

Unilateral Anterior Transposition of the Inferior Oblique Muscle for Correction of Hypertropia in Primary Position

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Purpose: To evaluate the correction of hypertropia in primary position with unilateral inferior oblique (IO) anterior transposition (IOAT). **Methods:** Ten patients with idiopathic (nonparalytic, restrictive, or dissociated vertical deviation) hypertropia with marked IO overaction, who underwent unilateral IOAT, were prospectively evaluated to observe the correction of the hypertropia in primary position. No previous ocular muscle surgery had been performed. Four patients had esotropia and two had exotropia. In addition to the proposed surgery, horizontal procedures were performed to correct horizontal deviation, but no vertical transposition of horizontal muscles was done. Four patients had hypertropia and IO overaction, without horizontal strabismus, and IOAT was the only procedure performed. The IO muscle was reinserted 1 mm laterally to the lateral extremity of the inferior rectus muscle insertion using only one suture. The statistical analysis was performed by Wilcoxon rank sum test. **Results:** The mean absolute correction in primary position was 18.1 prism diopters (PD) (range, 4 to 33), directly proportional to the size of the hypertropia before surgery. Nine of the 10 patients had a residual vertical deviation of ≤ 6 PD. After surgery, 4 patients (40%) presented limited elevation in adduction (-2) in the field of the operated IO, presumably caused by the antielevator effect of the transposed muscle, which did not improve during the follow-up period (range, 2 to 79 months). **Conclusion:** Unilateral IOAT is an effective technique for correction of large hypertropia associated with marked unilateral IO overaction. Some lower lid curvature deformity and some limitation of elevation were observed in forced upgaze in some patients, but this was of no cosmetic importance. (J AAPOS 2003;7:241–243)

Hypertropia in primary position may be associated with ipsilateral inferior oblique (IO) overaction.¹ In most cases, unilateral weakening of the IO is performed to reequilibrate forces in cases of unilateral superior oblique palsy or for correcting idiopathic (nonparalytic, restrictive) hypertropia. Raab and Costenbader¹ showed that after unilateral weakening of IO, there is a risk of developing a contralateral IO overaction or an increase in preexisting IO overaction. Goldchmit et al.² observed that unilateral recession of the IO produced a mean correction of hypertropia of $8.4 \text{ PD} \pm 4 \text{ PD}$ in 19 patients with essential hypertropia in primary position associated with primary IO overaction (mean hypertropia, $10.3 \text{ PD} \pm 3.2 \text{ PD}$).

Gonzalez and Cinciripini,³ Bremer et al,⁴ and Mims⁵ performed unilateral IOAT in three patients with superior

oblique palsy. The former investigators' patients had good results, but the other investigators reported marked overcorrections (hypotropia of the operated eye), suggesting that unilateral surgery be avoided.

No other study evaluating the magnitude of correction of idiopathic hypertropia in primary position with unilateral IOAT was found in the literature. The present study aimed to evaluate prospectively the effectiveness of this technique for correction of hypertropia in primary position.

METHODS AND MATERIALS

We prospectively evaluated the results of unilateral IOAT in 10 consecutive patients with idiopathic hypertropia (nonparalytic, restrictive, or dissociated vertical deviation [DVD]) > 10 PD in primary position and primary unilateral IO overaction ($+3$ or $+4$ on a scale from 0 [no overaction] to $+4$ [maximum increase in adduction]). The contralateral IO function was normal in all patients. No previous extraocular muscle surgery had been performed. Four patients had esotropia, and 2 had exotropia; for these 6 patients, the horizontal deviation was corrected in the same operation, but in no case were the horizontal muscles transposed vertically. Four patients presented with only hypertropia, and IOAT was the only operation performed. Superior oblique palsy was ruled out because Bielschowsky's phenomenon was not found in any patient.

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TABLE 1. Demographic and operative data for patients who underwent unilateral anterior transposition of the IO muscle for correction of essential hypertropia in primary position

Patient no.	Sex	Age at surgery (y)	VA OD/OS	Operated eye	Follow-up (mo)
1	F	4	1.0/1.0	OD	4
2	F	17	1.0/CF	OS	2
3	F	22	CF/1.0	OD	3
4	F	9	1.0/1.0	OS	79
5	M	7	1.0/1.0	OS	3
6	M	3	0.9/0.8	OS	67
7	F	16	CF/1.0	OD	27
8	M	12	0.5/1.0	OD	12
9	F	16	1.0/0.6	OS	24
10	F	8	0.6/1.0	OD	36
Mean		11.4 ± 6.2			25.7 ± 27.7

IO, inferior oblique; VA, visual acuity; OD, right eye; OS, left eye; CF, counting fingers.

No stereopsis was present in any patient when tested with the Titmus fly test.

Alternate cover or Krimsky test was used to measure the deviation according to the degree of patient cooperation as was the presence or not of amblyopia. Muscle function was always evaluated by the same examiner observing versions and ductions.

All the patients underwent IO disinsertion and reinsertion adjacent to the lateral end of the inferior rectus insertion, bunched with only one suture (Vicryl 6.0 J570, Johnson & Johnson, São Jose dos Campos, Brazil).

Data regarding sex, age, visual acuity and follow-up time are presented in Table 1. Statistical analysis of the results was performed by Wilcoxon rank sum test for two-samples nonindependent.

RESULTS

The results are presented in Table 2. Four of the patients developed -2 deficiency of elevation in adduction and abduction in the operated eye (on a scale from 0 [no limitation to elevation in adduction] to -4 [total limitation of elevation in adduction]), and this did not change with time (mean follow-up, 25.7 ± 27.7 months; range, 2 to 79). No patient developed hypotropia of the operated eye in primary position or complained of diplopia in upgaze. No postoperative palpebral fissure asymmetry was noted. Slight lower lid curvature deformity was observed in forced upgaze in four patients.

DISCUSSION

Clinically and statistically significant decreases in the preoperative hypertropia in primary position after unilateral IOAT was observed, proving that this technique is effective when IO overaction is +3 or +4, as in cases of hypertropia caused by primary IO overaction.

Nine of ten patients studied had a postoperative vertical deviation ≤ 6 PD. The only patient with a residual deviation of 8 PD in primary position did not require reop-

eration because he was satisfied with his cosmetic appearance. We considered that hypertropia of 5 or 6 PD does not represent an important cosmetic problem. Three patients obtained full correction of the hypertropia.

Guemes and Wright⁶ performed unilateral IO-graded anterior transposition in three patients having SO paresis with 20 PD of hypertropia in primary position and obtained correction of 20 PD, 11 PD and 18 PD, respectively. Those cases cannot be compared with ours because the IO reinsertion was parallel to the inferior rectus' lateral margin; thus, the posterior fibers—the most important to get the antielevator effect—had a very small ante-riorization.

In 1977, Goldchmit et al² analyzed the amount of hypertropia in primary position with IO recessions of 6 to 13 mm. The mean correction obtained was 8.4 PD ± 4.0 PD. We cannot compare our results with any of the above because different techniques were applied, and the mean preoperative hypertropia in primary position and IO overaction were different. Elliott and Nankin⁷ found elevation deficiency in primary position and adduction, respectively, in 73% and 80% of the patients submitted to unilateral IOAT. This elevation limitation was interpreted by Stager et al⁸ and Stager⁹ as being caused by the strong attachment of the IO to its neurovascular bundle, which acts as an ancillary new insertion that converts the muscle from an elevator to a depressor after the anterior transposition of its insertion. The muscle acquires an ascendant direction from this new origin toward the new scleral insertion, parallel to the inferior rectus. This anatomic description shows why the IO changes its action from elevator to depressor. Like the inferior rectus, its new depressor action manifests itself more markedly in abduction.¹⁰

After IOAT is performed, the muscle becomes an antagonist to its natural synergist, the superior rectus muscle. It can be said that the IO becomes an antielevator, ie, it limits the elevating action of the superior rectus, because both muscles contract simultaneously to supraduction innervation, thus accounting for the effectiveness of the IOAT for correction of hypertropia in DVD.

This effect is almost nonobservable when transposition is done bilaterally, according to Elliott and Nankin.⁷ The limitation of elevation caused by IOAT is manifested mainly in abduction, which results in a pseudo overaction of the contralateral IO.

Kushner¹⁰ demonstrated that the more distant from the lateral extremity of the inferior rectus insertion the IO posterior fibers are inserted, the greater is the limitation of elevation because these new fibers become more stretched. Mims & Wood¹¹ confirmed this notion in a recent publication about a study of 123 patients. This is probably the reason for not observing significant limitation of elevation in our cases because the IO was always bunched with only one suture, placed approximately 1 mm laterally from the lateral end of the inferior rectus insertion. By the geometry of the transposed IO, some limitation of elevation

TABLE 2. Postoperative results of unilateral anterior transposition of the IO muscle

Patient no.	Preoperative HT	Postoperative HT	Preoperative version*	Postoperative version†	Correction (%)
1	20 ^Δ	8 ^Δ	+3	0	12 ^Δ (60.0)
2	10 ^Δ	6 ^Δ	+3	0	4 ^Δ (40.0)
3	12 ^Δ	0	+3	-2	12 ^Δ (100.0)
4	25 ^Δ	3 ^Δ	+3	-2	22 ^Δ (88.8)
5	20 ^Δ	2 ^Δ	+3	0	18 ^Δ (90)
6	20 ^Δ	0	+3	-2	20 ^Δ (100.0)
7	30 ^Δ	0	+4	-2	30 ^Δ (100.0)
8	15 ^Δ	4 ^Δ	+3	0	11 ^Δ (73.3)
9	25 ^Δ	6 ^Δ	+4	0	19 ^Δ (76)
10	35 ^Δ	2 ^Δ	+3	0	33 ^Δ (94.2)
Mean	20.2 ± 9.5	3.1 ± 2.8		18.1 ± 8.8	82.2 ± 19.9

*Elevation in adduction of the operated eye.

†Elevation in adduction of the operated eye. The difference between preoperative and postoperative measures was statistically significant ($P < 0.01$). IO, inferior oblique; HT, hypertropia.

may still exist, which can be demonstrated by the apparent underaction of the operated muscle that we observed in 4 of our 10 cases.

With the exception of the expected incidence of small elevation deficiency in adduction and/or abduction, no other complication was found after surgery. We did not find any hypotropia of the operated eye, similar to what was observed by Goldchmit et al.² In this 1997 study, four patients underwent unilateral IOAT, and no hypotropia was found during the postoperative period. In 1986, different results were presented by Bremer et al,⁴ who reported three cases of hypotropia in primary position after IOAT in patients with unilateral superior oblique palsy. We never employed this technique for patients with superior oblique palsy, although Gonzales¹² reported three cases with good results. Patients with superior oblique palsy often have normal binocular vision and a hypotropia may cause diplopia. We were not concerned for postoperative diplopia in our patients because they had idiopathic strabismus with its characteristic suppression.

Because of the theoretical possibility of inversion of hypertropia, although this did not happen in any of our cases, we recommend employing this surgical procedure in eyes without potential to become fixators.

In conclusion, unilateral IOAT is an effective technique for correction of large hypertropia associated with marked IO overaction.

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