

# Clinical Features

## PERIODONTICS

### TREATMENT OF JUVENILE PERIODONTITIS

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Juvenile periodontitis (J.P.) has been described as a specific infection causing rapid loss of alveolar bone around one or more teeth in an otherwise healthy adolescent. Clinical features include: (a) circumpubertal onset (b) minimal plaque and calculus deposits (c) rapid localized or generalized bone loss (d) mobility (e) migration (f) often bilateral mirror image osseous defects (g) familial pattern of inheritance (h) higher incidence in black females.

Although the precise etiology and pathogenesis are as yet unknown, much has been recently learned. Juvenile periodontitis disease sites demonstrate a "unique microflora" that has been classified as five groups of gram negative anaerobic rods. Most notable is the organism "Actinobacillus actinomycetemcomitans" (or A.a.). Additionally, many of these patients exhibit defects in their immune system, specifically in lymphocyte transformation as well as P.M.N. chemotaxis and phagocytosis.<sup>1</sup> Although there are theories which attempt to explain the often localized and symmetrical pattern of bone loss and relative lack of inflammation, much is still to be learned.

Today we have evidence to suggest that treatment of J.P. should consist of a prescription of Tetracycline 250 mg. Q.I.D. for three weeks in order to eradicate the A.a. organism, in conjunction with local therapy.<sup>2</sup> The various treatment modalities may include one or more of the following: scaling and root planing, closed and/or open curettage, occlusal adjustment, active and/or passive tooth movement, osseous surgery, osseous grafting, dental autotransplantation and strategic extraction.

A treatment decision must of course be based on an accurate diagnosis and treatment plan. It is not within the scope of this article however to cover this exhaustively, but rather to demonstrate some examples of various treatment modalities performed on a single patient that has to date been followed over a seven-year period and was

initially reported in the Journal of the Canadian Dental Association in 1981.<sup>3</sup>

The initial examination and diagnosis was carried out in September 1978 and treatment was initiated shortly thereafter. Initial therapy consisted of oral hygiene instruction, scaling and root planing, and systemic Tetracycline. Although treatment of the teeth to be described (1.6, 2.6, and 4.6) was carried out concomitantly, the

treatment and follow-up of each tooth is illustrated and described separately.

**Tooth 4.6:** The osseous defect as visualized during periodontal surgery was extremely deep and it was decided to carry out an osseous grafting procedure (Fig. 1, 2). (The rationale and selection of osseous grafts is not within the scope of this article but interested readers are referred to the Thesis of the author, 1977 located at

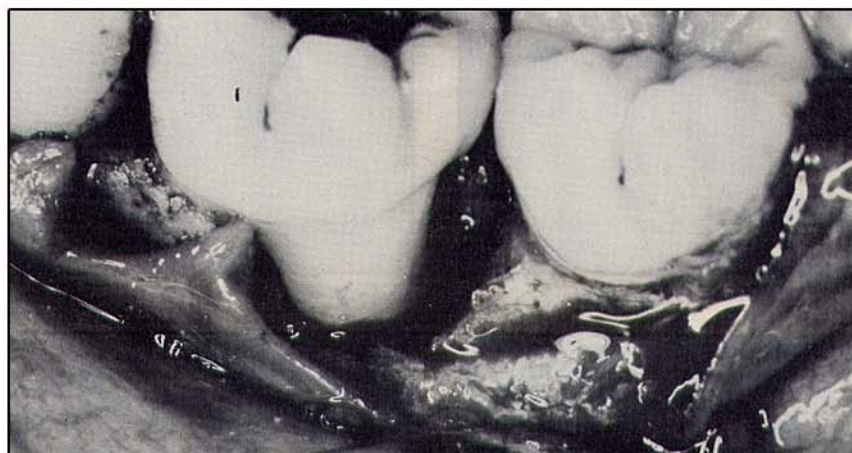


Fig. 1. Tooth #46 — deep osseous defect. Mirror image view.



Fig. 2. Tooth #46 — after placement of decalcified freeze-dried osseous allograft. Mirror image view.

the University of Toronto Dental Library.) Comparison of the initial and subsequent radiographs over a period of seven years reveal the gradual improvement over time (Fig. 3, 4). These radiographs demonstrate the initial, and post-osseous grafting healing periods at six weeks, six months, one year and seven years. It is important to note that a minimum of two years is required prior to assessment of repair when dealing with osseous grafts.

In addition to the osseous graft, passive

eruption was encouraged by periodically relieving the occlusal contacts. This was done because when a tooth erupts, the alveolus usually accompanies the root occlusally, which can have the effect of "levelling out" a crater or angular bony defect.

**Tooth 2.6:** Bone loss was so extensive that extraction was the only feasible treatment. At the same appointment, however, the extraction socket functioned as the recipient site for the autogenous transplan-

tation of the unerupted 2.8. After three weeks of nonrigid stabilization with the tooth in infraocclusion, routine maintenance therapy ensued. Examination of sequential radiographs over a seven year period revealed osseous regeneration as well as root development. Additionally, clinical post-op views at six weeks and seven years (Fig. 5) clearly demonstrate how the tooth erupted into occlusion. At present the tooth is functioning without signs or symptoms of pathology.

**Tooth 1.6:** Periodontal surgery revealed extensive bone loss that was not amenable to osseous grafting. The decision was made to attempt to maintain the tooth as a biological space maintainer over the short term. Regular recall visits and good home care were successful in maintaining the tooth over a three-year period. Due to the persistent sensitivity on the 1.6 and extreme satisfaction with the auto-transplant (that by this time had already been comfortably functioning for 18 months in place of the 2.6 position), it was decided that the 1.6 should be extracted and replaced with the auto-transplant 1.8 (Fig. 7) (Note the longer root length on the 1.8 (Fig. 7) as compared to the 2.8 (Fig. 6) which was transplanted 18 months earlier). Healing was uneventful until an endodontic fistula developed two and a half years later. Endodontic treatment failed and the tooth had to be removed (Fig. 8). Note the root development and apexification that occurred following the transplantation over a two and a half year period (compare Fig. 8 to 7). It should also be mentioned that although the 1.6 and 1.8 were lost, they did function as excellent

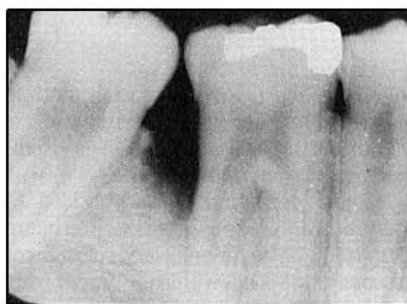
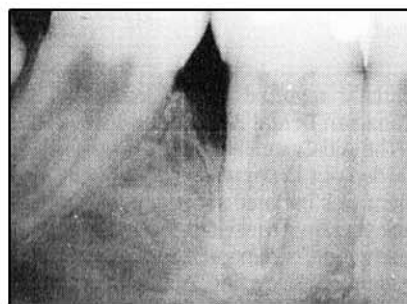


Fig. 3. Tooth #46 (i) Initial defect



(ii) 6 weeks after osseous graft



(iii) 6 months after osseous graft



(iv) 1 year after osseous graft

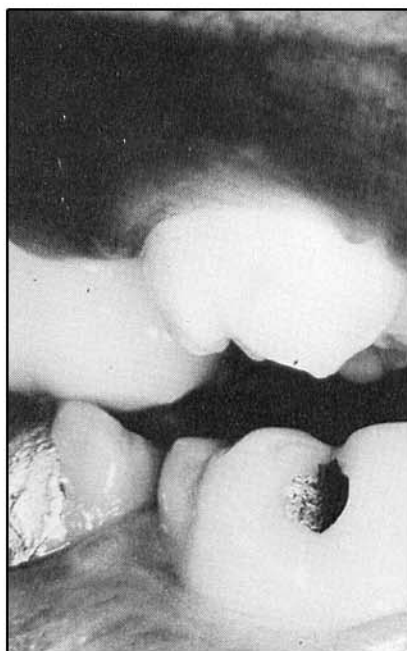


Fig. 5. Tooth #28 — 6 months after transplantation/7 years after transplantation.

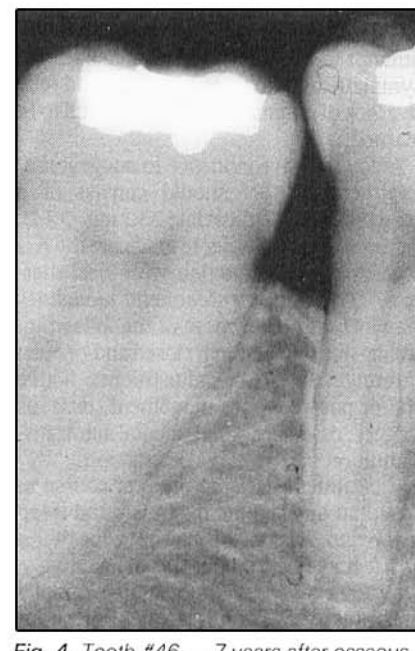
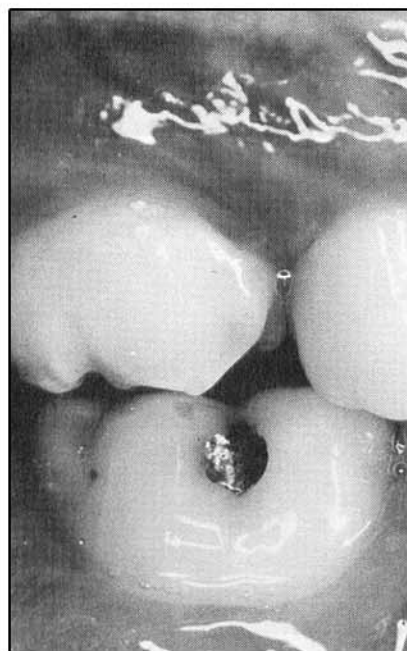


Fig. 4. Tooth #46 — 7 years after osseous grafting



biological space maintainers for five and a half years.

## Discussion:

This current report updates the developments concerning the transplanted tooth #28 initially reported by Arlin and Freeman in 1981<sup>3</sup>. As well, the treatment of the teeth #46 treated with an osseous graft, and the autotransplantation of #18 into the #16 extraction socket, is described.

Perhaps the two most striking features of J.P. are that (a) the periodontal attachment loss is commonly localized to the incisors, and mesial aspects of first molars, and (b) the extent of the destruction usually seems incommensurate with the (clinically evident) quantity of local factors and inflammation. One could hypothesize however that if this "specific infection" took place at the time when the first molars and incisors were erupting into the oral cavity, the ensuing development of an immune host response and/or subsequent establishment of bacterial antagonists and/or "burnout", would result in the subsequently erupting teeth being immune to this specific "infection" process. The plaque-retentive type of dental anatomy usually found on the mesial aspects of first molars might explain the predilection of this area to exhibit the most extensive attachment loss. The apparent absence of clinical inflammation and local factors led earlier investigators to hypothesize that J.P. was a dystrophic, and not an inflammatory disease. Recent investigations, however, have conclusively demonstrated that there does exist a sparse but virulent subgingival microbiota associated

with inflammation deeper within the pocket wall<sup>1</sup>. Osseous repair is possible if inflammation and infection are controlled.<sup>4</sup>

Various modalities for the treatment of J.P. have been reported in the literature. Systemic tetracycline taken for three weeks may eradicate the pathogenic microbiota of J.P.<sup>2</sup> and should be routinely prescribed for these patients. Osseous grafting into J.P. lesions<sup>5</sup> and eruption of affected teeth to achieve a modification of the osseous architecture has also been described<sup>6</sup>.

Baer and Gamble initially described transplantation of developing third molars in the sockets of first molars affected by J.P. Some cases demonstrated continued root development and eruption over a period of time following transplantation. The transplantation process in particular raises many interesting questions, for example, following transplantation what factors influence pulpal vitality, root development, tooth eruption, osseous regeneration and reformation of a functional periodontal ligament?

Histologically speaking, it is "Hertwig's Epithelial Root Sheath" that is necessary for root development and it is the "DENTAL FOLLICLE" that gives rise to the formation of cementum, periodontal ligament and bone<sup>8</sup>. It seems reasonable therefore that atraumatic surgery to protect the transplanted tooth is indicated in order to promote continued root development and regeneration of the periodontal attachment apparatus. Clinically, it has been found that vitality can be expected when the transplanted teeth exhibit open apices and that ankylosis can be minimized or eliminated if post-surgical stabilization is non rigid<sup>9</sup>.

Although it is not within the scope of this article to discuss all aspects of tooth transplantation, some clinical guidelines of dental auto-transplantation are listed below.

## Principles of Atraumatic Surgery:

- absence of acute inflammation at the recipient site
- minimize time of extraalveolar exposure of the transplant
- protect the soft tissues attached to the transplant
- remove interradicular bone at the recipient site to assure a passive placement of the transplant
- place transplant in infraocclusion
- non-rigid stabilization for 2-4 weeks
- prophylactic antibiotics.

## Endodontic Considerations:

- transplant teeth with open apices without undertaking prophylactic endodontic therapy
- institute interceptive endodontic therapy when signs of pulpal necrosis and/or inflammatory resorption is diagnosed.

## Signs of Transplant Ankylosis (Replacement Resorption):

- immobile transplant
- "high" percussion sound
- no eruption
- no periodontal ligament radiographically.

## Signs of Transplant Inflammatory Resorption:

- pulpal necrosis
- mobility



Fig. 6. Tooth #28 — immediately after removal; (compare stage root development to Fig. 7 illustrating #18 done 1 1/2 years later).



Fig. 7. Tooth #18 — immediately after removal (1 1/2 years after the 28 was removed, also compare to Fig. 8 illustrating #18 2 1/2 years post transplantation).



Fig. 8. Tooth #18 — immediately after extraction (2 1/2 years following its transplantation — compare to Fig. 7).

- extrusion
- percussion sensitive
- resorption cavities radiographically

## Summary:

Treatment was initiated in 1978 at which time the patient was 16 years of age. This report presents seven-year results utilizing various treatment modalities on a single patient afflicted with JP. The therapeutic result was excellent with the 4.6 and 2.8. The lower molar would seem to have a very good long term prognosis, while the long term fate of 2.8 is somewhat questionable. Even in the event that 2.8 is lost, the treatment of the 2.6 and 1.6 areas can be considered successful. Functioning as good biological space maintainers, the third molars have delayed the need for prosthetic treatment in an adolescent, at a time when prosthetic treatment would have been less than ideal.

Dr. Arlin presented this information in a table clinic at the Academy of General Dentistry Winter Clinic in Toronto in November 1985. He is a periodontist in private practice in Weston, Ontario.

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