Establishing and Maintaining Osseointegration Within the Functional Matrix

Martin Chin, DDS

Two common dentoalveolar defects are managed by a novel treatment approach. This article explains how the underlying mechanism that regulates bone physiology can be recruited to improve the response to surgical healing and presents the theoretical basis of this treatment method. Modern investigators have revealed the likely anatomic pathways through which this system operates. A specific set of rules can be defined to guide surgeons to design procedures that have the optimum potential for a successful outcome. These cases demonstrate that if this treatment method is followed, successful healing can be achieved.


This article presents a novel strategy for designing procedures for dentoalveolar surgery. The foundation of the treatment approach presented lies in the underlying processes behind the formation of facial bones in the embryo. These mechanisms are preserved in the adult as the method by which the skeleton is maintained and repaired. Scientists have postulated the existence of a system that creates facial bones and maintains them in a relative steady state, though the physiologic and anatomic basis of these processes has remained obscure. Modern investigators have now demonstrated the presence of an extensive fiber system within bone that likely serves as the scaffold upon which living structural bone forms and exists (Fig 1). The internal fiber system is itself a living system that is an intermediary component of a comprehensive regulatory process. This unrecognized system receives regulatory commands through a chain of anatomic structures, originating in the central nervous system. In the developing embryo, this network of fibers conducts signals into regions forming bone to affect the behavior of tissue systems and individual cells. In response to these specific instructions, cells alter their behavior to affect assembly of facial bones in situ. This same regulatory process maintains the morphology of bones

1Private Practice, San Francisco, California, USA.

Correspondence to: Martin Chin, Oral and Maxillofacial Surgery, 2100 Webster Street, Suite 303, San Francisco, CA 94115, USA. Fax: (415) 923-3845.
Email: mchin1@ix.netcom.com

©2016 by Quintessence Publishing Co Inc.
and participates in repair from injury or surgery in the adult. Understanding and incorporating these mechanisms into surgical design can extend the range of treatment possibilities and improve predictability. Surgical treatments that have failed with conventional procedures may be retreated with techniques that use these principles and reach successful outcomes.

Harvold pioneered the clinical application of preserved embryonic systems into surgical engineering. He developed a theory explaining the method by which embryos affect the self-assembly of their facial skeletons. Thoughtful study of embryos and children with craniofacial anomalies resulted in the conclusion that bone formation follows a specific set of rules. In order to form and maintain a skeletal unit, the environment into which the bone develops must include four essential components: (1) an anatomic volume sheltered from physical forces, (2) a source of bone-forming cells, (3) a source of neuromuscular input, and (4) an absence of pathology.

When initially conceived, this approach was an extrapolation of clinical and anatomic observations. The concept that bone healing at a surgical site could be directed by signals from the central nervous system was difficult to understand. For surgeons, the process lacked a clear physiologic and anatomic basis. Recent discoveries have improved our understanding of how this process might work. Aaron and Carter developed a technique to study undecalcified tissue that revealed the presence of a fiber network system within bone that interfaces with periosteum, muscle insertions, and probably periodontal ligaments. This network might be an unrecognized anatomic component of the functional matrix concept. Ingber demonstrated that individual cells receive mechanical signals from the environment and respond by altering their intracellular metabolism. The language by which the cells communicate along this pathway has yet to be deciphered.

**Clinical applications**

**Case 1**

An 80-year-old woman presented with a fractured implant at the maxillary left first molar site (Fig 2). The implant was removed with a trephine bur, resulting in a 6.5-mm-diameter defect that penetrated the sinus floor (Fig 3).

**Engineering the repair procedure**

The objective was to replace the implant in a single-stage surgery...
designed to meet the essential requirements of a bone-forming environment. In this surgery, an implant was suspended in the center of the defect and secured with a titanium mesh bone plate, thus establishing a stabilized zone within the osteotomy defect (Fig 4). The implant selected as the replacement had a larger diameter than the original but was still much narrower than the osteotomy. Neuromuscular regulation of this region is maintained under the primary direction of the periodontal ligament system of the premolar but also indirectly influenced by the major muscle systems maintaining the piriform buttress. The penetrating Sharpey fibers from the alveolar mucosa and the sinus membrane also participate in this process. Bone-forming cells must be delivered into the gap between the osteotomy walls and the implant cylinder. In this particular case, the amount of native bone at the site was sufficient; no additional cells were necessary. Antibiotics and primary closure of the gingiva were the strategies applied to minimize the risk of infection-related pathosis.

**Surgery**

When the implant was placed into the trephined explant site, it was clear that there was no contact between the bone and the cylinder (Fig 5). There was also no support from the sinus floor, which was removed along with the fractured implant. Therefore, a titanium mesh bone plate was attached to the implant by the cover screw. Small titanium screws were used to secure the mesh and implant (Fig 6). When the site was opened 10 weeks later, removal of the bone plate revealed the implant fully invested in normally appearing bone (Fig 7). The replacement implant had become fully stable and covered in newly generated bone. A radiograph taken at 8 months after surgery with the crowns in place shows the amount of bone surrounding the implant (Fig 8). The regenerated sinus floor had formed immediately coronal to the hemispherical implant apex.

**Discussion**

The ability to regenerate, from bone precursor cells, a skeletal unit into which a dental implant simultaneously osseointegrates represents a departure from conventional practice. This case demonstrates the feasibility of reconstructing alveolar deficiencies and establishing osseointegration of dental implants through design and assembly of bone-forming environments.

Bone biologists demonstrated the role of embedded Sharpey fibers in the healing of cylindrical osteotomies in a lamb model. Their study showed that healing is initiated by Sharpey fibers extending into the void from the osteotomy walls. Trabecular bone formed in response to the fiber scaffold (Fig 9).

This case shows that human response to cylindrical osteotomies for dental implants likely follows the same healing process. In this patient, the bone involved in the osseointegration interface was newly formed. The osteotomy void filled with bone following the same process revealed in the lamb model.
As a result, the implant became embedded in newly formed bone and an osseointegrated interface was established. The age of the patient did not compromise the healing process. In this treatment approach, the forces that regulate the patient’s existing bone morphology are deliberately focused on the healing of the reconstruction. These same forces promote integration of the reconstruction.

Fig 5 (left)  There is no bone contact with the implant cylinder.

Fig 6 (right) Two small screws affix the bone plate to the alveolar bone.

Fig 7 (left)  Following removal of the bone plate, bone can be seen around the polished implant collar; a result of remodeling. The implant is stable and fully invested in bone.

Fig 8 (right) Radiograph 8 months after implant replacement shows bone surrounding the cylinder. A new sinus floor has formed.

Fig 9  Sharpey fibers extend into the osteotomy establishing a fiber network upon which trabecular bone forms and organizes.
into the patient’s physiology. The result is a process that not only can improve the predictability of dentoalveolar reconstruction but also can be applied to treat disorders that are beyond the capability of conventional surgery.

Creation of osseointegration through this method raises questions as to the nature of the bone-implant interface. For this technique to succeed, the predilection of bone to establish and maintain a close relationship with implants can only result from forces that regulate the morphology and organization of a skeletal unit. The relationship of the bone to the implant surface is secondary.

Case 2

A 30-year-old woman presented for a second opinion in the treatment of a missing maxillary left canine and a large alveolar defect (Fig 10). She had been undergoing dental implant therapy to replace the missing tooth. Her dental history included four bone graft procedures performed by multiple oral surgeons, each of which resulted in failure. At the time of her evaluation, the maxillary left lateral incisor demonstrated Class II mobility and was stabilized by an orthodontic appliance. Another surgeon from whom she sought an opinion had recommended a fifth bone graft surgery and warned her that removal of the lateral incisor might be necessary as part of the procedure.

A radiographic view of the site shows a cleft-like alveolar deformity extending vertically to the level of the apex of the lateral incisor (Fig 11).

The labial and palatal cortical plates have been lost. The lateral incisor root is exposed into the defect, and moderate bone loss has occurred on the mesial aspect of the first premolar. The gingiva overlying the site is scarred and deficient.

**Engineering and design before surgery**

This asymmetrical defect was challenging to repair because the mesial aspect contained a tooth with substantial periodontal damage (Fig 12). The key was to design a bone-forming construct that both replaced the missing tooth and corrected the periodontal defect. A stable result could not be achieved unless the periodontal defect was corrected as well.

The first step in the evaluation process was to identify the forces working to regulate and maintain the existing deformity. The bone covering the mesial aspect of the first premolar was under regulation of the tooth’s periodontal ligament system. Sharpey fibers, which function as an extension of periodontal ligaments, penetrated the lamina dura and then continued into the thin layer of bone covering the root. The bone at the anterior aspect of the defect was under the regulation of the lateral incisor and its damaged periodontal ligament system. Bone loss extended all the way to the root apex because the periodontal system had been damaged on both the distal and facial root surfaces. A conventional bone graft
placed into this defect would not meet the essential requirements of a bone-forming environment because the system that would support its success had been compromised. Even if an adequate number of bone precursor cells could be delivered to the defect, the other components essential for bone formation were lacking, and the reconstruction would fail. The center of the defect could not be mechanically stabilized because of the loss of the labial and palatal bone plates. Distance prevented neuromuscular regulation by the periodontal ligament systems from reaching the center of the defect. There was also no signaling source at the ridge crest adjacent to the lateral incisor, where the periodontal ligament system had been damaged. The shortage of viable soft tissue challenged efforts to establish a nontension closure, predisposing the wound site to breakdown and bacterial incursion.

In summary, failure was the likely outcome if a fifth bone graft was undertaken with no attempt to alter the environment. Nonetheless, extraction of the lateral incisor would not improve the conditions because it would increase the size of the defect and the available sources of neuromuscular regulation might not reach the center.

**Design of bone-forming environment**

In this case, a segmental osteotomy fragment could be transported into the defect to establish two bone-forming chambers (Fig 13). The periodontal ligament system of the first premolar could then be repositioned into close proximity to the damaged lateral incisor so that the available bone-forming regulation potential could be focused into the site of greatest need. To regenerate the alveolar bone and periodontal ligament, a source of cellular regulation was essential. The bone-forming chamber to be established between the lateral incisor root and the first premolar root would have the potential for regeneration. Transport of the segment would collapse the defect volume into a narrow gap (Fig 14) and create a stabilized space. Bone precursor cells would be delivered to the site using recombinant human bone morphogenetic protein-2 (rhBMP-2) carried on collagen sponges.

Neuromuscular regulation would be administered by the periodontal ligament system of the first premolar. As the bone overlying the mesial aspect of the first premolar began to demineralize in response to the rhBMP-2 device, its embedded Sharpey fibers would become directly exposed to the bone-forming chamber. Sharpey fibers are not affected by the demineralization process, making them an ideal vector to deliver regulatory signals to this environment, provided the site is isolated from bacterial incursion. When transportation of the segment was completed, it would carry 9 mm of gingiva into the site, which would be used to establish a nontension closure. Rapid healing of the soft tissue would establish a barrier to exclude oral and nasal bacteria from the regeneration chamber.

The second bone-forming chamber created by movement of the segmental osteotomy, at the pterygomaxillary junction, is also ideal for bone formation because it receives powerful neuromuscular signaling from the muscles originating from the pterygoid plates. This
chamber consolidates rapidly and therefore does not require additional bone precursor cells.

Surgery
Surgical findings revealed multiple anatomic challenges (Fig 15). The ridge was narrow and vertically deficient. Bone loss on the lateral incisor extended apically on the distal root surface nearly to the apex. The labial aspect of the lateral incisor root was exposed at the coronal half. The first premolar had moderate bone loss at the distal aspect and substantial bone loss on the labial aspect.

A standard segmental osteotomy commonly used in orthognathic surgery mobilized the maxillary left posterior quadrant. The edentulous portion was resected. Protraction of the osteotomy segment transported the first premolar into the reconstruction site. In this surgical design, the periodontal ligament system of the first premolar was the primary source of neuromuscular signaling into the bone-forming chamber. Positioning of this critical element of the bone-forming system is important. To be most effective, the Sharpey fiber network housed in the alveolar bone surrounding the premolar must be positioned for optimal access to the bone-forming chamber. This required careful adjustment of the vertical osteotomy for close positioning of the roots of the lateral incisor and the premolar. In addition, the segment was displaced laterally and fixated with a titanium bone plate (Fig 16), placing the damaged lateral incisor root in a relative cavity that allowed the premolar periodontal network to extend its signaling into a graft volume labial to the lateral incisor root surface.

A collagen sponge carrying 2 mg of rhBMP-2 was positioned in the constructed bone-forming chamber (Fig 17). A second collagen sponge containing 2 mg of rhBMP-2 was placed over the lateral maxillary osteotomy. The pterygomaxillary bone-forming chamber did not receive any grafting material.

The area between the premolar root and the damaged portion of the lateral incisor had the most critical need of regeneration. The morphogenetic protein increased the population of bone precursor cells in the regeneration chamber. In response, bone covering the mesial aspect of the first premolar began to demineralize, and in the process exposed the Sharpey fibers of the premolar periodontal ligament system. These fibers became surrounded by new bone precursor cells. According to the working theory of bone-forming environments, mechanical signaling from Sharpey
fibers regulates the behavior of bone-forming cells. If effective, the bone-forming construct would form a new skeletal unit that becomes integrated into the local physiology.

A radiograph of the site taken 4 months after surgery shows calcified tissue filling the space between the roots (Fig 18). The consolidating graft visibly extends coronally beyond the native bone covering the mesial surface of the premolar root. There is no evidence of a new lamina dura at this time.

As bone formed in the constructed environment, it had to interact with the damaged root surface of the lateral incisor. Histologic analysis was needed to clarify what was occurring between the root surface and bone graft.

The radiographic appearance at the 4-year follow-up reveals that substantial repair had occurred (Fig 19). There was now a well-defined lamina dura, suggesting the presence of Sharpey fibers extending from a periodontal ligament system. The bone between the roots had developed normal-appearing trabecular architecture. The alveolar crest remained blunted, but this might be improved by paralleling the roots. However, the patient and orthodontist elected to remove the appliance and progress into orthodontic retention.

At the 4-year follow-up (Fig 20), the lateral incisor was favorably positioned and was now immobile. There was no need for prosthetic therapy to replace the missing canine as this was a complete arch of natural teeth.

Discussion

This case illustrates the value of following a deliberate diagnostic protocol before initiating surgical treatment. Given the clinical presentation of a missing canine and deficient bone volume, treatment planning a bone graft might seem reasonable. The complete diagnosis, however, must extend beyond the simple recognition of the missing structures. The diagnosis must begin by understanding the physiology that is operating to create and maintain the deformity. This information is then used to design an environment that will support healing and long-term stability. When analyzing the feasibility of a bone graft to treat the existing defect, it should become clear that the existing sources of neuromuscular regulation are too distant from the regeneration site to exert a positive regulatory effect. Successful healing is therefore unlikely, and an alternative strategy should be considered. Currently, introducing an artificial

Fig 18 (left) Four months after surgery, a radio-opacity partially fills the regeneration chamber. There is no clear sign of a lamina dura adjacent to the lateral incisor distal root surface.

Fig 19 (right) Forty months after surgery, the radiograph shows signs of functional regeneration. There is a distinct lamina dura adjacent to the lateral incisor root. The bone filling the regeneration chamber has assumed a normal trabecular appearance.

Fig 20 The patient has a continuous dental arch and stable occlusion. The lateral incisor is stable.
source of neuromuscular signaling is beyond our capability. In this case, an osteotomy became part of the surgical plan to address this challenge. It was necessary to position the available neuromuscular system so it could be focused on the regeneration site.

If the lateral incisor is expected to function long term, the damage to its periodontal ligament system resulting from prior bone-grafting attempts need to be corrected. Simply surrounding the root with calcified material would not represent functional regeneration. If regeneration was successful, the site would remodel and organize to develop a normal structural architecture. Using the osteotomy to position the operating periodontal ligament system of the premolar into close proximity to the damaged lateral incisor could allow recruitment of the regulatory ability to the advantage of the regeneration process. The follow-up radiograph taken 4 years after surgery shows development of a lamina dura at the site of prior bone loss, which suggests that a functioning periodontal system had been restored. The association of cortical bone formation and presence of an embedded Sharpey fiber system is based on recent work by bone biologists.1 Using immunohistochemistry and special cryomicrotomy, investigators have shown that Sharpey fibers penetrate the full thickness of the cortical bone and continue into the medullary space. These studies have demonstrated that the boundary between the cortex and the medullary space is characterized by a change in fiber chemistry. This suggests that formation of cortical bone, including the lamina dura, is related to the specific structure and chemistry of the Sharpey fibers contained within. Replacement of the lost lamina dura in this case suggests that a Sharpey fiber system has been restored as well.

Conclusions
Successful healing of the two cases presented contradicts the outcomes predicted by conventional concepts. Study of these result anomalies is important for two reasons. First, we must recognize that our existing understanding is incomplete and that searching for better explanations is our responsibility as scientists. Second, improving our appreciation of how biologic systems work opens the door to new and improved therapies. Safer and more efficient treatment is the objective.

This article describes surgical approaches for two disorders that commonly present to clinical practice. Based on the pattern of human embryonic development, a set of essential parameters for successful bone growth is outlined. These parameters are the theoretical basis for designing a deliberate and consistent protocol to plan and perform maxillofacial procedures. Analyzing prospective treatment plans with these parameters in mind can reveal pitfalls and alert the surgeon to situations that may be predisposed to failure. The two clinical cases presented were drawn from clinical experience that spans 35 years, and the outcomes are typical of many cases treated in this manner. Questioning the accepted biology of surgical practices that we perform every day may be uncomfortable, but the potential benefit is great. Refining and extending our understanding of these procedures can yield many new methods to improve treatment capabilities.

Acknowledgments
This article is adapted from the author’s forthcoming book, Surgical Design for Dental Reconstruction with Implants: A New Paradigm (Quintessence Publishing, 2015). The author reported no conflicts of interest related to this study.

References