Hourglass Maxillary Collapse in Repaired Postalveolar Clefts of the Palate*

GEORGE F. CRIKELAIR, M.D.
RAPHAEL PRICE, M.D.
BARD COSMAN, M.D.

The patient with a repaired complete cleft lip and palate routinely exhibits some degree of alveolar arch asymmetry and maxillary deformity. It is not usually recognized, however, that the patient with only a postalveolar palate cleft may also present a maxillary abnormality. The purpose of this paper is to draw attention to this defect in view of its characteristic appearance, its clear association with surgery, and the possibility that it may be avoided by simple surgical caution.

Material and Cases

The postalveolar cleft palates operated upon at the Columbia-Presbyterian Medical Center from 1958 to 1968 were reviewed. Two patients demonstrated a significant deformity of the alveolar arch.

Case 1. This 17 month old child was operated upon for the closure of a postalveolar cleft palate. No cleft of the primary palate was present. The edges of the cleft were pared, releasing incisions were made around the alveolar rim laterally, and mucoperiosteal flaps were elevated (von Langenbeck technique). The hamular processes were fractured at their bases, the greater palatine neurovascular bundles were preserved, and the palate was sutured in layers medially leaving the relaxing incisions open. Healing was uneventful. Speech development was good. However, at age 10, dental models demonstrated an “hourglass” maxillary collapse without apparent anterior-posterior shortening (Figure 1 A).

Fig. 2. This child was operated upon at age 4 at which time a push-back procedure was done closing the anterior portion of a wide horseshoe type postalveolar palate defect. At age 6 a von Langenbeck procedure closed the posterior palate. Small turn-over palate flaps were used at ages 8 and 9 to close a palato-nasal fistula. Dental models at age 21 showed severe “hourglass” deformity (Figure 1 B).

Discussion

Maxillary deformities in the repaired cleft lip and palate patient occur in four main categories (Figure 2). One is medial collapse of the cleft segment of the alveolus. This is ultimately manifest as a maxillary cross-
bite. A second is anterior-posterior shortening of the maxilla. This results in maxillary retrognathism or relative mandibular prognathism. A third is decrease in inferior-superior height of the maxilla on the cleft side. This is seen in the tilting up of the cleft segment off the plane of occlusion, usually most notably near the cleft itself. Fourthly, there is buckling inward, or hourglass deformity of the lateral portions of the alveolar arch. It is this last defect which may also be seen in the patient with a repaired post-alveolar cleft palate.

The degree to which lip or palate surgery or the congenital defect itself is responsible for the first of these maxillary deformities is moot (I). There is no question that some unoperated cleft lip and palate patients already show evidence of cleft segment collapse. Some others exhibit rapid collapse following cleft lip repair and prior to palate repair. However, with better lip surgery the incidence of maxillary cross bite has been reduced and some authorities report only a third of patients to show this

FIGURE 1. (A) Maxillary arch above and mandibular arch below. Medial collapse is noted in the region of the upper first and second premolars. The collapse is slightly greater on the right side than on the left. (B) Severe hourglass deformity is noted in the maxillary arch with constriction most pronounced in the region of the cuspid, first and second premolar teeth. Mandibular arch seems slightly narrowed also.
defect in any significant degree (1). Better surgery and the abandonment of traumatic methods have been credited with the reduction in numbers of patients showing the stigmata of anterior-posterior maxillary shortening noted by Graber (2). A majority of patients continue to show some degree of the tilting effect even when arch form is excellent. But it is to the last form of maxillary defect, that of the hourglass deformity which may occur in both complete palate clefts and postalveolar clefts, to which our attention is now directed.

This abnormality was first emphasized by Foster who termed it “buckling collapse” (3). He demonstrated its appearance in 11 of 102 complete unilateral cleft lip and palate repairs, 4 of 47 bilateral complete clefts and 7 of 18 postalveolar cleft palates without cleft lip. It is into this last group that our patients fit. The hourglass configuration is, of all the maxillary defects, the most clearly related to surgery. No unoperated patients have been reported showing this deformity (4). Yet it is difficult to understand its cause. If operative scar contracture were “binding” the palate as is suggested by the thin-waisted, corseted appearance, then one would expect the deformity to be more common since scarring is the normal concomitant of the procedure. But the hourglass collapse is so uncommon that most authors neglect to mention any possibility of maxillary collapse in the postalveolar cleft palate patient. And it was so uncommon in our experience that the two cases presented were initially thought to be unique. Further, if scar contracture alone were the cause, one would expect the effect to be most marked on the cleft segment of a unilateral complete cleft palate. However, as Foster pointed out, when the deformity was noted in a unilateral cleft lip and palate patient, the buckling was bilateral and essentially equal on both the cleft and non-cleft segment (3). And last, if scar contracture were the sole explanation one would expect the deformity to be limited to those patients with multiple palatal procedures rather than occurring, as in Case 1, in patients with but a single, seemingly uncomplicated operative intervention.

Recent experimental work may give a better basis for understanding the mechanism of this deformity. Using dogs, Kremenak, et al. elegantly expanding on initial work by Herfert, demonstrated striking decreases in transverse maxillary diameter when a mucoperiosteal flap was raised 1 mm. lingual to the teeth and a 4 mm. strip of the elevated tissue was removed from the flap edge before it was replaced (5, 6, 7, 8). This experimental design is the equivalent of a mucoperiosteal palate flap procedure which leaves the releasing incision area bare. As shown by these authors, the maxillary growth defect produced is not dependent on the elevation of the flap alone, nor upon the sacrifice of the palatine nerves and vessels, but upon the denudation of the palatal shelf bone just medial to the teeth.

While the transfer of animal results to humans is hazardous, the applicability of these findings to the hourglass deformity seems real. That not
FIGURE 2. Categories of Maxillary Deformity. (A) Medial collapse of the cleft segment in a complete unilateral cleft palate. (B) Anterior-posterior maxillary shortening and constriction. (C) Inferior-superior shortening of the maxilla on the cleft side with upward tilting most notable at the cleft itself. (D) Hourglass or buckling defect with medial collapse of lateral alveolar ridges.
all palate closures are followed by the buckling defect may be explained by the hypothesis that so close an approach to the tooth line by usual releasing incisions is rare and that denudation to be deleterious must be close to the alveolar ridge. The occasional case in which the surgeon does denude this critical area would then be the case to show transverse growth arrest, i.e., buckling; since this would be done bilaterally, the symmetry of the effect in the unilateral complete cleft palate may be explained. If these hypotheses are valid, the elimination of releasing incisions or their careful placement well away from the tooth line so as to avoid denudation of the palatal shelf just medial to the alveolar process may obviate this form of maxillary collapse.

Summary

Alveolar arch buckling or hourglass deformity is a type of maxillary collapse occasionally seen following the repair of both complete and post alveolar cleft palates. It is the only form of alveolar arch deformity found in the postalveolar cleft palate patient. The defect is clearly related to surgery. The mechanism of its cause is uncertain but may be a denuding injury to the palatal bone shelf immediately adjacent to the tooth line rather than operative site scar contracture. Elimination of releasing incisions or their placement further away from the alveolar ridge is suggested as a potential means of preventing this form of maxillary collapse.

References