MANDIBULAR BONE GROWTH INDUCED BY A HYDROXYLAPATITE-COATED SUBPERIOSTEAL IMPLANT: A CASE REPORT

John M. Fish, DDS
Carl E. Misch, DDS, MDS

KEY WORDS

Subperiosteal
Bone growth
Hydroxylapatite

History of the tripodal mandibular subperiosteal implant and the evolution of its design are discussed. Basic principles of bone physiology are reviewed especially as they relate to bone response to loading. Modeling and remodeling are controlled by a strain-related environment. Modeling can alter the shape and volume of bone. This aspect of bone growth has been reported with transosteal implants. A case report is presented in which apparent bone growth occurred following placement of a hydroxylapatite-coated subperiosteal implant. The implant was successfully revised following an acute infection around one of the permucosal sites.

John M. Fish, DDS, *is a diplomate of the American Board of Oral Implantology and a fellow in the American Academy of Implant Dentistry. Address correspondence to Dr Fish at PO Box 665, Hildebran, NC 28637.*

Carl E. Misch, DDS, MDS, *is the director of the Misch Implant Institute, an associate professor at the University of Pittsburgh, and an adjunct professor at the University of Alabama at Birmingham. He is also a diplomate of the American Board of Oral Implantology and is co-chairman of the International Congress of Oral Implantologists.*

INTRODUCTION

The mandibular complete denture is the most unstable, unretentive, and perhaps the most unpredictable appliance offered in routine dental practice. During function, it moves approximately five times more than the maxillary denture.[1] Obviously, these problems are magnified when a lower denture opposes a full arch of natural dentition.

Dental practitioners can face a daunting challenge when attempting to restore function to a patient with a highly resorbed (Misch/Judy Division C-h or Division D) mandible. The treatment of choice in these cases may involve autogenous bone grafting and subsequent placement of endosseous root form implants. This option is used primarily when a fixed prosthesis is desired or stress factors are great.

However, if bone grafting is contraindicated, funds are limited, and/or patient expectations dictate the use of a completely implant-supported removable prosthesis, a subperiosteal implant often becomes the treatment of choice. The subperiosteal affords other advantages, including anterior and posterior support as well as the potential to restore large amounts of vertical dimension in the implant-borne prosthesis.

LITERATURE REVIEW

Gustav Dahl of Sweden first proposed a subperiosteal design and insertion protocol in 1937.[2] It was first introduced to the United States by Goldberg and Gershkoff[3] in 1948 and has since evolved through major changes in materials, design, surgery, and prosthetics. A group of Americans, including Jermyn, Bodine, Cranin, Herschfus, Lee, Linkow, Mentag, and Weber, continued to influence substructure de-

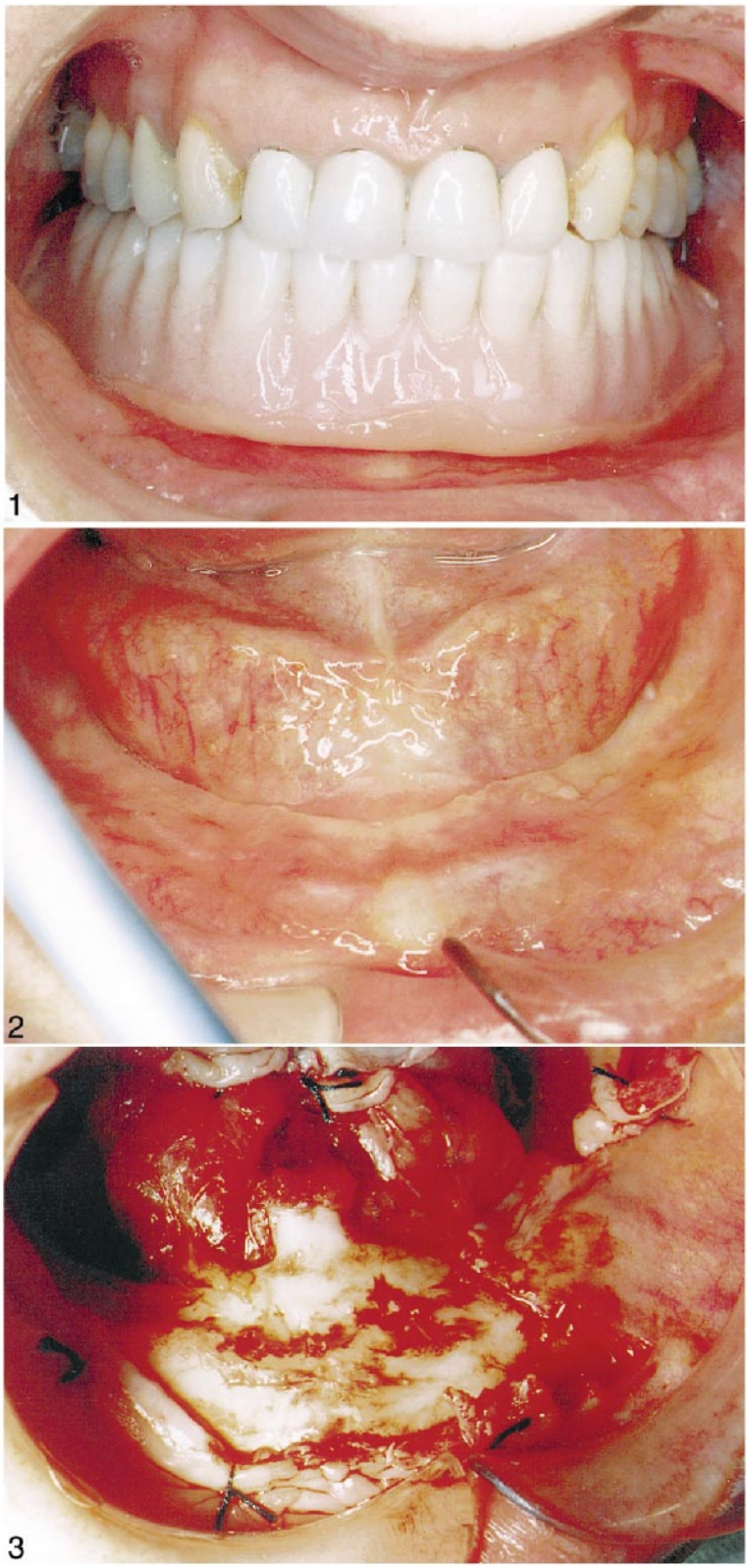

Figure 1. Preoperative view of the patient's lower denture April 11, 1985.
Figure 2. Preoperative view of the patient's severely atrophic edentulous lower arch.
Figure 3. Stage I surgery. Note the high genial tubercles and genioglossus insertion.

sign, which became rather consistent over the subsequent 20 years.[4] Design trends included reduced bulk of metal on the ridge crest with primary support transfer to more peripheral areas of the mandible such as the external oblique ridges, digastric fossae, and the mental protuberance. Later, James[5] further extended the support concept to include the lateral ascending rami.

Other variations included discontinuation of the primary strut over the mental foramen if less than 5 mm of bone height existed between the foramen and the ridge crest.[4] This concept underwent further evolution until the support system consisted of three separate ''islands,'' with one in the mandibular symphysis and bilateral support in the external oblique and ascending rami. This tripodal design[6,7] totally avoided the possibility of ''settling'' of the implant into the mental neurovascular bundles with resultant paresthesia.

BONE MECHANICS AND PHYSIOLOGY

It is well known that bone density and volume can be influenced by functional loading as well as hormonal factors.[8] Even when there is an estrogen imbalance, functional loading is still capable of effectively competing for available calcium, thereby maintaining bone mass.[9] As early as 1892, Wolfe[10] observed that trabecular bone will realign itself in response to functional demands. Strain placed on bone tissue can actually precipitate a chain of events within the bone cell, which facilitates bone maintenance and potentially bone growth.

Frost[11] has postulated that cortical bone has a physiologic loading zone between 50 and 1500 microstrain units (ME). Loads below this range were considered ''trivial'' loads insufficient to cause a bone response. Conversely, loads greater than this range were able to cause a potentially pathologic overload state. Loads within the physiologic range stimulate cortical bone remodeling by an organized method that per-

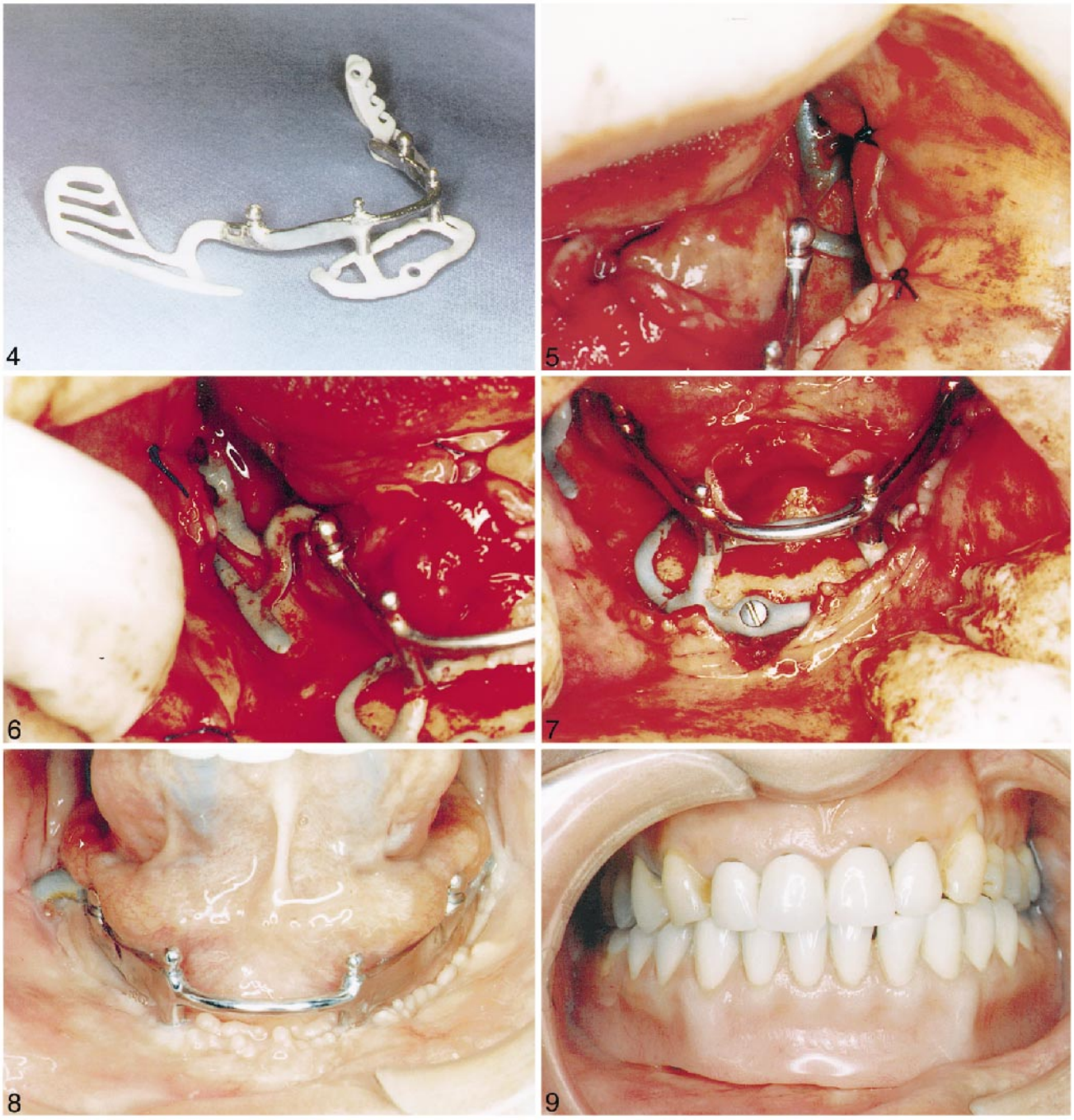

Figure 4. The hydroxylapatite-coated mandibular subperiosteal implant.
Figure 5. Stage II surgery: left posterior.
Figure 6. Stage II surgery: right posterior.
Figure 7. Stage II surgery: mental region with Luhr fixation screw. Note close adaptation of implant to underlying bone.
Figure 8. Five weeks postoperative photos. Note delayed healing around right posterior permucosal site.
Figure 9. Anterior view of patient's complete mandibular overdenture January 31, 1986.

mits the bone to remain primarily lamellar in histologic sections. If the microstrain of bone is allowed to remain between the physiologic and mild overload zone, an actual volume increase in the bone may result. In a study by Hassler *et al*,[12] experimentally induced stresses in the range of 2.48×10 N/mm^2 have been observed to cause bone growth. On the other hand, stresses greater than 6.9×10 N/mm^2 were observed to cause cell necrosis.

It has further been shown that bone responds differently to static versus cyclic loads. Studies with avian ulnae

with static loads actually caused a 13% decrease in the cross-sectional area whereas cyclic loads caused a 24% increase in the cross-sectional area.[13] It has also been shown that the greater the rate of applied strain, the larger the increase in bone volume.[14] Furthermore, many cycles of low magnitude strain can cause a bone response similar to fewer cycles of larger (but still physiologic) loads.[15]

CASE STUDY: A HYDROXYLAPATITE-COATED MANDIBULAR SUBPERIOSTEAL

The patient was a 50-year-old Caucasian woman who first presented to the office in April 1985 for a complete examination. Her mandible was extremely atrophic (Figs 1, 2) with dehiscent mental neurovascular bundles (See also the preoperative radiograph, Fig 10). The patient was treatment-planned for a hydroxylapatite (HA) coated tripodal subperiosteal implant. This design was chosen primarily because it avoided aggressive surgery in the areas of the mental neurovascular bundles. The HA coating was elected because it could allow the potential for an osseointegrated interface and might facilitate a better fibrous attachment to the overlying gingiva and mucosa.

The first-stage bone impression was performed in October 1985 using oral sedation and local anesthetic. The full arch dissection was performed in the usual manner except that the area overlying the left mental bundle was left fully attached, exposing only the areas mesial and distal to this landmark (Fig 3). The external oblique ridges, ascending rami, superior genial tubercles, mandibular symphysis, and digastric fossae were all exposed and identified. These anatomic landmarks were impressed using a polyvinyl siloxane (PVS) wash material in an acrylic custom tray made directly on the mandible. A tentative centric jaw relation record was made at the patient's estimated occlusal vertical dimension using a PVS wash impression in the patient's existing denture. The surgical site was then closed with interrupted 3–0 Surgilon sutures. Routine postoperative instructions were given.

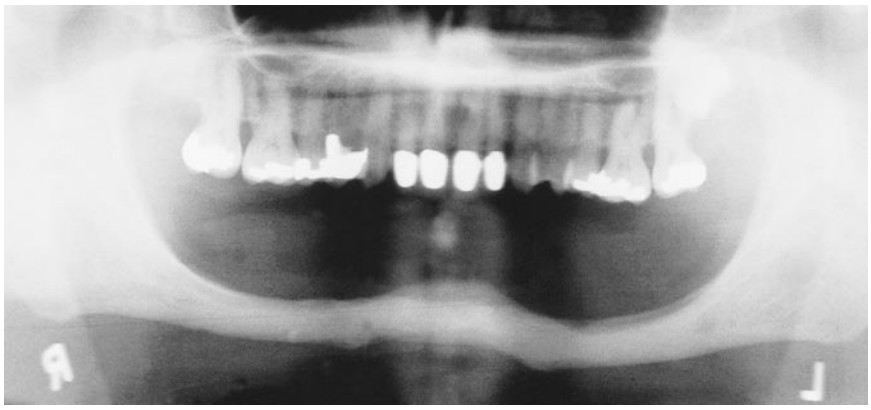

Figure 10. Preoperative panoramic radiograph clearly showing the patient's atrophic mandible and opposing full arch of natural dentition (dated April 11, 1985).

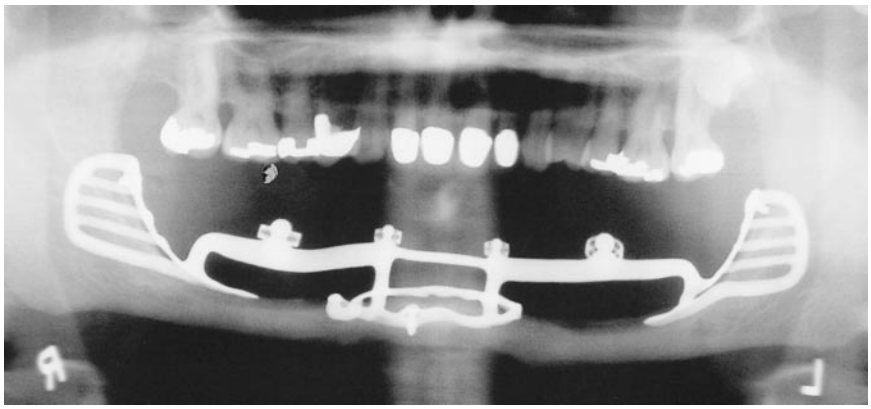

Figure 11. Postoperative panoramic radiograph of the completed subperiosteal implant (dated December 31, 1985).

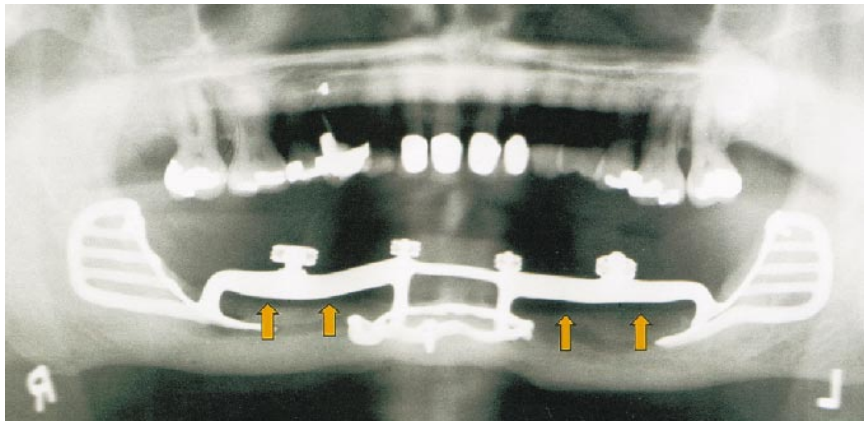

Figure 12. Panoramic radiograph of the patient on March 1, 1999. Arrows denote bone regeneration along superior aspect of the mandible.

Implant design included a significant increase in the occlusogingival dimension of the mesobar distal to the two anterior posts (Fig 4). It was hoped that this might better resist occlusal forces from the natural dentition and possibly help compensate for its long

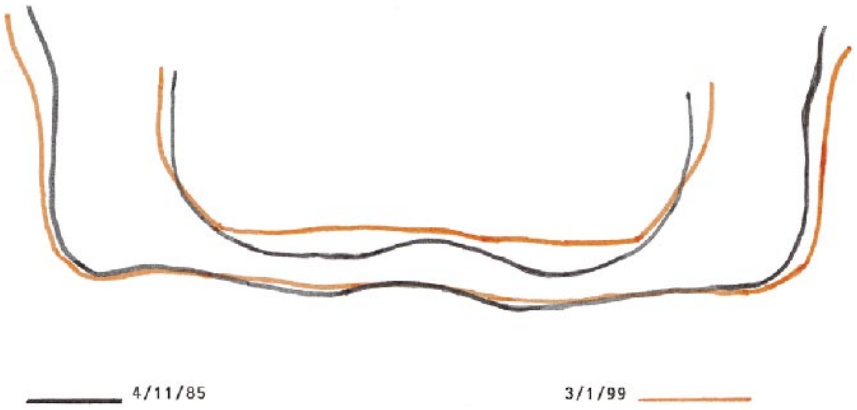

Figure 13. Tracings of preoperative (April 11, 1985) and 14-year postoperative (March 1, 1999) panoramic radiographs.

span and lack of supporting substructure. It is well documented that an increase in the vertical dimension of a metal ''beam'' can have a profound effect on its flexibility. In fact, stiffness increases relative to the cube of the occlusogingival dimension. Therefore, doubling of the prosthesis height results in eight times less deformation.[16]

Because all of her alveolar bone had resorbed superior to her inferior alveolar neurovascular bundles, there was ample vertical height available to increase the height of the bar without risk of violating the occlusal plane. A removable ''O-ring''-retained overdenture was also treatment planned.

The second stage insertion of the implant was performed approximately 3 weeks later in November 1985. (Figs 5, 6, 7) Again, incisions were designed to avoid the dehiscent mental neurovascular bundles. The implant was carried to place and its close bony adaptation was verified using a periodontal probe. Three vitallium retention screws were placed for fixation, two in the external oblique ridges and one in the mandibular symphysis slightly off the midline (Fig 7). The case was closed again with 3–0 Surgilon sutures and the implant was placed into immediate function with a processed removable, tooth-colored O-ring-retained temporary. This prosthesis had been fabricated by the dental laboratory and designed to approximate the configuration of the patient's final overdenture.

The patient experienced some delayed initial healing around the right posterior permucosal post (Fig 8). This was apparently because of the fact that the HA coating was inadvertently placed on the permucosal portion of the implant, which extended well into the oral cavity. Subsequent attempts to remove the HA coating by polishing proved ineffective.

The fabrication of the overdenture began approximately 2 weeks after the implant was inserted. The prosthetic appliance was delivered in January 1986. The patient subsequently moved from North Carolina to the West Coast (Fig 9). Sporadic communication with her over the next several years was recorded in her chart. She was not seen again in this office for a period of over 13 years.

When she returned in March 1999, her chief complaint was a history of acute swelling and discomfort around the right posterior implant permucosal site. During her 13-year absence, she had a new overdenture made but had no history of any other problems. The patient did, however, report difficulty flossing because of a decrease in the amount of space between her lower ridge and the mesobar. The initial clinical impression was that the subperiosteal implant had settled into the hard and soft tissue, and as a consequence less vertical space was available for her oral hygiene procedures.

However, her panoramic radiograph revealed a very significant finding. The X ray gave the appearance of vertical bone growth of the body of the mandible, especially evident superior to the inferior alveolar neurovascular bundle, extending into the symphysis area. Indeed, comparison of the original radiographs (Figs 10, 11) with the one taken at this exam (Fig 12) showed an approximate 70–80% increase in bone height. The orange arrows in Fig 12 denote the areas of bone growth superior to the neurovascular bundle. Tracings of her preoperative panorex (April 11, 1985) superimposed upon her panorex of March 1, 1999, are displayed in Fig 13. This further supports the presumption of significant bone growth.

Clinical examination revealed some swelling, erythema, and purulence in the lower right posterior permucosal (Fig 14) site, which interfered with proper seating of her denture. It appeared that plaque contamination of the HA coating in this area had led to a chronic inflammatory response with granulation tissue formation. It was decided to surgically reopen this area in an attempt to salvage the distal portion of the implant.

At her surgical visit, the patient was given intravenous sedation with midazolam, dexamethasone, and meperidine. After administration of local anesthesia, the right posterior permucosal site was re-entered (Fig 15). After meticulous degranulation of this area, it was noted that the distobuccal minor strut that supported the permucosal post had fractured (Fig 16). This strut was carefully removed (Fig 17). A most significant finding upon re-entry was the abundant overgrowth of bone completely engulfing the right posterior subperiosteal portion of the implant (Figs 16, 17). Bone growth was also clearly seen superior to the mental foramen that previously had been dehiscent (Fig 18). Clearly, significant bone growth had occurred.

The remaining exposed metal was aggressively decontaminated with a slurry of pumice and tetracycline, then polished with white stones and rubber

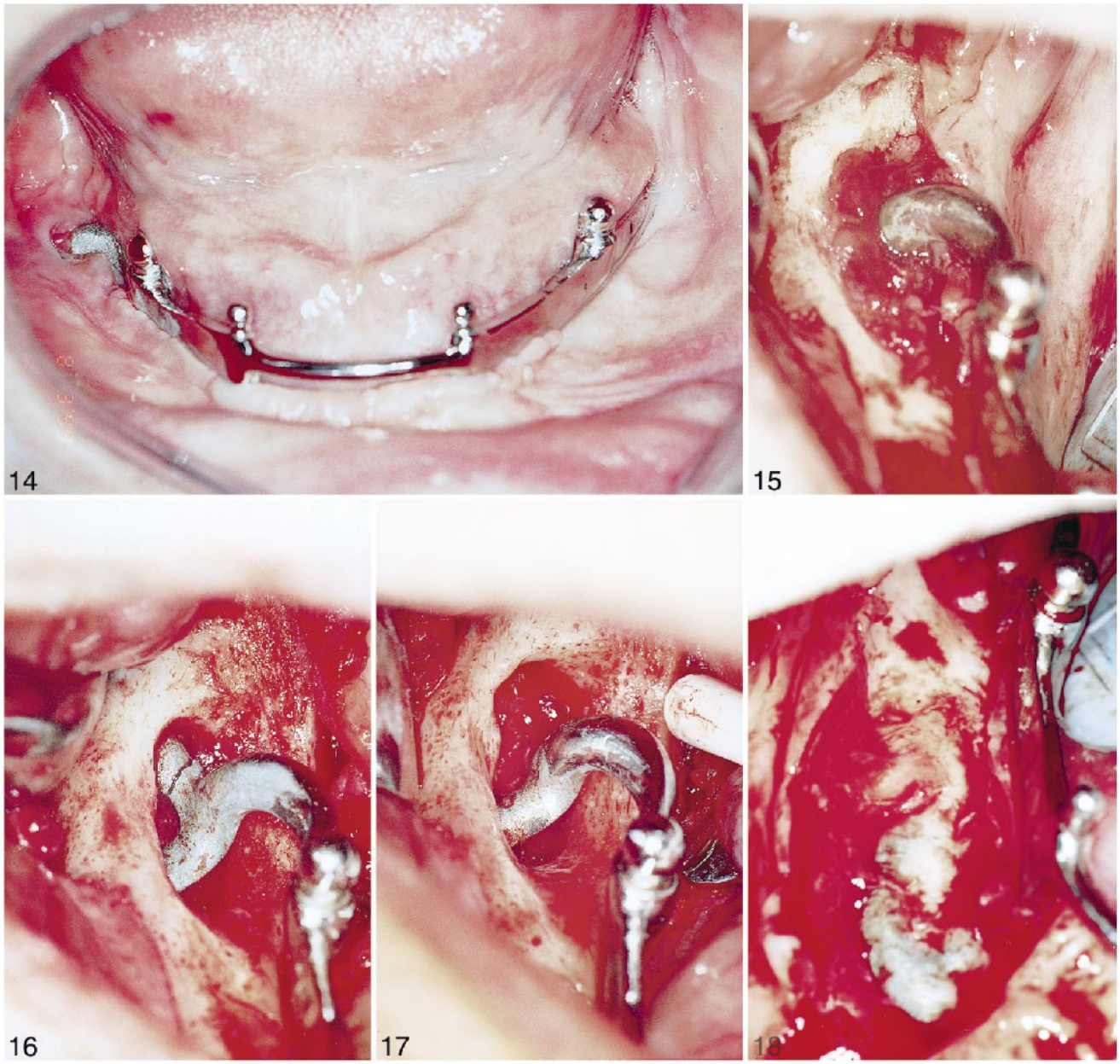

Figure 14. Re-examination on March 1, 1999. Note the acute infection with purulence in the right posterior permucosal site.
Figure 15. Surgical site showing the strut surrounded by granulation tissue.
Figure 16. View of the implant substructures following ''degranulation'' of the surgical site showing the strut surrounded by bone. Note that the distobuccal minor strut had fractured.
Figure 17. View of the surgical site after removal of the fractured minor strut and decontamination and polishing of the remaining metal.
Figure 18. Another view of the surgical site showing bone growth superior to the mental nerve bundle.

points. The use of tetracycline has been advocated by Wittrig *et al*[17] to promote cellular growth adjacent to an implant. The bony defect was filled with a 50/50 mixture of dense HA and demineralized freeze-dried bone with a small amount of ampicillin powder. Bactericidal antibiotics such as ampicillin have been successfully used in maxillary sinus augmentation.[18] Use of ampicillin, even in high concentrations, has not been shown to be destructive to bone-inductive proteins.[19] The site was closed with interrupted Vicryl sutures. She was given a prescription for hydrocodone/APAP, ibuprofen (600 mg) qid, and chlorhexidine rinse.

The patient was seen 5 days later for a routine postoperative visit. She reported some paresthesia on the right side of her lower lip. At the 2-week

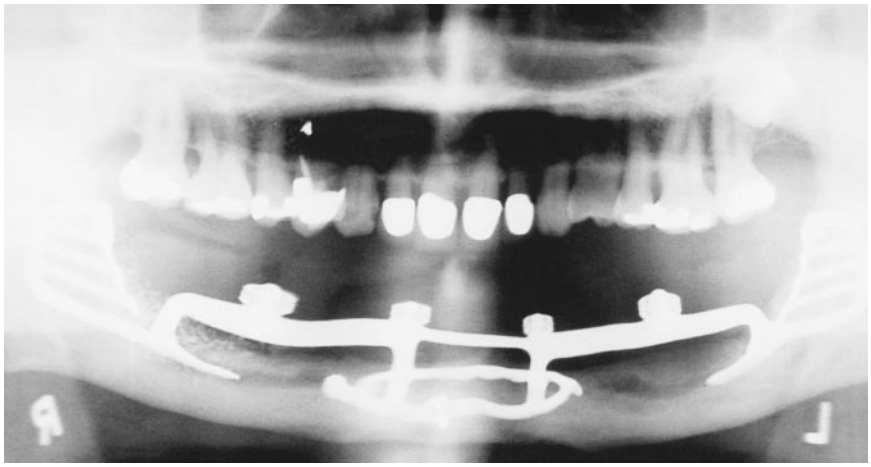

Figure 19. Panoramic radiograph taken post ''re-entry'' of lower right permucosal site showing hydroxylapatite graft material and alteration of the framework.

postoperative exam, her soft tissue healing was very good. Minor adjustments were made to her occlusion and to the flange of her existing denture. A postoperative panorex radiograph shows the presence of dense HA in the patient's lower right area (Fig 19).

The patient then returned to the office 6 months later. Her previously reported paresthesia was no longer a problem. Soft tissue healing was excellent (Fig 20, 21). Fabrication of a new lower overdenture was done (Fig 22). Careful attention was given to her occlusal scheme in order to provide cuspid disclusion and to lighten occlusal forces in the right posterior. This was done to protect the already weakened substructure on her lower right side.

Discussion

The Bosker transmandibular implant (TMI) is a one-stage device designed to rehabilitate the atrophic mandible without the need for bone grafting procedures.[20–23] It is similar to Small's mandibular staple-type implant and has four permucosal posts allowing a ''rigid-box-frame'' design to support an overlying mesobar.[24,25] The TMI implant is fabricated from a gold alloy, rather than a titanium alloy like the staple. Its design allows for the provision of a totally implant-supported prosthesis that is cantilevered distally from the four posts located in the mandibular symphysis area. Its use has been advocated in mandibles with as little as 4 to 6 mm of vertical bone height without the need for bone grafting procedures.[20–23,26]

One purported advantage of the TMI system is its ability to promote mandibular bone growth. Bosker *et al.*[27] evaluated 128 patients over a 6-year period treated with TMI implants. They were able to demonstrate in 74 cases significant bone volume growth at the superior border of the mandible.[27] Barber *et al*[24] conjectured that this bone growth is due to the conversion of occlusal pressure into tensile forces at the superior cortex of the mandible distal to the two lateral permucosal posts. This, they believe, could account for the observed apposition of bone.

Cortical bone density is likely the most influential factor determining bone stiffness and, ultimately, strength. Therefore, mechanical strain in response to loading is highly dependent on cortical bone density.[28] Mechanical properties of bone are also highly dependent on the rate of loading. Experimentally, high rates of strain can cause bone to act both stronger and stiffer. This means that at high strain rates bone can resist higher loads before brittle deformation and failure.[29] Bone can adapt and reduce strain by a number of different mechanisms. It can occur by apposition and/or resorption or even by changes in mineral content, resulting in the eventual alteration of elasticity versus stiffness.[30–32]

Although a high degree of variability exists, the mandible has been described as transversely isotropic. This means that it has the same mechanical

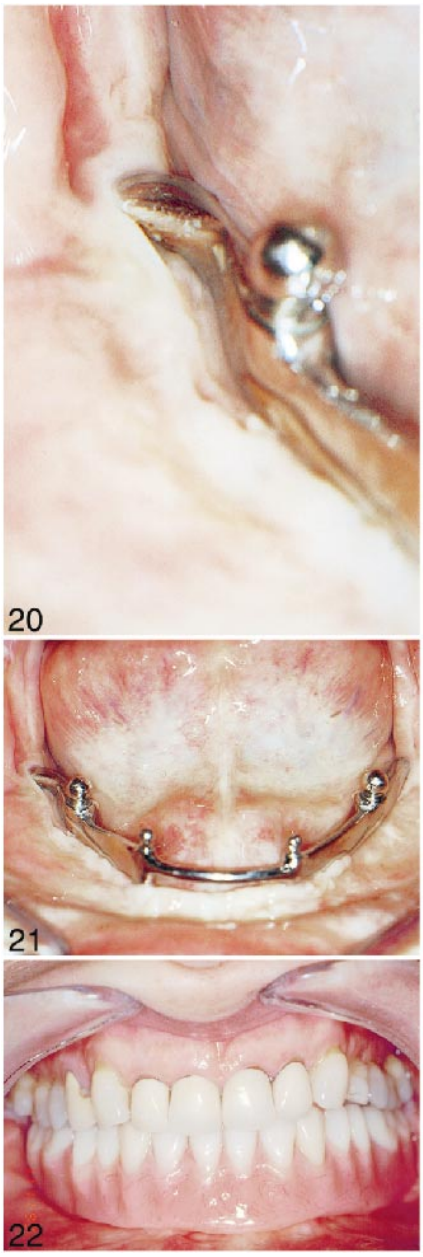

Figure 20. Seven-month post-''re-entry'' view (October 6, 1999) of the right permucosal site showing optimal soft tissue healing.
Figure 21. Full arch view of the salvaged implant.
Figure 22. Final view of the patient's completed overdenture.

properties in two of the three axes. The mandible is stiffest around its arch and can be likened to a long bone that has been bent into a curved beam.[33] Clinically, the densest bone is located between the mental foramina. Bone in the posterior mandible generally has less trabeculation and, correspondingly, less density. However, mandibular cortical bone plays the most important role in the dissipation of masticatory forces.[34,35]

The mandible has flexure toward the midline associated with opening and protrusive movements. This flexure has been measured to be 0.8 mm on the first molar region and increases as the measurements are made more distal.[36–38] In addition to horizontal flexure, the mandible may have torsion during parafunction, from the effect of the masseter muscle attachments. The degree of mandibular flexure increases as the size of the body of the mandible decreases, and may even approach the cube of the difference. Hence, a 27-fold increase in flexibility may result from a one-third decrease in bone volume. A subperiosteal implant may restrict the movement of the mandible if it obtains a direct bone interface, just as mandibular flexure is reduced when a full arch splint is applied to the teeth.

In the case presented here, it is hypothesized that the combination of mandibular flexare, implant design, the HA coating, and the amount of load during function and parafunction could all be interrelated factors allowing bone growth to occur. The combination of these factors during cyclic loading could have allowed a condition of physiologic microstrain to the bone. After many cycles of loading in this environment, the apposition of bone along the superior surface of the mandible became evident.

The apparent increase in bone height could at first glance be thought to be a result of the settling of the subperiosteal implant. However, the canals were dehiscent at Stage I and II surgery, and the preoperative and postinsertion panoramic radiographs also demonstrate no bone over the canals bilaterally (Figs 10, 11). The 13-year postoperative X ray and direct vision at reentry surgery clearly indicate bone above the canal. Hence the presence of a physiologic microstrain environment causing an increase in bone volume is supported—similar to the development of the mandibular tori found on occasion in a parafunction patient. Potential for vertical bone growth after placement of a TMI implant has been reported in the literature.[27] It is speculated that tensile forces to the crest of the posterior mandible during occlusal forces on the implant is the reason for this finding. However, regeneration of bone in vertical height and volume following placement of a subperiosteal implant has not been previously documented.

CONCLUSION

The subperiosteal implant has been in use for over 50 years. It has undergone continuous evolution in design, technique, and materials. The tripodal subperiosteal of the mid 1980s addressed the concern of the dehiscent mental nerve bundles of the Misch/Judy Division D mandible. Mandibular bone growth has been reported with the use of the Bosker TMI implant. This case report observed bone growth on a patient with a 13-year-old subperiosteal implant—a finding that has not previously been reported.

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