Treatment of Localized Juvenile Periodontitis (Periodontosis)

A Review

David B. Krill* and Hiram R. Fry†

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LOCALIZED JUVENILE PERIODONTITIS IS A DISEASE of the adolescent periodontium characterized by rapid alveolar destruction around molar and incisor teeth of the permanent dentition. Early treatment methods were variable and often empirical due to lack of knowledge concerning etiology. Elucidation of factors associated with the disease has led to different therapeutic approaches. A comprehensive review of these modalities is presented.

The concept of periodontosis as a distinct clinical entity was introduced by Gottlieb[1] in 1923 in a report of a fatal case of influenza. He noted that the collagenous periodontal ligament fibers had lost their orientation and had become loose connective tissue. He also observed extensive resorption of the root apices and a widened periodontal ligament space, which he termed "diffuse atrophy." The degenerative histologic appearance led Orban and Weinmann[2] to describe the condition in three stages: (1) degeneration of the periodontal ligament, (2) apical epithelial proliferation, and (3) pocket formation. However, Wannemacher[3] had earlier questioned the degenerative nature of the disease, pointing out that an inflammatory process was dominant. He noted that even in the presence of deep pockets, the gingiva *appeared* healthy, but bleeding was produced upon probing with a blunt instrument. Bernier[4] and Ramfjord[5] also dissented from the degenerative theory with the observation that inflammation was always present at the histologic level.

The molar-incisor pattern of bone loss was first observed by Wannemacher in 1938.[3] Tenenbaum et al.[6] stated that the disease was found primarily in adolescent females and displayed a distinctive radiographic pattern. Radiographic examination revealed arc-shaped bone loss in the molar region, starting from the mesial surface of the second molar and extending to the distal surface of the second bicuspid. Bone loss was also noted in the incisor areas, with the mandibular bicuspid area showing the greatest resistance to the disease. This condition was further clarified by Baer[7] in an article that forms the basis for clinical diagnosis of what is currently recognized as a distinct disease entity. He defined periodontosis as a disease of the periodontium occurring in an otherwise healthy adolescent which is characterized by a rapid loss of alveolar bone about one or more teeth in the permanent dentition, with minimal accumulation of plaque, and little or no clinical inflammation. He described two forms of the disease: a localized form in which only permanent first molars and incisors are affected, and a more generalized form. Other criteria described by Baer were: (1) age of onset 11 to 13 years—terminating at age 20, (2) vertical bone loss in the molar regions with characteristic "arc-like" defects and horizontal bone loss in the incisiors, (3) bilaterally symmetrical patterns of bone loss in the molar regions, (4) rapid progression of the disease, (5) predilection for females, (6) tendency to occur in familial patterns, and (7) lack of involvement of the primary dentition.

No uniform terminology for the condition has been accepted. In the recent literature the most frequently used term for the molar-incisor form has been localized juvenile periodontitis (LJP).[8] There has been less consensus concerning nomenclature of the generalized form. In 1977 Socransky[9] described the microbiota associated with a "rapidly destructive periodontitis" as separate from that seen in "periodontosis." More recently Page et al.[10] have described a form of "rapidly progressive periodontitis" as a distinct clinical condition. How these conditions may relate to the generalized form of periodontosis described by Baer[11] is unclear. In this review the term LJP will be used for the localized

* Formerly a Resident in Periodontics, University of Tennessee at Memphis, Memphis, TN 38163. Currently in private practice in Akron, OH.

† Professor and Director of Postgraduate Periodontics, University of Tennessee at Memphis.

form, except when "periodontitis" is quoted directly from the literature.

A series of studies by Newman et al.[12–14] and Newman and Socransky[15] established a credible microbial component for the etiology of LJP. They isolated "five periodontosis groups" which are largely Gram-negative anaerobic rods (78% of the total cultivable flora), the majority of which could not be identified by conventional methods of classification. The periodontopathic potential of these organisms has been demonstrated in germ-free rats.[16] Of interest relative to treatment was the fact that all groups were found to be susceptible to tetracycline.[15]

Considerable progress has been made in identifying and characterizing the microorganisms in LJP.[17–19] Numerically important organisms include various Gram-negative facultative rods, mainly *Actinobacillus actinomycetemcomitans* (A.a.) and *Capnocytophaga.* Barbanell et al.[20] demonstrated cytotoxic activity of endotoxin from LJP micoroorganisms.

Differences in lymphocytes,[21,22] polymorphonuclear leukocytes (PMNs),[23,24] structure of immunoglobulins,[25] immunoglobulin levels,[21] and complement activation[26] have been reported between patients with LJP and healthy controls, or between LJP patients and those with other forms of destructive periodontal disease. More recently, Genco et al.[27] and Ranney et al.[28] have reported precipitating antibodies to bacteria strongly associated with LJP, but not from patients with other forms of periodontal disease.

There have been few direct genetic analyses regarding LJP. Fourel[29] has classified those studies into two groups: (1) familial patterns and (2) consanguinity determinations. Newman[30] suggested that familial patterns may result from: (1) genetic predispositions to specific groups of bacteria, (2) a genetically determined immunodeficiency, and (3) faulty or impaired formation and maintenance of periodontal tissue integrity. (For a more complete review see Saxen, 1980.[31])

Various therapeutic approaches to LJP have been described, usually in the form of isolated case reports. Few controlled studies have been carried out, and no comprehensive review of treatment modalities has been published. This article offers such a review.

REVIEW OF TREATMENT

Early treatment methods

The clinical management of LJP has not received the same intensity of interest as its etiology and pathogenesis. Robinson[32] in 1951 stated that local therapy is of little avail in "periodontosis," and until the last decade little had been published on treatment approaches. This tended to reinforce the opinion that extraction of the involved teeth was the most practical approach to treatment. Baer and Everett,[33] however, in 1970 opposed early extraction of maxillary first molars. They noted that the maxillary sinus enlarges by pneumatization from the 3rd month of fetal life until age 18, thereafter becoming more stable. Likewise, he observed that accelerated pneumatization of the maxillary sinus into the resultant edentulous ridge occurs following extraction, thereby complicating any future periodontal therapy. This led him to the opinion that these teeth should be retained as long as possible.

One of the first studies involving treatment was performed by Tenenbaum et al.[34] in 1957. Patients with LJP were divided into five groups. Group I received "local dental treatment" consisting of oral hygiene instructions, root and soft tissue curettage, selective tooth grinding, and periodontal surgery over a period of 13 months. During the last 7 months, the patients received daily doses of vitamins A, B complex, C, D, and dibasic calcium phosphate. Group II received the same local dental treatment but without the vitamin-calcium-phosphate therapy. Group III received only vitamin-calcium-phosphate therapy. Group IV consisted of female patients who were given daily 50-mg doses of the hormone methylandrostenediol. (The rationale for this hormone therapy was the positive response seen when similar hormones were used in treating postmenopausal and senile osteoporosis.) Group V consisted of selected patients from the first and second groups who had completed their "local dental treatment." Subsequently, splints were constructed of orthodontic bands and wires for temporary stabilization of the involved teeth.

Evaluation of the five groups over a period of 3 years showed that local dental treatment with or without the vitamin-calcium-phosphate therapy improved the clinical appearance of the tissues and reduced pocket depth. Comparable radiographic improvement did not occur, however. Group III (vitamin-calcium-phosphate therapy alone) and Group IV (hormone therapy) showed no appreciable clinical or radiographic improvement. Group V ("local dental treatment" with or without nutritional therapy, combined with splinting) was the only group to show radiographic improvement. This consisted of a small increase in the per cent of radiographic alveolar bone fill, plus an increase in the number of regions showing a "lamina dura" at the crest. Due to this response, the authors undertook permanent splinting in some of the patients. In a follow-up study[35] three of the patients were recalled 17 to 20 years after the permanent splints had been inserted. The patients had not received regular dental care and the gingival tissues were in poor condition, but no teeth had been lost due to periodontal disease, leading the authors to speculate as to the value of long-term stabilization in advanced periodontal conditions.

Another early method of treatment was proposed by Everett and Baer[36] in 1964. They reasoned that if the affected teeth were taken completely out of occlusion through repeated occlusal or incisal grinding, progressive tooth eruption would occur. In this process of

eruption, the involved cemental surface, as well as the apically located healthy cementum and adjacent bone, would move occlusally, thereby reducing the depth of the defect. The authors described the steps of the procedure as follows: (1) The involved tooth is taken completely out of occlusion. Molars are narrowed buccal lingually as they are shortened. (2) Periodontal therapy is performed consisting of a flap operation, removal of soft tissue from the osseous defect, and root planing. (3) Repeated occlusal grinding and root planing are carried out until a satisfactory bone contour is achieved. Three cases were presented to illustrate encouraging results. Unfortunately, no distinction was made between the amount of osseous repair attained by the surgical therapy and the amount resulting from the occlusal relief and subsequent eruption. Stein[37] has also reported success using this technique.

Comment. A critical review of these early reports reveals that successful and sometimes impressive results were obtained. Most, however, did not compare the results of one form of treatment against another, nor did the results lend themselves to statistical analysis. Greenstein et al.[38] recently found no relationship between the presence of a crestal lamina dura and other clinical parameters of health or disease. This casts doubt upon interpreting the value of a particular treatment modality based on the radiographic appearance of the crestal bone.

Effectiveness of Antibiotics

In their study of the predominant cultivable microbiota in LJP, Newman and Socransky[15] reported finding five groups of organisms, all of which showed some susceptibility to tetracycline, *in vitro*. Strains of the organisms from Groups III and IV were later characterized as strains of *Actinobacillus actinomycetemcomitans* (A.a.),[17] an organism which has been widely implicated in the etiology of LJP. Subsequent studies have confirmed the effectiveness of tetracycline[39,40] and minocycline[39] against A.a. *in vitro*. Gordon et al.[41] have shown that with standard oral doses of tetracycline, 250 mg taken 4 times daily, concentrations found in gingival crevicular fluid are two to four times greater than in serum. For these reasons tetracycline has been incorporated as an adjunct to many other forms of therapy, as will be reported in this review.

Spiromycin, a macrolide antibiotic, has been investigated in the treatment of periodontal disease due to its secretion in the saliva and crevicular fluid and retention in osseous tissue for long periods.[42] Harvey et al.[43] treated a group of LJP patients with 500 mg of Spiromycin 3 times a day for 5 days, followed by 500 mg 2 times a day for 5 more days. For most patients, antibiotic administration was the only treatment performed. Clinical and radiographic examinations were carried out, and subgingival plaque samples were examined by fluorescent and dark-field microscopy. Harvey reported improvement clinically, radiographically, and microbiologically (reduction of *B. melanogenicus* and spirochetes).

In a group of LJP patients, Phenoxymethyl penicillin was administered orally in conjunction with flap surgery.[44] Controls received a placebo in conjunction with surgery. In both groups plaque, gingival inflammation, gingival bleeding, and pocket depths were decreased, while attachment levels and radiographic bone height increased. No differences were present between the two groups, indicating that treatment results were not enhanced by the oral administration of penicillin.

Metronidazole has been shown to be effective against many organisms associated with destructive periodontitis. However, it has shown little activity against A.a. *in vitro* and has not been recommended in the treatment of LJP.[45]

Comment. In clinical trials penicillin has been ineffective as an adjunct to the treatment of LJP, and metronidazole has been ineffective against A.a. *in vitro*. Insufficient information exists to properly evaluate other antibiotics, such as Spiromycin. Therefore, in light of current information, tetracycline and minocycline appear to be the antibiotics of choice in the treatment of LJP.

Nonsurgical Therapy

Although most current studies and case reports on LJP involve surgical therapy, several investigations have found nonsurgical therapy to be successful. Gold[46] reported clinical and radiographic improvement in a 14-year-old LJP patient treated by a 6-week regimen of biweekly scaling and root planing under local anesthesia combined with tetracycline administration (250 mg 4 times a day for 1 week, then 250 mg once a day during the 6 weeks of subgingival therapy). A subsequent report by Genco et al.[47] in which they achieved similar results lends support to this modality of therapy.

Slots and Rosling[48] studied the effectiveness of subgingival debridement, topical Betadine solution and systemic tetracycline in suppressing subgingival A.a., and other microflora in 20 deep periodontal pockets and 10 normal sites in six LJP patients. Treatment was performed in three stages over a period of 22 weeks. Stage one consisted of plaque control instructions and at least 6 hours of scaling and root planing. Stage two involved the placement of Betadine-saturated gauze into the pockets for 10 minutes. Stage three involved the use of systemic tetracycline, 1 gm per day for 14 days. The composition of the subgingival microflora was determined at frequent intervals by direct microscopic examination for spirochetes and selective culturing for A.a. and *Capnocytophaga*. Clinical changes were determined by measuring pocket depth, attachment level, gingival inflammation, presence or absence of suppuration, and radiographic alveolar bone mass.

The microbiologic results showed that scaling and

root planing reduced the number of spirochetes, A.a., and *Capnocytophaga*, but the organisms were not completely eliminated. Betadine had little effect on the subgingival microflora. Systemic tetracycline, on the other hand, suppressed spirochetes, A.a., and *Capnocytophaga* to almost undetectable levels. Clinically, scaling and root planing reduced plaque and gingival inflammation and also resulted in a small reduction in pocket depth, but did not arrest the loss of periodontal attachment in all areas. Following tetracycline therapy, an average attachment gain of 0.3 mm occurred. Four sites lost 1 to 2 mm of attachment. In these sites high numbers of A.a. had returned. The authors concluded that A.a. is an important etiologic agent in LJP and that A.a. infections cannot be resolved by root surface debridement alone. The combination of root surface debridement and systemic tetracycline was successful in most, but not all sites treated.

Local chemotherapy in the form of sodium hypochlorite solution was investigated by Adcock et al.[49] Twenty-two diseased sites from patients with LJP were sampled by dark-field microscopy and anaerobic plating, then treated with "chemical curettage." This involved administration of sodium hypochlorite, pH 13.95, into the gingival crevice for 90 seconds. The hypochlorite was then neutralized with 5% citric acid, and the sulcus was instrumented with five to six strokes of the curette. After the curettage, the sampling was repeated and continued over a 90-day observation period. The chemical curettage resulted in elimination of spirochetes and fusiforms immediately after treatment. At the end of the 90-day observation period, the spirochetes had returned, but in lower numbers than were originally present. Fusiforms, however, showed an increase over the original numbers. Results obtained from anaerobic plating were not statistically significant.

Surgical Therapy

Surgical therapy for LJP has been proposed by many authors. Gjermo[50] stated that it is often indicated to provide access to deep subgingival plaque. This was also proposed by Waerhaug,[51] who reported success in the treatment of 21 patients with LJP. Treatment was aimed at the removal of all supra- and subgingival plaque. He stated that in areas where pocket depth exceeded 5 mm, total plaque removal could not be achieved by scaling alone and therefore that surgical therapy for "pocket elimination" was indicated. Directly comparing the effectiveness of nonsurgical and surgical therapy in eight LJP patients, Kornman and Robertson[52] followed a three-stage clinical protocol: (1) scaling and root planing, (2) scaling and root planing with concurrent systemic tetracycline (1 gm daily for 28 days), and (3) periodontal surgery with the same regimen of systemic tetracycline. The decision to progress to the next treatment stage or to place the patient on a 3-month maintenance recall program was based on clinical findings of suppuration, bleeding upon probing, and probing depth. Scaling and root planing had virtually no effect, and all patients progressed to Stage 2. Five of eight patients also required surgery (Stage 3). Examination of the associated microflora revealed that scaling and root planing plus tetracycline (Stage 2) were effective in sites that were high in either A.a. or black pigmented *Bacteroides* (BPB). However, in sites high in both A.a. and BPB, surgery was required.

Lindhe and Liljenberg[53] reported on 16 cases of LJP treated surgically and followed for 5 years. Examination performed before and after treatment included oral hygiene status, gingival conditions (bleeding with probing), probing depth, and attachment levels. Treatment consisted of a 2-week dosage of tetracycline (250 mg 4 times a day) and surgical therapy performed 1 to 2 days after the tetracycline therapy was initiated. The surgery consisted of a Modified Widman Flap with curettage of the bony defects and root surfaces. Patients were recalled every 4 weeks during the first 6 months postoperatively and every 3 months thereafter. Treatment resulted in up to a 95% decreased incidence of bleeding with probing, decreased probing depth, and gain of clinical attachment. Although not standardized, the radiographs obtained showed evidence suggestive of considerable bone fill.

Baer and Socransky,[54] Hoge and Kirkham,[55] Van Swol,[56] Barnett and Baker,[57] and Jaffin, et al.[58] have also reported success with open (surgical) curettage and replaced flaps coupled with the use of systemic antibiotics.

Popper[59] reported a sequence of treatment that produced encouraging results without the use of adjunctive antibiotics. Initial preparation consisted of scaling, root planing, and curettage under local anesthesia plus any needed occlusal adjustment. This was followed by surgical therapy consisting of full thickness replaced flaps, debridement, and decortication of the osseous defects and vigorous root planing. Ashrafi et al.[60] have also reported success in two cases treated by open debridement without adjunctive antibiotics.

When bone grafts have been used in conjunction with surgical therapy, results have been encouraging. Kaslick et al.[61] and Oshrain and Kaslick[62] placed autogenous bone chips in osseous defects as a method of treating LJP lesions. They combined this therapy with relief of the occlusal surfaces of the involved teeth as described by Baer before and during surgery, as well as 1 month later.[36] Burnette and Stewart[63] described a case in which osseous coagulum from an extraction site was used with success in a LJP lesion. Interestingly, they also performed a bone swedging procedure from an edentulous ridge to the mesial aspect of a maxillary molar. This was also reported to be successful. Evian et al.[64] reported a case of LJP in which the lesions were associated with occlusal trauma. Therapy was directed against both inflammatory and occlusal components of

the disease. Following oral hygiene instructions and scaling and root planing, a maxillary bite plane was constructed. Periodic subgingival scaling and adjustment of occlusion were then undertaken. This therapy resulted in clinical improvement as seen by reduced pocket depth, tooth mobility, and improved gingival tone. Radiographic examination showed evidence suggestive of osseous fill. Surgical therapy was then performed, consisting of internal beveled incision, full thickness flaps, curettage of the osseous defects, root planing, and osteoplasty. Bone removed by osteoplasty was placed in the osseous defects. The flaps were apically positioned and the osseous grafts were covered by free soft tissue autografts as described by Ellegaard et al.[65] This resulted in further reduction of pocket depth and radiographic suggestion of further improvement in osseous topography.

DeMarco and Scaletta[66] used frozen autogenous hip marrow obtained by a cut-down procedure in molar/incisor defects in a 15-year-old female. Six weeks postsurgically, pocket depths were reduced from 8 to 12 mm to approximately 4 to 5 mm in all areas. Mattout and Roche[67] reported a case of an LJP lesion treated by frozen autogenous iliac marrow after surgical curettage had been unsuccessful. The iliac marrow was implanted around a mandibular first molar which had severe defects on the buccal and mesial aspects and in the furcation area. The surgical area was re-entered 1 year postoperatively. Surprisingly, bone filled the furcation, but only partially filled the defect on the mesial aspect.

Sugarman and Sugarman[68] presented four cases treated by either osseous coagulum or frozen autogenous hip marrow. Success was determined by decreased pocket depth, no bleeding with probing, and increased density of bone radiographically. In one of these patients, BoPlant (a product of boiled bovine osseous tissue) was also used as a grafting material in an angular defect on the mesial aspect of a mandibular first molar. This attempt failed and the implant sequestered 5 months postoperatively. The area was re-entered, thoroughly curetted and sutured, and subsequently healed uneventfully. The authors concluded that successful treatment may be obtained through a variety of therapeutic approaches.

Yukna and Sepe,[69] in a clinical study, evaluated the use of freeze-dried bone allografts combined with local and systemic tetracycline. Twelve cases were reported. After initial therapy consisting of systemic tetracycline, oral hygiene instructions, scaling and root planing, and occlusal adjustment, surgery was performed. Flaps were elevated, osseous defects debrided, and root planing and intramarrow penetration performed. The graft material composed of a 4:1 mixture of freeze-dried bone, and tetracycline powder was then implanted. Furcation defects equal to or greater than 3 mm in depth, as well as interproximal defects, were implanted. Patients were recalled every 10 days for 1 month, then every 3 months. Fifty-one of 62 defects were re-entered 1 year postoperatively. In 98% of the defects, bone fill equal to or greater than 50% was achieved. The average fill was 80%. Twenty-two of 23 furcas gained closure of 50% or more.

In a more recent article Mabry et al.[70] compared 16 LJP patients placed in one of two treatment groups. Group 1 received systemic tetracycline therapy consisting of two 10-day regimens (250 mg QID) 60 and 30 days before surgical treatment, followed by a similar 10-day regimen immediately postsurgically. One half of the defects in Group 1 were treated by open debridement and by freeze-dried bone allografts/tetracycline powder in a 4:1 mixture (Group 1a). The remaining defects (Group 1b) received open debridement alone. Group 2 received no systemic antibiotics. One half of the defects in Group 2 received open debridement plus freeze-dried bone allografts without tetracycline powder (Group 2a); the remaining defects received open debridement alone (Group 2b).

The best results in terms of bone fill and per cent resolution of the osseous defects were obtained with systemic tetracycline and the freeze-dried bone allograft/tetracycline implant (Group 1a). This treatment regimen was also most effective in gaining fill in furcation defects. Both implanted groups demonstrated superiority over open debridement in mean defect fill and per cent resolution of the osseous defect, along with greater success in furcations.

Comment. A few authors have reported clinical success with nonsurgical therapy in conjunction with systemic tetracycline. Others, using clinical and microbiologic parameters, report only partial success or continued progression of the disease. Many reports of clinical success have been made in conjunction with surgical procedures. Most have employed systemic tetracycline as adjunctive therapy. Where both nonsurgical and surgical methods were employed, surgery was considered necessary to achieve control of clinical and microbiologic parameters in the more involved sites. Results of surgical therapy with and without various forms of osseous implants appear to be equally impressive, particularly in terms of radiographic bone fill. Only one study has directly compared the two methods by way of re-entry. The results indicate that at least one type of implant (freeze-dried bone with or without tetracycline powder mixture) produced greater bone fill than surgical debridement alone. Little evidence has been presented regarding the long-term stability of these results, and evidence regarding the type of attachment obtained is lacking. Concerns regarding possible sensitization of patients to tetracycline in conjunction with topical administration have been raised by the Ad Hoc Committee on Pharmacotherapeutics (AAP Newsletter 15(1), February, 1980). Recent events have also caused concern regarding the possible transmission of viral-

related diseases through the use of allogenic materials, including freeze-dried bone implants.

Tooth Transplantation

In cases where bone loss is severe and first molar teeth are considered hopeless and must be extracted, transplantation of unerupted third molars into the site occupied by the first molars has been performed. This was first discussed by Baer and Gamble,[71] who listed a set of conditions to be met before a high rate of success could be obtained: (1) the third molar should have roots developed to a point of bifurcation equal to 2 to 3 mm, but should not have completed more than 90% of its root development; (2) the mesiodistal width of the unerupted third molar must be no larger than the first molar it is to replace; and (3) the transplanted third molar should be placed in infraclusion and kept out of occlusal contact for at least 3 to 4 weeks following its insertion into the first molar site. The authors reported four cases treated by the above criteria. Two showed success; a third became nonvital after 1 year, but responded well to endodontic therapy. The fourth case showed some evidence of root resorption, but the bony defect had repaired. Citing Hoffman's[72] findings that a developing hamster molar transplanted subcutaneously continues to develop and form bone, periodontal ligament and cementum, Baer and Gamble[71] hypothesized that the transplantation might induce new periodontal tissues to form as the roots continued to develop and the tooth erupted.

In a more recent study, Borring-Moller and Frandsen[73] reported 15 cases of autologous third molar transplantations to sites where the first molars had been lost to LJP. The cases were followed for 7 years. The authors stated that no attempt was made to remove any pocket epithelium or granulation tissue present in the socket. When necessary, burs and ronguers were used to shape the alveolus of the first molar to fit the third molar. If the mesiodistal width of the third molar was greater than the width of the recipient site, approximal surfaces of the teeth adjacent to the site were ground, polished, and painted with 2% sodium fluoride. In six cases, stabilization of the transplanted teeth was obtained with a suture from the buccal gingiva across the occlusal surface to the lingual. This suture was left in place for 1 week. In the remaining nine cases, less tissue support was present and the transplanted teeth were stabilized with 0.16 mm wire for 6 weeks. At the end of the 7-year observation period, no teeth had pocket depths greater than 3 mm, no attachment loss was noted, and there was radiographic evidence of repair of the osseous defects.

Similar clinical and radiographic success in an isolated case was reported by Arlin and Freeman.[74] They stated that even if a transplanted tooth was eventually lost, the transplant would provide a young patient with a temporary replacement until prosthetic replacement was feasible.

Comment. Tooth transplantation studies report considerable success in achieving radiographic osseous repair with minimal sulcus depth. However, examination of some of the post-treatment radiographs raises questions concerning possible altered or impaired root development of the transplanted teeth. With advances in alternative forms of therapy, it would seem prudent to reserve this type of treatment for carefully selected cases.

Orthodontics

In another treatment approach, when hopeless first molars must be extracted, orthodontics may be used to move the second and third molars into the position of the first and second molars, respectively. Goldstein and Fritz[75] advocated delaying orthodontic treatment for 3 months after extraction, as they found that when teeth were moved earlier than this into an area affected with LJP, they may become similarly afflicted. Compton et al.[76] stated that, in addition, adequate time must be allowed for proper healing of the extraction site to ensure the maintenance of proper attachment and support during tooth movement. In their reported case, orthodontic treatment was delayed for 1 ½ years after extraction.

Adjunctive Endodontics

In extensive lesions, as with adult periodontitis, pulpal tissues may become affected. Prince and Lilly[77] reported on a 16-year-old (LJP) patient with such severe bone loss that the authors treated the teeth endodontically before initiating periodontal therapy. The pulpal tissues, which did not exhibit hemorrhage upon access preparation and extirpation, were submitted for histologic examination. Both fibrosis and diffuse calcification were seen. Although these findings may be present in clinically healthy pulpal tissue, they are uncommon in a patient of this age. After endodontic therapy and prior to periodontal therapy, pocket depths on the affected teeth decreased 2 to 3 mm. The authors stressed that this case does *not* justify performing endodontic therapy on all teeth affected by LJP. It does, however, illustrate that in advanced lesions an endodontic as well as periodontic component may be present and as in adult periodontitis, some resolution of the lesion may be anticipated after endodontic therapy.

In cases where severe bone loss affects only one root of a multirooted tooth, endodontics, tooth resection, and restoration with a fixed partial denture have been proposed as an alternative to extraction.[78]

Comment. Consistent success in treating LJP appears to be achieved using traditional initial therapy (scaling, root planing, oral hygiene instruction, and occlusal adjustment), followed by surgical therapy in the form of open curettage. Augmentation of the open curettage by various methods of osseous grafting also appears to be successful, although such problems as expense, availability, and safety of the donor material must be con-

sidered. Based on the limited data available, osseous grafting combined with systemic and/or local tetracycline therapy offers promise for further investigation. The combination of effectiveness against LJP-associated bacteria and the concentration in gingival crevicular fluid have made tetracycline the drug of choice in LJP therapy.

Direct comparison of open curettage with other forms of reattachment therapy such as citric acid root conditioning has not been reported; however, a pilot study has been conducted by the authors of this article.

In cases where first molar teeth are considered hopeless and are to be extracted, two options are available: (1) orthodontic repositioning of the second and third molars, and (2) transplantation of the third molars to the first molar site, provided the third molar is in the correct stage of root development.

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Send reprint requests to: Dr. Hiram R. Fry, Department of Periodontics, University of Tennessee School of Dentistry, 875 Union Ave, Memphis, TN 38163.