Management of atrophic mandible fractures has been a challenge for maxillofacial surgeons for years. Techniques that have been used are splints, external pins, split ribs wire osteosynthesis, and rigid fixation. Results with each of these techniques have had variable success depending on the amount of atrophy that the patient underwent before injury. There has been an evolution of treatment over the years in which closed techniques have been gradually replaced by open techniques. In 1976, Bruce and Strachan suggested that closed reductions should be tried first and that if an open reduction was required, wire mesh was preferable to a bone plate. In 1979, Marciani and Hill, after reviewing 33 well-documented cases, recommended that a closed reduction of fractures of the atrophic mandibular body be done. In the second Chalmers J. Lyons Academy study, published in 1993, Bruce and Ellis noted that 81.5% of the 104 patients had an open reduction and internal fixation with a bone plate. In 1996, Luhr et al presented their results in 84 patients with atrophic edentulous mandible fractures treated with compression bone plates. In 2006, Wittwer et al reviewed their outcomes of the treatment of 30 patients treated with different plating systems. They concluded that the more atrophic a fractured mandible is, the more rigid the fixation of the fracture needed to be. Tiwana et al, in 2009, suggested that for ideal healing of edentulous, atrophic mandible fractures, bone grafting is needed in addition to a large reconstruction plate and a bone graft.

The patient population with atrophic mandible fractures is different in several ways from the average individuals who present for management of traumatic injuries. In general, they are an older population with concomitant medical issues. Bruce and Ellis noted that their patients had an assortment of medical illnesses including cardiovascular disease and chronic respiratory disease. Injuries tend to be more evenly distributed between genders, and falls tend to account for a higher percentage of the cause of these injuries. Long periods of edentulism lead to atrophy of the mandible in height and width, particularly in the body region of the mandible. As a consequence, there is an increased incidence of unilateral and bilateral body fractures, as this area of the mandible becomes more susceptible to fracture. Eyrich et al had 34 patients with atrophic mandible fractures ranging in age from 38 to 89 years with a mean age of 70.5 years. Of their patients, 26% were female and 74% were male. Wittwer et al noted that the cause of the injury in 23 of their 30 patients was a fall. Their age group was 42 to 91 years with a mean of 72 ± 13.8 years. Bruce and Strachan noted that falls accounted for 30% of the injuries that they saw.

Nonunion and fibrous union were and are a well-recognized complication when treating these patients. Bruce and Strachan stated that there was a 20% incidence of nonunion after treatment of these types of fractures. Later studies with more aggressive techniques have shown the incidence of nonunion and malunion to be approximately 10%. Luhr et al developed a classification of atrophy based on the height of the mandible in the body region, with a Class I atrophy being 16 to 20 mm, a Class II atrophy 11 to 15 mm, and a Class III atrophy being 10 mm or less. The patients with Class III atrophy have the greatest incidence of nonunion. In 1974, Boyne and Upham presented the successful treatment of 12 cases of nonunions after atrophic mandible fractures. All were treated with autogenous marrow and titanium mesh. In 1976, Bruce and Strahan suggested that bone grafts should be considered when treating an edentulous mandible fracture unless the patient was medically compromised. Six of the 25 patients treated by Luhr et al had primary bone grafts placed as part of the treatment of their atrophic mandible fractures. The rationale for primarily grafting a mandib-
ular fracture with less than 10 mm of height is that a corticocancellous bone graft would augment the osteogenic potential of the atrophic bone and would speed healing. That rationale is still accepted today by many surgeons. That it may speed healing by transplanting live bone marrow cells and allowing osseoinduction and osseoconduction is not debatable. However, the question remains: without functional stimulation, how long does the augmented bone remain? In addition, what are the consequences associated with the morbidity of the second surgical site?

In the early 1970s and 1980s, augmentation bone graphs were routinely obtained to facilitate prosthetic management of patients with severe atrophy. Baker et al followed 22 patients 4 to 10 years after autologous onlay rib grafts. With a mean follow-up of 6.2 years, these investigators found that most of these patients had returned to the baseline amount of bone. In an elderly patient with significant medical comorbidities who may have an unsteady gait, harvesting either an anterior iliac graph or a tibial bone graft has the potential to significantly affect the patient’s function. However, there are possible alternatives to an autogenous graft available.

As the cross-sectional area of the mandible decreases, the amount of internal buttressing decreases, and there is less resistance to the opposing muscle groups of the mandible. As the load-sharing capacity is decreased, the plate used to counteract the stresses and strains placed on the mandible needs to be larger or load bearing. Unfortunately, the plate may be bigger than the cross-sectional area of the lateral surface of the mandible where it is traditionally placed. In 2006, Madsen and Haug published an article with a biomechanical focus comparing the placement of a reconstruction plate placed along the inferior border of the mandible versus one placed along the lateral border of the mandible for simulated atrophic mandibles. In this study, there were no significant differences between the 2 groups. The clinical application of inferior border plating has been published more recently.

Although placing a plate at the inferior border of the mandible challenges the traditional concepts of placement of plates for fractures, it appears to work for these complex cases. For the last 4 years, our group has been routinely placing reconstruction plates at the inferior border of the mandible without the use of bone grafts for both unilateral and bilateral Classes II and III edentulous body fractures. These fractures are approached from a submandibular incision or an apron incision when the fractures are bilateral. The plates have been prebent before arriving in the operating room. Six and 8 mm screws are placed in the symphysis and angle region, avoiding perforation into the oral cavity (Figs 1, 2). Recently, in a patient with an extremely atrophic mandibular fracture, recombinant bone morphogenic protein (rhBMP, off-label use) was placed in addition to the inferior border plate to augment osteogenic potential of the remaining mandible (Figs 3-7). The patient’s mechanism of injury was a fall, and she had several comorbidities. There was family concern regarding the morbidity of a second surgical site. At 2 months after her injury, however, she was able to return to her part-time job.
Discussion

Patients with atrophic mandibular fractures in general are an older population who differ from routine fracture patients in several ways. They have both physiologic and anatomic differences resulting from the aging process and loss of masticatory forces. Although traditional techniques have suggested that closed management of these patients’ fractures was the best choice, we do not share these beliefs. Smaller plating systems do not allow the proper buttressing to allow function and healing. We agree with the paper by Tiwana et al that large load-bearing plates are the best choice.6 Large load-bearing plates do allow healing of both the fracture and function for the patient. The plates can be positioned at either the lateral aspect of the mandible or at the inferior border with good success. Although technically more challenging to bend and place at the inferior border, these plates will not interfere with subsequent denture use. Few of our patients with this degree of atrophy and medical comorbidities go on to have dental implants placed after repair of their fractures; therefore, we question the routine use of bone grafts for bulk for subsequent implant placement.

When the mandible approaches 5 mm or less, the osteogenic potential is markedly decreased. In these patients and in those with continuity defects, we believe that additional osteogenic induction is needed. Several recent studies have been published using recombinant bone morphogenic protein (rh-BMP-2) in humans. At this point, its use for many of our common facial bone defects is off label.17,18 The consensus appears to be that it may be indicated for several problems seen in the facial region. It has been the authors’ experience that when rh-BMP is used, there is pronounced swelling in the region. The swelling should be anticipated and a plan made to deal with it. The use of rh-BMP in our case was driven by the minimal osteogenic potential in a mandible that was less than 5 mm thick and by the possible morbidity from a donor site that may affect the patient’s gait.
A 2007 Cochrane database review on the management of fractured edentulous atrophic mandibles suggested that there was inadequate evidence for the effectiveness of a single approach either closed or open for these types of fractures. It suggested that until a high level of evidence is available, treatment decisions should continue to be based on the clinician’s prior experience. In our group, patients with Class II and Class III atrophy are treated with large reconstruction bone plates placed at the inferior border of the mandible without a bone graft. In cases with exceptional atrophy or a continuity defect, rhBMP may be an acceptable alternative that does not carry the morbidity of a second surgical site. Although our series is not as long or as large as the group from Louisville, to date we have not had a nonunion with our approach in these very atrophic mandible fractures.

References