Anteriorization of inferior oblique muscle and downward transposition of medial rectus muscle for lost inferior rectus muscle

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A 6-year-old boy who had been treated with bilateral medial rectus muscle recessions 3 years earlier for congenital esotropia was undergoing bilateral inferior oblique muscle recessions to correct inferior oblique muscle overaction. The right inferior rectus muscle was inadvertently cut during this surgery and was irretrievable. To manage this complication, the medial rectus muscle was transposed toward the inferior rectus insertion and the inferior oblique muscle was anteriorized. At the 1 year follow-up visit, no infraduction deficit was present on downgaze and only 8^{Δ} of left hypertropia was present in primary position.

A lost muscle is one of the most devastating complications of strabismus surgery. If not treated, a lost muscle will lead to consecutive strabismus, diplopia, and a large limitation of eye movement in the direction of the lost muscle. A lost inferior rectus (IR) muscle is very rare. Parks[1] reported only one case of a lost IR muscle during 20 years. When it occurs, intraoperative retrieval of the muscle may be unsuccessful, and alternative treatment strategies often are necessary to minimize the remaining deficit. Several surgical procedures have been described to treat a lost IR muscle; however, they resulted in limitations in ocular movements, marked deviations, or diplopia.[1-7] The risk that these procedures might cause anterior segment ischemia also raises some concerns.[5-7] Considering these potential outcomes, we describe how we transposed both the inferior oblique (IO) muscle and the medial rectus muscle to treat a lost IR muscle.

Case Report

A 6-year-old boy who had previously been treated with bilateral medial rectus muscle recessions for congenital esotropia at 3 years of age was referred to the strabismus clinic. Preoperative examination revealed an uncorrected visual acuity of 20/20 in both eyes and a cycloplegic refraction of +0.50 diopters in both eyes. Ocular motility examination revealed orthotropia in primary position, 15^{Δ} of exotropia in upgaze, 10^{Δ} of esotropia in downgaze, and approximately 3+ IO overaction in both eyes (Figure 1A and B). Fundus examination was unremarkable. A surgical procedure was planned with the goal of collapsing the V pattern and weakening the overacting IO muscles.

At surgery, an inferotemporal conjunctival incision was placed 8 mm from the limbus to isolate the left IO muscle. The IO muscle was disinserted and reattached, bunching the muscle at a point 1 to 2 mm temporal to the insertion of the IR muscle. On the right side, however, during IO muscle isolation, the IR muscle was cut and crushed unintentionally in its mid-portion. Although the stump of the IR muscle was present at the attachment site, an attempt to retrieve the proximal portion of the muscle was unsuccessful. The IO muscle was then isolated and reattached, bunching the muscle 1 mm anterior and temporal to the lateral corner of the anatomic insertion of the IR muscle. The previously recessed medial rectus muscle was then disinserted and transferred downward by a full tendon width to the medial corner of the anatomic insertion of the IR muscle.

One year after the operation, ocular motility was within normal limits in right, left and downgaze, and no infraduction deficit was noted. Prism and cover testing revealed 8^{Δ} of left hypertropia in the primary position, 3^{Δ} of left hypertropia in 30° downgaze, and 25^{Δ} of left hypertropia in 30° upgaze (Figure 1C andD).

Discussion

A lost extraocular muscle can occur during either strabismus or vitreoretinal surgery or after trauma.[1] A lost IR muscle is very rare, and its prevalence varies in different types of muscle surgery.[1] In many cases, however, retrieval of the IR muscle is impossible, and alternative methods are necessary to minimize the damage caused. Congenital absence of IR muscle is similar to acquired loss of IR muscle. A corrective procedure for congenital absence of IR muscle was published in 1914 by McDannald.[8] He performed a tenotomy of the superior rectus muscle and a downward transposition of the half-tendon of the horizontal recti muscles to the presumed IR muscle insertion. Casten[9] transposed the lower half of the horizontal rectus muscles and performed a myotomy of the IO muscle. The final outcome was reported to be successful. Giller[10] reported using the same procedure, but his patient was left with a restriction in infraduction. Cooper et al[11] transposed the full tendon of the horizontal rectus muscles to the pre-

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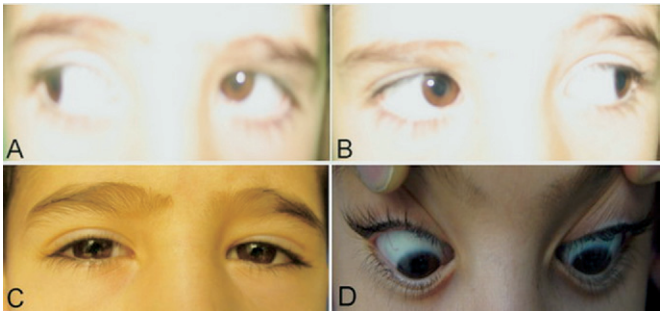

FIG 1. A and B show marked preoperative inferior oblique muscle overaction. C and D show postoperative examination in primary and reading positions.

sumed IR insertion, which also left the patient with a deficit in infraduction. Taylor and Kraft[12] treated aplasia of the IR muscle with a full tendon transposition of the horizontal rectus muscles, a mirror image of the Knapp procedure, which resulted in a small hypotropia in primary position. Paysse et al[3] treated 2 patients who had traumatic rupture of the IR muscle. Both patients underwent an inferior transposition of the inferior halves of the medial and lateral rectus muscles without disinsertion (modified Jensen transposition procedure). Both patients had a persistent small overcorrection in primary gaze position. Some authors[4-7] have reported treatment of lost IR muscles by anteriorization of the IO muscle alone without transposition of the horizontal rectus muscles. These patients were left with limitation in upgaze[4,6] and downgaze[4-7] and diplopia in primary position.[4,5] One common result of these treatments was a marked increase in the deviation in downgaze.[4-7] We suspect that transposing the medial rectus muscle in addition to anteriorization of IO muscle may have improved the infraduction movement of the eye, which decreased the resultant diplopia in reading position, which is specially important in a school-age child.

In our patient, limitation of downgaze was not present after the operation. It is not fully understood whether this was caused by downward transposition of the medial rectus muscle or anteriorization of the IO muscle. Downward transposition of the medial rectus muscle did not produce any adduction deficit.

Anteriorization of the IO muscle was first reported by Elliot and Nankin[13] based on the principles described by Scott. This technique was used to treat overacting IO muscles, dissociated vertical deviation, and superior oblique paresis. In our patient, the IO muscle was bunched temporal to the IR muscle insertion. One of the complications of this procedure is the limitation of upgaze in abduction, described as the antielevation syndrome by Kushner.[14,15] In the present case, the medial rectus muscle was attached to the nasal part of the previous IR muscle insertion, but instead of transposition of the lateral rectus muscle, the IO muscle was attached to the temporal part of the previous IR muscle insertion. This procedure decreased the likelihood of anterior segment ischemia and improved downward movement without marked limitation.

It should be noted that, in our patient, the IO muscles were operated bilaterally, resulting in motor fusion and adaptation. Unilateral surgery may not produce the desirable results attained in this case. However, this technique appears to have a role in cases where surgery on 3 rectus muscles is not desirable. To the best of our knowledge, this is the first published case of a lost IR that was treated with anteriorization of IO muscle and downward transposition of the medial rectus muscle with acceptable results in primary and reading positions.

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