

Biomechanical Induction of Mandibular Alveolar Growth in Edentulous Growing Children

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ABSTRACT

Objective

It has traditionally been believed that alveolar bone growth and development is dependent upon dental development. The occurrence of alveolar bone development after placement of osseointegrated mandibular implants in two edentulous children is reported here. The fact that alveolar bone development occurred in the absence of natural teeth suggests that its growth and preservation is dependent upon biomechanical forces rather than the presence of teeth, as traditionally thought.

Study Design

Serial panoramic and clinical evaluations were carried out on two children, aged seven and nine years old, for five years, following placement of osseointegrated implants and their prosthetic rehabilitation.

Results

Both children showed impressive alveolar bone growth that occurred to the extent it had to be surgically reduced to permit proper hygiene around their fixed hybrid prosthesis.

Conclusions

It is proposed that a biomechanical "message," instead of a biochemical-biological one controls alveolar bone growth, and that the implants permit biomechanical loading that controls alveolar bone growth to exist in the mandible.

INTRODUCTION

A large body of data has been collected over the last 50 years delineating in great detail the role that biomechanics plays on bone growth and development.¹ We know that loads on bone causes strains that:

a) result in mechanically controlled bone modeling which responds predictably to different levels of mechanical usage;²

b) increase bone strength due to architectural changes secondary to the time-average history of repeated loads;^{3,4} and

c) create internal bone stresses.³

In normal growing bone, these architectural changes result in increased bone mass and increased bone strength, which compensate for the demands placed on bone as bone stress increases.⁵⁻⁷ Disuse of bone, on the other hand, as seen in patients with profound paralysis, results in a decrease of bone mass and to a lesser degree bone size.^{1,5,6} It follows that these same mechanisms are at work in the mandible whereby stimulation of bone size and shape during growth are in part the result of biomechanical forces generated within bone.⁴

It traditionally has been taught that the alveolar bone of the jaw develops secondary to tooth development and eruption, and in the absence of teeth, alveolar bone does not occur.⁸⁻¹¹ Similarly, it is known that not only does the alveolar process not develop if teeth are absent, but the alveolar process also undergoes regressive remodeling when teeth are lost. The reasons for these phenomena are not completely understood.

It has been shown that, biomechanically, peri-implant compressive strains that occur during implant placement also lead to dissipation of heat to the immediate surrounding.¹² This in turn will lead to apoptosis in the immediate periphery, namely the surrounding bone. Additionally, it is well known that the level of osteocyte death is directly correlated with the amount of bone resorption. Biomechanically, this can be related to the level of strain imparted by the implant and its placement. This

combination of heat and strain can lead to bone resorption and a decrease in implant stability; however, during the healing process, it is clear that some biologic change occurs which not only improves implant stability, but may even aid in the repair and subsequent growth of the alveolar bone. In 1892, Wolff demonstrated that bone is impressively responsive to the mechanical stresses and loads placed on it. The principle of Wolff's law is summarized by the following: skeletal elements are strategically placed to optimize strength in relation to the distribution of applied loading, and that the mass of the skeletal elements is directly related to the magnitude of the applied loads.¹³ Other well known studies more specifically described the biomechanical interactions: tension results in fibrous connective tissue, shear forms cartilage, and compression produces bone.¹⁴ In 1960, Pauwels¹⁵ determined that physical factors can cause stress and deformation of mesenchymal progenitor stem cells and that such mechanical stimuli could possibly alter the gene expression, increasing the development of specific cells. Later studies have also confirmed that that local tissue stresses and strains alter both the pressure on bone cells and cell differentiation.¹⁶

Nevertheless, ongoing research still is attempting to determine the exact mechanism by which the mechanical signals stimulate remodeling and regeneration in bone. At all anatomic levels – tissue, cellular, and molecular – a chain of events occur due to biomechanical forces that leads to continued growth and development of bone. It is entirely plausible that dental implants placed in a growing child, whose bony metabolism is significantly more active than normal adults, would only enhance this chain of events. The excellent blood supply, coupled with the increased capacity for osseous healing, give children an even greater advantage for implant success. At the osseous tissue level, mechanical stimulus via stress/strain can help

guide the bone toward different patterns of tissue formation. These mechanical signals are then transmitted cellularly, altering cell shape, size, and extracellular matrix components. Subsequently, these cellular signals are transferred to the molecular level, where intra-cellular signaling will alter the cell activities.¹⁷

Here, we report on two children, ages seven and nine years with oligodontia where alveolar bone has failed to develop and subsequently developed after biomechanical stimulation of the mandible occurred secondary to masticatory function via osseointegrated implant supported fixed prosthesis.

It is postulated that the development of alveolar bone during growth and development is in large part secondary to biomechanical stimulation, rather than simply to the development of teeth as has been traditionally believed.

PATIENT REPORTS

Case I

A patient with ectodermal dysplasia was seen initially at age nine when he presented for possible osseointegrated implant reconstructive surgery. He had a significantly osteoporotic mandible without an alveolar process and associated masticatory dysfunction. He exhibited the characteristic stigmata of ectodermal dysplasia including fine-sparse hair, facial dysmorphism, and partial anodontia. A panoramic film confirmed the presence of two maxillary deciduous and two permanent teeth and no mandibular alveolar bone (Figures 1A and 1B). On the basis of the clinical examination, cephalometric and panoramic radiographic evaluations, it was recommended that he undergo mandibular surgical reconstruction via five osseointegrated implants, a fixed prosthetic appliance in the mandible, and a removal full maxillary denture.

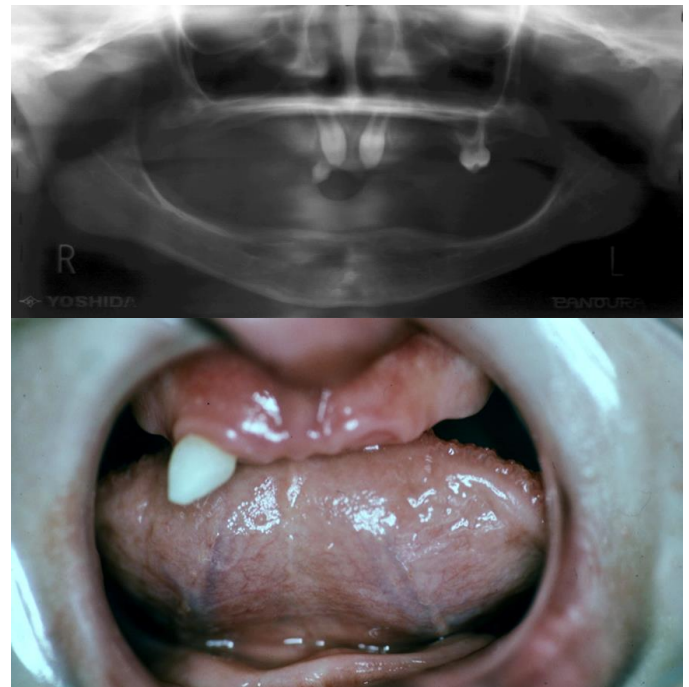


Figure 1A (top), Pre-treatment October 1987 of a 9 YOM with ectodermal dysplasia. B (bottom), Intraoral photograph of 9 YOM with ectodermal dysplasia.

At age 11, the patient underwent placement of five mandibular Bränemark implants. All implants osseointegrated and a lower implant-supported hybrid fixed prosthesis and a maxillary full denture were made for the patient (Figure 1C). At the one year visit, it was noticed that significant alveolar bone growth, both around the implants in the anterior mandible, and distal to the implants in the posterior jaw, had occurred. During the next year, the alveolar bone continued to demonstrate exuberant growth that interfered with the patient's ability to exercise good hygiene around the lower prosthesis. At this point, it was deemed necessary to excise some of his bone. At 14 years old, the patient was able to maintain his alveolar bone growth and mandibular height. (Figures 1D and 1E)

Case 2

This patient is the younger brother of case 1, evaluated at nine years of age. He also presented with stigmata of ectodermal dysplasia, an atrophic mandible without any alveolar bone, severe hypodontia (Figures 2A and 2B), and associated masticatory dysfunction. As was the case with his brother, on the basis of the physical examination, cephalometric and panoramic radiographs, the patient underwent placement of five Bränemark implants in his lower arch at age 11. (Figure 2C) He tolerated the procedure well and all of the implants osseointegrated. An implant-borne hybrid fixed lower prosthesis and a removal maxillary full denture were fabricated four months after implant placement.

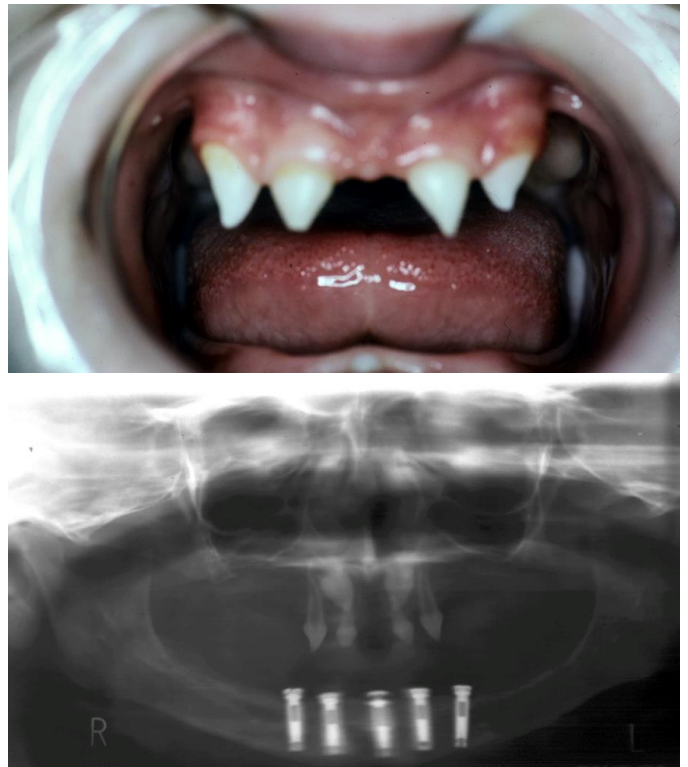


Figure 2A (top), Intraoral photo of patient's younger brother at 9 years old. B (middle), Patient's 9 year-old brother was also diagnosed with ectodermal dysplasia. C (bottom), 11 YOM post Bränemark implants in mandible.

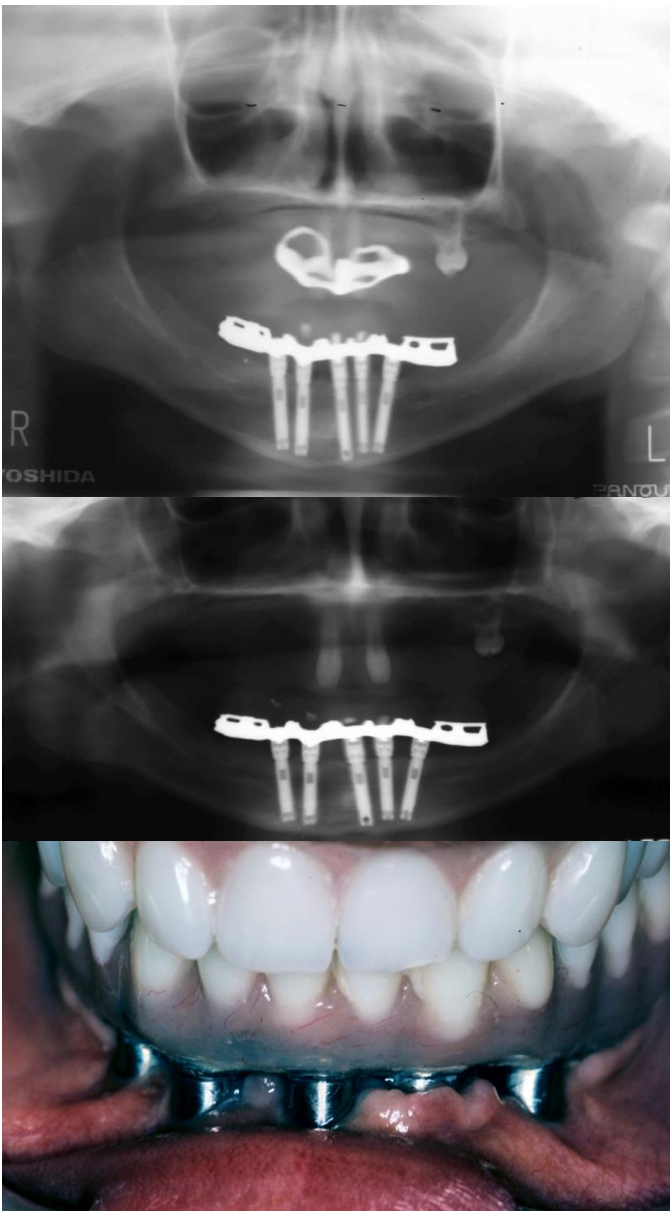


Figure 1C (top), Same patient, at 11 years old with maxillary prosthetic device; fixed hybrid Bränemark implant in the mandible. D (middle), Same patient, at 14 years old with successfully osseointegrated mandibular implants. E (bottom), Clinical photograph with implant supported over-dentures.

During his one-year follow-up visit, an exuberant growth of alveolar bone was observed on the radiographs. (Figure 2D) Since there was interference with hygiene of the implants around the prosthesis, the alveolar bone was reduced surgically. Two years later, the patient presented with the same problem and underwent a second bony reduction. (Figure 2E) At age 16, five years after placement of his implants, he has excellent radiographic evidence of maintenance of alveolar bone height. (Figure 2F)

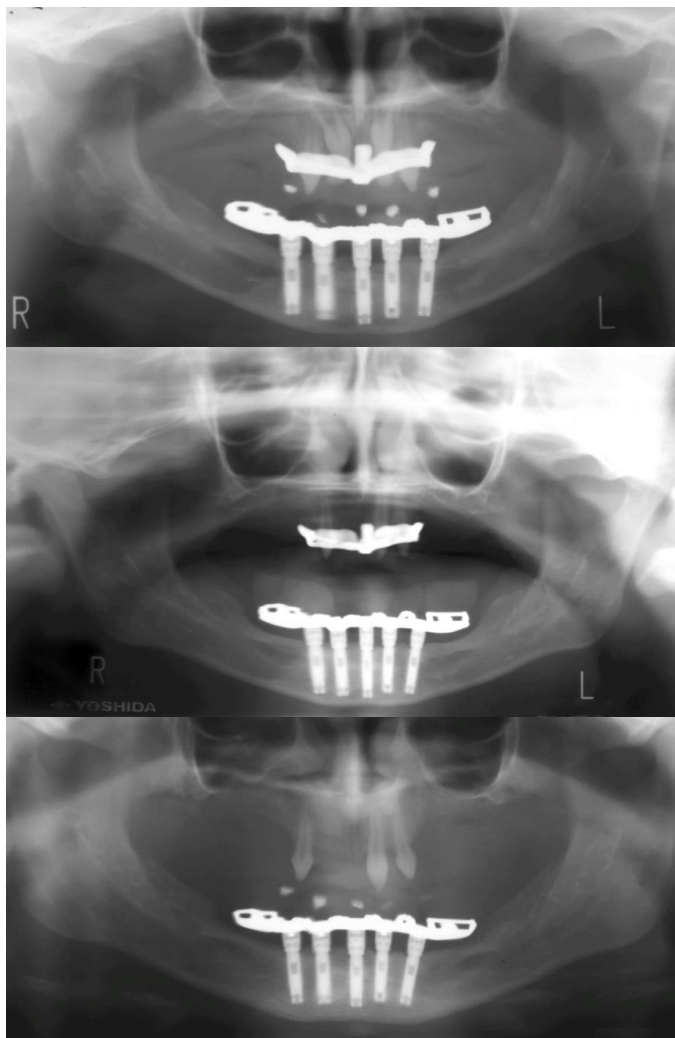


Figure 2D (top), 12 YOM now one year post-implant placement with full prosthesis. E (middle), 14 YOM with successfully integrated implants. F (bottom), 16 YOM 5 years post implant placement.

DISCUSSION

Traditional beliefs regarding alveolar bone development espoused in current anatomy and orthodontics texts are that alveolar bone development is dependent upon and directly related to the underlying tooth development and eruption.^{8-11, 18} This hypothesis has not been adequately tested and herein we present data refuting it.

Biomechanical stimulation of bone development has received considerable attention during the past decade.^{1-11, 18-21}

Both genetics and biomechanical factors are responsible for the adult size and shape of bones. Multiple reports exist in the literature that supports the existence of two separate mechanisms for bone growth: endochondral growth and bone remodeling.²⁰⁻²⁴ Epiphyseal growth via endochondral ossification basically determines the length of bones. Modeling determines their size and shape. Modeling shapes bone and increases bone mass in response to bone loading.^{20, 22-24} It follows then, that increasing mechanical usage increases bone mass. Disuse on the other hand results in a net bone loss.^{21, 24}

Supporting information on the role of biomechanical factors in alveolar bone development exists. A complete description of mechanically controlled remodeling and modeling effects on bone gain, losses and turnover follows out this paper's objectives. Nonetheless, it is interesting that in a recent paper by Taylor, an edentulous patient is presented in whom growth of the mandibular alveolar process occurred after placement of osseointegrated titanium implants.²⁵ In this instance, the posterior mandible grew 3 mm in the vertical dimension below an implant-supported fixed prosthesis. In the absence of systemic disease or metabolic disorders, the

author postulated that the increase in mandibular loading following placement of a fixed prosthesis. He speculated that the presence of anterior osseointegrated implants supporting the fixed prosthesis allowed the patient to function at a level close to that of a dentate person and that the changes in bone function resulted in structural alterations and proliferation of bone.

In this report, we present two patients in whom a similar development of alveolar bone was observed in the mandible following placement of osseointegrated

titanium implants and fabrication of implant-supported prostheses. In contrast to the patient reported by Taylor, who was 50 years old, our patients were young (ages nine and eleven) and alveolar bone development occurred in the entire mandible. Both children developed large amounts of alveolar bone. The younger brother developed a larger amount of bone growth, which is likely due to the fact that his facial growth potential exceeded that of the older brother.

Although it is well accepted in the literature that osseointegrated implants do not re-establish natural alveolar bone volume, it also has been reported that implants do increase the density of mineralization of the remaining adjacent bone.²⁶⁻²⁹ It is postulated that the bone growth observed in the patients reported here is the direct result of biomechanically stress-induced bone stimulation caused by the presence of functionally loaded implants.³⁰

Currently, concerns regarding implant placement in actively growing children certainly warrant exploration. There are obvious drawbacks, including interfering with the position and eruption of underlying permanent dentition; however, this discussion is not the main focus of our paper. Our patients suffered from

ectodermal dysplasia complicated by anodontia; as such, there were no underlying teeth on which to negatively affect. Even reports on the drawbacks of implant placement indicate that an osseointegrated implant would act as an ankylosed primary tooth, and would appear submerged into the alveolus; thus implying that alveolar bone growth could possibly continue.³¹

It is interesting to postulate that the increase in bone height in the posterior mandible of these patients may be the result of increased tension loads on the mandible without significant bending. This type of bone strain is known to reach the modeling threshold of affected trabeculae and to produce bone drifts, which result in bone mass increase.³² A similar mechanism may be at play in the patients reported by Bukes et al. and Render in whom spontaneous alveolar ridge bone growth occurred under fixed prosthesis pontics in the posterior mandible.^{33, 34} These authors hypothesized that functional stress due to mandibular flexure may be the etiologic factor in bone apposition

CONCLUSION

We postulate that the alveolar bone development observed in these patients is primarily the result of biomechanical stimulation of the mandible secondary to increased masticatory function may possible by the presence of osseointegrated implants.

This loading and the resultant biomechanical effects on bone are likely similar to those induced by the presence of natural teeth. Both the internal strains and the surface tension and compression forces appear to be equal in nature and theoretically should produce the same bone growth results. Early placement of implants may be beneficial in young patients to stimulate alveolar bone development.

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