

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. The incidence is approximately 0.1-0.2% in North America. Clinical features include: (a) circum-pubertal 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 5 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. 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 250mg. Q.I.D. for 3 weeks in order eradicate the A.a. organism, in conjunction with local therapy. 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 I have been treating over a 6½ year period to date.

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, I will illustrate the treatment and follow-up of each tooth 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 the University of Toronto Dental Library.) Comparison of the initial and subsequent radiographs over a period of 6 years demonstrate the gradual and



Fig. 1. Tooth #46 - extremely deep osseous defect

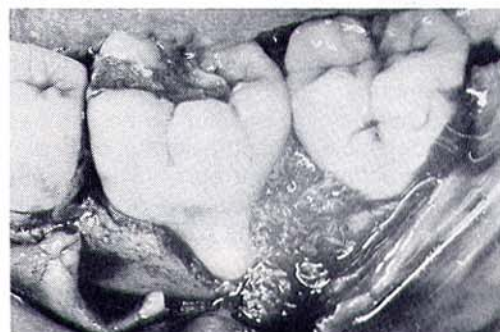


Fig. 2. Tooth #46 - immediately after placement of decalcified freeze-dried cancellous allograft

slow improvement over time (Fig. 3, 4, 5, 6 and 7). It is important to note that a minimum of 2 years is required prior to assessment of repair when dealing with osseous grafts.



Fig. 3. Tooth #46 - initial defect

Fig. 4. Tooth #46 - 6 weeks post transplantation

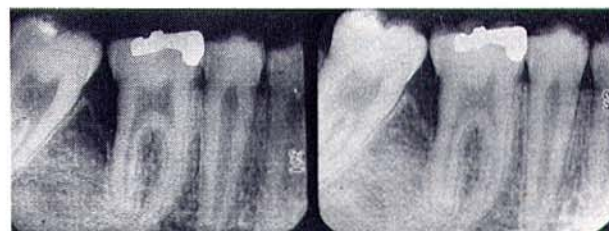


Fig. 5. Tooth #46 - 6 months post implantation

Fig. 6. - Tooth #46 - 1 year post implantation

In addition to the osseous graft, passive eruption was encouraged by periodically relieving the occlusal contacts as illustrated diagrammatically in Fig. 8; this was done because when the tooth erupts, the alveolus also 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 extrac-

tion was the only feasible recourse. At the same appointment, however, the extraction socket functioned as the recipient site for the autogenous transplantation of the unerupted 2.8 (Fig. 9). After 3 weeks of non-rigid stabilization with the tooth in infraocclusion, routine maintenance therapy ensued. Examination of sequential radiographs over a 6 year period show osseous regeneration as well as indications of root development. (Fig.

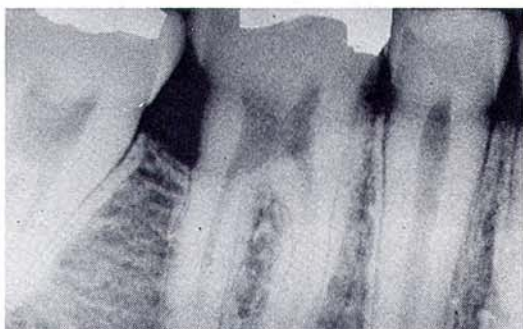


Fig. 7. Tooth #46 - 6 years post implantation

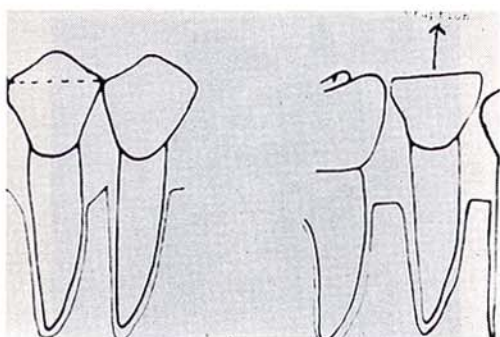


Fig. 8. Illustration of how "eruption" can "level out" a bony defect



Fig. 9. Tooth #28 - immediate post extraction - compare to Fig. 21, i.e., tooth #18 1½ years later



Fig. 10. Tooth #28 - unerupted

Fig. 11. Tooth #28 - 2 weeks post transplantation



Fig. 12. Tooth #28 - 6 weeks post implantation

Fig. 13. Tooth #28 - 3 months post transplantation



Fig. 14. Tooth #28 - 6 years post transplantation

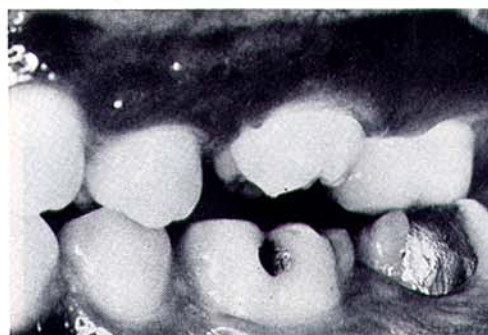


Fig. 15. Tooth #28 - 6 weeks post transplantation



Fig. 16. Tooth #28 - 6 months post transplantation

10 through 17). Additionally, clinical post-op views at 6 weeks (Fig. 15), 6 months (Fig. 16), and 6 years (Fig. 17), clearly demonstrate how the tooth has erupted into occlusion. At present, the tooth is functioning without signs or symptoms of pathology.

Tooth 1.6: Periodontal surgery revealed extensive bone loss (Fig. 18) 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



Fig. 17. Tooth #28 - 6 years post transplantation

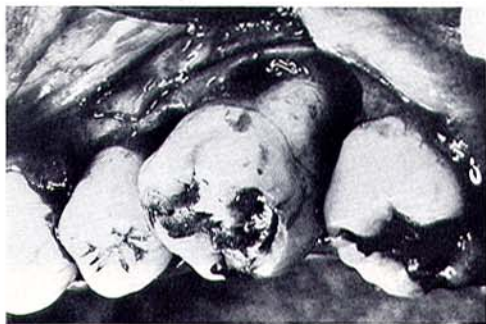


Fig. 18. Tooth #26 - extensive bone loss not amenable to osseous grafting



Fig. 19. Tooth #16 - 3 years post open curettage

were successful in maintaining the tooth over a 3-year period (Fig. 19). Due to the persistent sensitivity and extreme satisfaction with the auto transplant that by this time had already been done in the second quadrant,

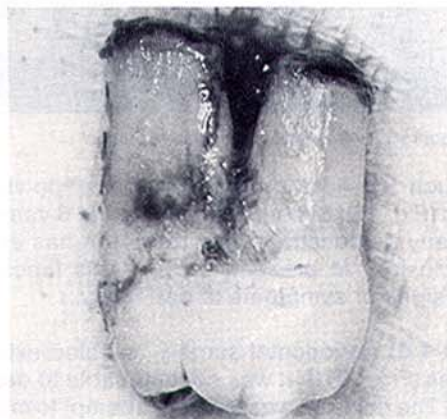


Fig. 20. Tooth #18 - immediate post extraction - compare to Fig. 9 taken 1 1/2 years earlier

it was decided that the 16 be extracted and be replaced with the auto transplant 1.8 (Fig. 20). (Note the longer root length in the 1.8 as compared to the 2.8 (Fig. 9) which was transplanted 1 1/2 years earlier.) Healing was uneventful until an endodontic fistula developed (Fig. 21) 2 1/2 years later. Endodontic treat-



Fig. 21. Tooth #18 - endodontic complications 2 1/2 years post transplantation

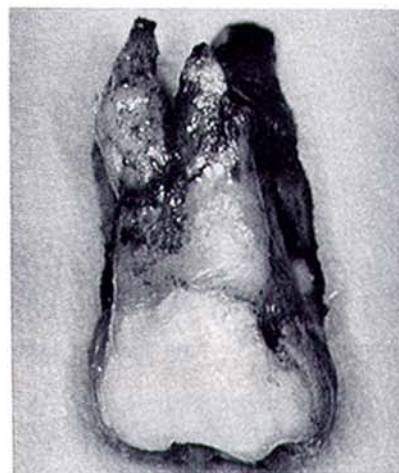


Fig. 22. Tooth #18 - immediately post extraction 2 1/2 years post transplantation - compare to Fig. 21.

ment failed and the tooth had to be removed (Fig. 22). Note the root development and apexification that occurred in situ, following the transplantation, over a 2 1/2 year period (compare Fig. 22 to 20). It should also be mentioned that although the 1.6 and 1.8 were lost, they did function as excellent biological space maintainers for 5 1/2 years.

Summary: Treatment was initiated in 1978 at which time the patient was 16 years of age. 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.