repair was seen. On the mesial and buccal of the maxillary right first molar, substantial bone apposition resulted in closure of the furcations (Figs. 3B and 5B). The defect on the mesial of the maxillary right lateral incisor demonstrated osseous repair (Fig. 4B). After 1 year, the bilateral moat-like defects on the distal of the mandibular first molars, as well as the lingual furcations, filled with bone (Figs. 3, D, F and 6B).

In Case #2, at 2 years postoperatively, radiographic evidence of osseous repair was noted on the mesial of the maxillary right first molar (Fig. 7B).

Case #3, over a 4-year period, has demonstrated increasing radiographic repair on the mesial of the mandibular and maxillary first molars (Fig. 8, B, D, F and H). Bone-fill was also seen on the distal of the maxillary right central incisor. In February 1983, an osseous defect was seen on the distal of the mandibular right first molar, a previously uninvolved site.

At 22 months, Case #4 has shown repair on the mesial of both maxillary first molars (Fig. 9, B and D).

DISCUSSION

The authors considered publishing only one case to illustrate the results of therapy. However, to provide documentation that successful results could be predictably obtained, four cases have been presented which include 12 osseous defects with preoperative and postoperative clinical measurements and radiographs. Each outlined case demonstrated several classical features of juvenile periodontitis as described by Baer[6]: distinctive radiographic pattern of alveolar bone loss, familial pattern, early age of onset, apparent lack of relationship between local etiologic factors and presence of deep periodontal pockets and rapid rate of progressive breakdown.

Individuals underwent intensive oral hygiene instruction to try to ensure their cooperation. They were taught brushing and flossing; however, only Cases #1 and #2 consistently performed good oral hygiene. Despite the presence of supragingival plaque and some marginal gingivitis in Cases #3 and #4, all four patients demonstrated osseous repair in all defects. This may be accounted for by the surgery and the extended tetracycline therapy. The rationale for antibiotic coverage was based upon the concept that JP is an infection of bacterial etiology and its use has appeared to enhance osseous repair in JP patients.[30] Tetracycline was also considered the drug of choice since it has been shown to be effective against *AA*[31] and achieves a gingival fluid level 2 to 10 times greater than blood serum.[32,33] The initial dosage (250 mg q.i.d.) and duration of administration were based upon a previous report which used antibiotics as an adjunct to therapy.[34]

The flaps were positioned at the alveolar crest leaving osseous defects denuded, as described by Prichard.[35] Application of Prichard's technique for intrabony defects to 2–3-walled and hemi-circumferential defects achieved satisfactory results. It permitted granulation tissue to form in the defect and theoretically prevented the apical downgrowth of epithelium along the root surface, which results in a long junctional epithelium instead of a connective tissue attachment.[35]

Published reports differ regarding the advantages of bone grafts as compared to "open clean out" procedures. Many investigators have claimed better results after using a graft material,[36–38] whereas others have reported success without grafts if optimal plaque control is practiced.[39–41] Recently, successful results in JP patients have been reported after using freeze-dried bone allografts[27] and autogenous bone grafts.[25,26] The patients presented here received no graft material. The clinical and radiographic results indicate that osseous defects associated with JP have the potential to repair without grafts. On Case #1, confirmation of osseous repair was obtained during reentry procedures performed to complete pocket elimination. It was observed that 2–3-wall, 3-wall and hemi-circumferential defects demonstrated osseous fill. Elimination of the defects was the result of osseous repair and probably some crestal bone resorption.[39–41] In addition, it should be noted that perforation of the alveolar plate lining defects

was not performed. This procedure has often been mentioned by authors;[27,37-39,42] however, there is no evidence that this has enhanced repair.

Simultaneous therapy of both inflammation and hypermobility was avoided since the current literature has indicated that it may be unnecessary.[39-41,43] Early investigators assumed that occlusion played a significant role in the initiation, progression and management of juvenile periodontitis.[2,20] However, clinical experimentation has definitively shown that occlusal trauma does not initiate loss of attachment or cause pocket formation.[21-23] Initial studies attempting to verify the codestructive theory failed to demonstrate that occlusal trauma in the presence of inflammation resulted in accelerated attachment loss.[45,45] More recently, in animal models, there is evidence that both supports[46] and refutes[47] this concept. Two other human studies of the relationship between occlusion and angular defects indicated that periodontal defects correlated with the location of plaque rather than exerting a codestructive effect.[48,49]

The results obtained in the presented cases support Polson and Heijl's[43] contention that simultaneous management of inflammation and hypermobility may not be required. The patients were treated without occlusal therapy and subsequent to resolution of inflammation, osseous repair occurred and hypermobility decreased. Contrary to Evian et al.,[26] who had concluded that successful treatment of JP cases required combined inflammatory and occlusal therapy, the results of these cases demonstrates that successful treatment of JP can be achieved by control of infection and inflammation.

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