Torsion as a Contributing Cause of the Anti-elevation Syndrome

Burton J. Kushner, MD

Background: The anti-elevation syndrome is an adverse outcome of anterior transposition of the inferior oblique muscle. The presumed cause is an excessive anti-elevating force vector that occurs with attempted elevation in abduction. This causes apparent overaction of the contralateral inferior oblique muscle due to fixation duress. It has been suggested that excessive residual extorsion may help explain this phenomenon. ***Methods:*** Fundus photographs to assess torsion were evaluated by masked observers in 18 patients who had undergone anterior transposition of the inferior oblique muscle. Eight of the patients were found to have the anti-elevation syndrome and 10 were not. ***Results:*** Patients with the anti-elevation syndrome had more extorsion (mean, 16.6° ± 3.4°) than the patients who did not have the anti-elevation syndrome (mean, 8.8° ± 2.3°). This difference was significant ($P < .0001$). In addition, 2 patients who initially did not show the anti-elevation syndrome were found to have an increase in their fundus extorsion after they subsequently developed the anti-elevation syndrome. Two patients who had the anti-elevation syndrome showed a marked decrease in fundus extorsion after the anti-elevation syndrome was surgically eliminated by converting the anterior transposition to a simple recession. ***Conclusion:*** The presence of substantial extorsion may contribute to the cause of the anti-elevation syndrome after inferior oblique muscle anterior transposition. Lateral placement of the posterior (lateral) corner of the inferior oblique muscle at the time of surgery may cause substantial extorsion after surgery. (J AAPOS 2001;5:172-7)

Anterior transposition of the inferior oblique muscle is a popular option for treating dissociated vertical divergence (DVD) when associated with inferior oblique muscle overaction.[1-6] By transposing the insertion of the inferior oblique muscle anterior to the equator, a force vector that opposes elevation is created, which in turn reduces DVD.[7] An adverse outcome of this procedure has been named the *anti-elevation syndrome*.[8] Although in some ways this syndrome resembles residual inferior oblique overaction, it differs from that entity in several respects. Careful observation of a patient with the anti-elevation syndrome reveals a definite limitation of elevation in abduction.[8] Unlike residual inferior oblique overaction, minimal or no elevation of the adducting eye occurs on direct horizontal side gaze in the anti-elevation syndrome. However, a marked upshoot and splaying outward (divergence) of the adducting eye are present on attempted elevation in side gaze. This creates a V or Y pattern (Figure 1). Also, there is typically a noticeable bulging of the lower eyelid on attempted upgaze.[9] Although this latter finding occurs in patients after anterior transposition of the inferior oblique muscle irrespective of the occurrence of the anti-elevation syndrome, it is not seen in patients who have undergone standard inferior oblique weakening surgery (eg, recession or myectomy) nor in patients with previously unoperated inferior oblique overaction. Thus, the presence of this sign can be helpful in diagnosing the anti-elevation syndrome in a patient whose prior surgical history is unknown.

The anti-elevation syndrome can be eliminated by converting the prior anterior transposition of the inferior oblique muscle to a standard recession, although this may risk a recurrence of the DVD.[8] The resolution of this pseudo–inferior oblique overaction by changing a more powerful inferior oblique weakening procedure (anterior transposition) to a less powerful weakening procedure (standard recession) is the opposite of what one would expect if one was actually treating residual inferior oblique overaction. Finally, when the correction of bilateral but asymmetric anti-elevation syndrome had been attempted unilaterally by retroplacing one of the anteriorly transposed inferior oblique muscles, the pseudo–inferior oblique overaction in the contralateral eye was eliminated. However, it was unchanged in the eye in which the anteri-

From the Department of Ophthalmology & Visual Sciences, University of Wisconsin, Madison, Wisconsin.
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Reprint requests: Burton J. Kushner, MD, Department of Ophthalmology & Visual Sciences, 2870 University Ave, Suite 206, Madison, WI 53705 (e-mail: bkushner@facstaff.wisc.edu).

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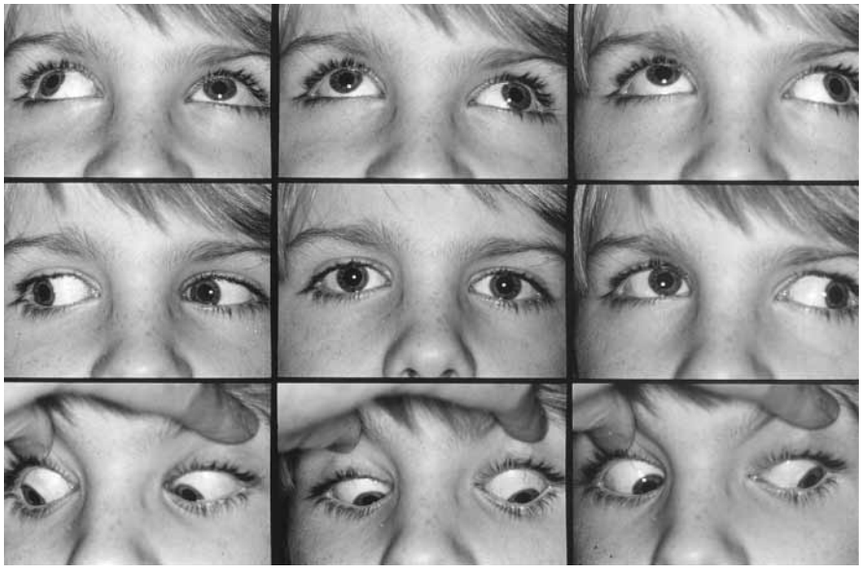

FIG 1. Patient with anti-elevation syndrome after prior bilateral inferior oblique anterior transposition. A large V pattern is present. There is excessive elevation of the adducting eye on attempted upgaze secondary to restriction of elevation of the abducting eye on upgaze.

or transposition was revised.[8] This confirms that the anti-elevation syndrome differs from residual inferior oblique muscle overaction.

It has been suggested that the anti-elevation syndrome is caused by an excessive anti-elevating effect of the transposed inferior oblique muscle. This force vector is greater in abduction than in adduction and hence restricts elevation of the abducting eye. On attempted elevation in side gaze, the adducting eye elevates excessively due to fixation duress. The fact that forced ductions are normal to elevation in abduction in patients with the anti-elevation syndrome has led to speculation that the restriction of elevation is innervational rather than mechanical.[9] Further anterior placement of the insertion would be expected to increase the anti-elevating torque, and further posterior placement should decrease the anti-elevating torque. This is analogous to how advancement or recession of a rectus muscle increases or decreases, respectively, the torque the muscle creates. Although the inferior oblique muscle normally has its greater elevating action in adduction, Stager et al[10,11] have suggested that the neurovascular bundle serves as the new functional origin of the muscle after anterior transposition. This hypothesis has been used to explain why the limitation of elevation is greater in abduction than in adduction in patients with the anti-elevation syndrome.[8] The anti-elevation syndrome has been shown to occur more frequently if the posterior temporal corner of the inferior oblique muscle insertion is spread out laterally at the time of anterior transposition, rather than bunched up with the anterior nasal corner. It is also more likely to occur if the new insertion of the inferior oblique muscle is placed anterior to the insertion of the inferior rectus muscle or if some of the inferior oblique muscle is resected.[8]

Although the recent contributions by Stager et al about the anatomy of the neurofibrovascular bundle clarify some aspects of the pathophysiology of the anti-elevation syndrome, some characteristics of it are yet to be explained. When an eye is under mechanical duress, it normally assumes a position that minimizes the length of the restricting elements. One might therefore expect that if the restriction of elevation is greater in abduction, patients with the anti-elevation syndrome should show an A pattern rather than a V pattern. Why the eyes assume a more divergent position in upgaze when the restriction is greater in abduction needs clarification. In my original description of the anti-elevation syndrome, I speculated that if there is substantial residual extorsion, the presence of a V or Y pattern might be understandable.[8] As has been previously described, substantial extorsion will cause the superior rectus muscle to have an abducting vector and the lateral rectus muscle to have a depressing vector,[12,13] both of which can contribute to the pattern observed in the anti-elevation syndrome. Theoretically, if extorsion is contributing to the Y or V pattern in the anti-elevation syndrome and if that syndrome can be corrected with conversion to a standard inferior oblique recession, then that corrective surgical procedure should be associated with a decrease in extorsion. At the time of my initial report, I suggested that this subject deserves further investigation.[8] If, in fact, there is substantially more extorsion present in patients who develop the anti-elevation syndrome after inferior oblique anterior transposition than in those who do not, the mechanism for the V or Y pattern can be explained. The purpose of this study is to investigate the role that torsion may play in the occurrence of the anti-elevation syndrome.

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TABLE. Descriptive parameters of patients

	No. of patients	Age at surgery, y (mean ± SD)	Sex (male/female)	Eye analyzed (OD/OS)	Preop IOOA* (mean ± SD)	Time of postop outcome examination, mo (mean ± SD)	Post-operative extorsion, ° (mean ± SD)
Anti-elevation syndrome	8	5.6 ± 2.0	5/3	4/4	+2.6 ± 0.9	7.2 ± 0.9	16.6 ± 3.4
Control subjects	10	5.8 ± 1.9	6/4	5/5	+2.7 ± 0.9	7.1 ± 0.9	8.8 ± 2.3

*IOOA Denotes inferior oblique overaction graded on a scale of +1 to +4.

SUBJECTS AND METHODS

This series consists of consecutive patients seen by me as part of or subsequent to my original report of the anti-elevation syndrome who met certain inclusion criteria.[8] They all underwent bilateral anterior transposition of the inferior oblique muscle according to the technique previously described for the purpose of treating bilateral DVD, which was intermittently manifested in at least one eye and associated with bilateral inferior oblique overaction. Before the operation, the amount of inferior oblique overaction was graded subjectively on a scale from +1 to +4. Objective fundus extorsion before surgery was simply graded as being absent, borderline, present, or markedly present, based on subjective assessment using indirect ophthalmoscopy. All patients were seen by me in follow-up at least 6 months after the inferior oblique muscle surgery and, in accordance with the methodology described below, had to be sufficiently cooperative for fundus photography as an inclusion criterion for this study. This latter requirement resulted in a minimum age of 3 years for inclusion. In all but 4 patients, the inferior oblique muscles in both eyes were placed level with and just temporal to the temporal end of the inferior rectus muscle insertion. The posterior lateral corner of the cut end of the inferior oblique muscle was bunched up with the anterior nasal corner, as has been previously described.[8] The exceptions were the 4 patients from my original report in whom the inferior oblique muscles either were placed anterior to the inferior rectus muscle insertion or had their posterior lateral corners spread out laterally at the time of transposition.

The outcome determination date for this study was the first examination that was at least 6 months after the inferior oblique anterior transposition procedure. At that clinic visit, the patient was diagnosed as either having or not having the anti-elevation syndrome based on the diagnostic criteria described above and previously published in detail.[8] At the time of that examination, fundus photographs to assess torsion were taken in a manner similar to that previously described.[14] For this study, however, head positioning was strictly controlled to avoid inadvertent tilting by the use of the CROM (cervical range of motion) device, as has also been previously described in detail.[15] Briefly, this apparatus is a headband to which a compass and gravity meters are attached that can assess the presence of a head tilt with an accuracy of 1°. The fundus images were printed as 8 × 10-inch photographs and were each graded by 1 of 2 graduate students who served as examiners and who were masked as to the patient's clinical findings. Torsion on the photographs was assessed in a manner similar to that described by Santiago et al.[16] This consisted of drawing a line from the center of the optic disk to the fovea and measuring the amount of torsion with a protractor to the nearest 0.5°. Because the anti-elevation syndrome is usually symmetric in my experience (if the anterior transposition surgery was performed symmetrically), either the right eye or left eye from each patient was chosen randomly and used for data analysis. To evaluate reproducibility of the measurement technique, each observer graded 5 photographs twice on different occasions without knowing that he or she had previously graded that same photograph. In addition, 6 photographs were graded once by each of the 2 examiners to assess interobserver reliability. The test of reproducibility of the measurement technique confirmed that the 2 findings always agreed within 1° for all photographs undergoing a second analysis. Of the 6 photographs that were graded by each of the 2 masked examiners, there was agreement within 1° in 5 of the photographs and within 2° in the sixth. This indicates a high degree of reproducibility and interobserver reliability with the methodology.

In addition, there were 2 patients seen during the course of this study who did not have the anti-elevation syndrome when initially photographed 6 months after surgery but did show the anti-elevation syndrome on subsequent visits. They also underwent fundus photography at the time the anti-elevation syndrome was diagnosed, and those photographs were similarly analyzed. Finally, there were 2 patients who had the anti-elevation syndrome by the sixth month outcome determination date, who later underwent surgery to correct the anti-elevation syndrome. This consisted of moving the inferior oblique muscle insertion to a point 4 mm posterior to the temporal corner of the inferior rectus insertion bilaterally. These 2 patients also underwent fundus photography approximately 6 months after the second operation.

RESULTS

This series consists of 10 patients who underwent inferior oblique anterior transposition who did not develop the anti-elevation syndrome (control group) and 8 patients who did develop the anti-elevation syndrome. See the Table for descriptive details. Of the control group patients,

all had the inferior oblique muscle placed level with and just temporal to the temporal corner of the inferior rectus muscle insertion. Of the 8 patients who had the anti-elevation syndrome, 2 had the inferior oblique muscle placed 2 mm anterior to the inferior rectus insertion, and 2 patients had the temporal posterior corner spread out laterally at the time of inferior oblique anterior transposition. These 4 patients were included in my original report.[8] In the remaining 4 patients, the inferior oblique muscle was positioned in the same manner as was done in the control group.

All 18 patients in this study were determined to have objective fundus extorsion present before surgery. In 3 patients in the anti-elevation group and in 3 patients in the control group, it was believed to be markedly present before surgery. As seen in the Table, the mean fundus extorsion after surgery was significantly greater in the anti-elevation syndrome group (16.6° ± 3.4°) than in the control group (8.8° ± 2.3°), and this difference was significant ($P < .0001$; unpaired t test). Of the 2 patients who did not have the anti-elevation syndrome when first studied and who later developed it, extorsion on the first outcome evaluation was found to be 6.0° and 9.0°, respectively. The second examination (after the anti-elevation syndrome had developed) revealed fundus extorsion of 18.0° and 18.5°, respectively. The 2 patients who manifested the anti-elevation syndrome initially were found to have fundus extorsion of 21.0° and 18.0°, respectively, in the eye chosen for analysis. After repositioning the inferior oblique muscle posteriorly, the same eyes had a decrease in extorsion measuring 7° in each patient for a final extorsion measurement of 14.0° and 11.0°, respectively. In both patients, this surgery successfully eliminated the limitation of elevation in abduction and collapsed the Y pattern.

DISCUSSION

This study shows that patients who develop the anti-elevation syndrome after inferior oblique anterior transposition have significantly more extorsion after surgery than patients who do not develop that syndrome. This observation clarifies several previously unexplained findings of the anti-elevation syndrome. For the reasons stated earlier, one would expect a restriction of elevation in abduction to cause an A pattern. But if there is a greater amount of extorsion accompanying that restriction, the vector changes caused by the extorsion may cause the divergence in upgaze seen in the anti-elevation syndrome. As a result of the extorsion, the superior rectus muscle will have a partial abducting vector, and the lateral rectus muscle will have a partial vector for depression.

It has also become evident to me that the anti-elevation syndrome frequently is not present initially after inferior oblique anterior transposition but can develop as much as several years later. Guyton and Weingarten[13] have suggested that patients with a fusion disruption may develop a sensory torsional drift. This observation, combined with the late increase in extorsion seen in the 2 patients who developed the anti-elevation syndrome long after surgery, may explain the late occurrence of the anti-elevation syndrome that is seen in some patients.

FIG 2. Diagram of an anteriorly transposed inferior oblique muscle as seen from below. The neurofibrovascular bundle is the *thin black line*. If the neurofibrovascular bundle is considered to be the functional origin of the inferior oblique muscle, the muscle fibers between this functional origin and insertion are almost parallel to the inferior rectus muscle. The farther the temporal the posterior lateral corner is placed *(straight arrow),* the greater the extorsional vector produced *(curved arrow).*

It has been suggested that putting the posterior lateral fibers of the inferior oblique muscle on substantial stretch may contribute to the development of the anti-elevation syndrome.[10,11] Because of Stager et al's work, it is recognized that lateral placement of the posterior lateral corner of the inferior oblique muscle will make the posterior lateral fibers somewhat more taut.[10,11] However, this mechanism, as the sole explanation for the occurrence of the anti-elevation syndrome, seems lacking to me in several respects. I have previously calculated that a 5-mm lateral positioning of the posterior lateral corner of the inferior oblique muscle would only increase the stretch on the posterior lateral fibers by approximately 0.5 mm.[8] Also, intraoperatively, the posterior lateral fibers of the inferior oblique muscle do not feel taut to me even when the posterior lateral corner is held in a far lateral position. As previously stated, passive ductions to elevation in abduction are normal in patients with the anti-elevation syndrome. If the posterior lateral fibers of the inferior oblique muscle were excessively taut, one would expect passive ductions to be abnormal for elevation in abduction. Nevertheless, evidence exists that lateral spreading of the posterior lateral corner does increase the incidence of the anti-elevation syndrome.[8] It seems another pathophysiologic mechanism may be important. Specifically, the effect on torsion of lat-

eral spreading of the posterior-lateral corner of the inferior oblique muscle could be relevant. The initial theoretic description of the anterior transposition operation by Scott[7] focused primarily on the anti-elevating effect that would be created by putting the inferior oblique insertion anterior to the equator. Later, Apt and Call[17] speculated that bunching the posterior corner anteriorly would decrease the torsional action of the inferior oblique muscle. At the time they proposed this technique, knowledge of how the neurofibrovascular bundle may act as a new functional origin of the inferior oblique muscle after anterior transposition had not yet been elucidated.[10,11] Anterior bunching of the posterior fibers would decrease the torsional action of the inferior oblique muscle if the functional course of the inferior oblique muscle was still running from the anatomic origin at the nasal orbital wall to the new insertion. Such positioning of the inferior oblique muscle would shift its force vector to run more parallel with the y-axis and decrease the torsional effect of the muscle. However, if the neurovascular bundle does indeed act as the functional origin of the transposed inferior oblique muscle, the rationale for moving the posterior corner forward needs to be reevaluated. Mumma[18] has shown that horizontal transposition of a vertical rectus muscle creates a torsional vector in a direction toward the initial insertion of the muscle. This has been subsequently confirmed by others.[19] For example, temporal transposition of the inferior rectus muscle would induce an extorsional shift. If the inferior oblique muscle is transposed to the temporal corner of the inferior rectus insertion, then the new course of the muscle from its functional origin (the neurofibrovascular bundle) to its new insertion approximately parallels the course of the inferior rectus muscle. But if the inferior oblique muscle insertion is just temporal to the inferior rectus muscle insertion, this would have a similar effect as a small temporal transposition of the inferior rectus muscle (thus causing extorsion). Further temporal spreading of the posterior lateral corner of the inferior oblique muscle would further increase the extorsional vector (Figure 2). Therefore, bunching the inferior oblique closer to the inferior rectus would cause less extorsion than spreading the muscle out temporally. The suggestion by Apt and Call[17] to bunch the inferior oblique muscle insertion at the time of the anterior transposition seems justified; however, the mechanism is probably different than the one they considered. It is not the anterior bunching of the muscle that minimizes extorsion, it is the less temporal positioning that has that effect. Also, if placing the inferior oblique muscle just temporal to the inferior rectus muscle insertion still mimics a temporal transposition of the inferior rectus muscle, then increasing the force generated by the inferior oblique muscle by either resecting it or placing it anterior to the inferior rectus insertion would increase the extorsional action. All of these can contribute to the anti-elevation syndrome.

One additional mechanism for the anti-elevation syndrome deserves mention. Temporal placement of the transposed inferior oblique muscle should also theoretically increase its mechanical advantage with respect to the z-axis, especially under the duress of upgaze, and hence contribute to the Y pattern. However, this mechanism alone does not explain the increased occurrence of the anti-elevation syndrome with greater amounts of postoperative extorsion. The relative importance of this possible mechanism is impossible to determine from this study.

These data are consistent with the recent study by Santiago et al,[16] which looked at torsional changes found after inferior oblique anterior transposition. Although they found an initial decrease in extorsion after surgery, the reduction in extorsion had dissipated by 10 weeks. This observation is consistent with the concept that anterior transposition of the inferior oblique muscle creates a force vector for extorsion. Once the transposed muscle heals and takes up its slack, the extorsion becomes evident. Because forced ductions are normal for elevation in abduction in patients with the anti-elevation syndrome, late contracture of the transposed muscle is an insufficient hypothesis to explain its occurrence. However, the change in the direction of the force vectors of the rectus muscles, secondary to the increased extorsion, will explain the clinical findings of the anti-elevation syndrome. This mechanism is also consistent with the concept of a sensory extorsional drift as proposed by Guyton and Weingarten.[13] Notably, Santiago et al[16] found that a much greater torsion was present after inferior oblique anterior transposition if there was residual inferior oblique overaction than if the inferior oblique muscle function was normal after surgery. Because their study was conducted before the anti-elevation syndrome was described,[8] it is tempting to speculate that some of the patients they described as having residual inferior oblique overaction in fact had the anti-elevation syndrome. If so, their findings are completely consistent with my data.

One additional possible interpretation of these data deserves mention. If anti-elevation syndrome is related to increased excyclotorsion, then it is also possible that anti-elevation syndrome results from a greater amount of excyclotorsion before the operation, rather than as a result of surgical technique. To test this hypothesis, accurate quantitative data about the amount of extorsion present before surgery would be necessary. Unfortunately, such data are not available for the patients in this study. Nevertheless, I believe that explanation is unlikely for several reasons. Although in this study, preoperative torsion was not quantified exactly, it was assessed according to a subjective scale. The number of patients in the control group who had marked extorsion before surgery was exactly identical to the number in the group that developed the anti-elevation syndrome (3 in each group). Also, the late development of the anti-elevation syndrome in 2 patients, which was accompanied by a later increase in postoperative excyclotorsion, supports the concept that it is extorsion after surgery, rather than before surgery, that is causative. Finally, the occurrence of the anti-elevation syn-

drome has been shown to correlate with magnitude of the anterior transposition of the inferior oblique, or the amount of lateral spreading of the posterior lateral corner of the muscle.[8] These observations suggest that the occurrence of the anti-elevation syndrome is more related to variables in surgical technique, rather than preoperative factors. Nevertheless, a prospective study would be necessary to definitively address this issue.

This study is to be viewed in light of several other limitations. Ideally, patients would have been photographed before surgery as well as afterwards to see how the torsion changed in each specific patient. Because this was not done, that information is unavailable. As indicated earlier, a prospective study would be needed to definitively clarify the role played by preoperative extorsion. Also, the diagnosis of the anti-elevation syndrome is somewhat subjective. I do believe, however, that once its diagnostic criteria are understood, anti-elevation syndrome becomes easy to differentiate from inferior oblique overaction.

In conclusion, it appears that the anti-elevation syndrome is associated with significant residual extorsion. Although this study does not definitely prove that extorsion is the cause to the anti-elevation, it is strongly suggestive thereof. This hypothesis also explains why the anti-elevation syndrome is associated with a V or Y pattern instead of an A pattern, and why it can develop a long time after surgery. It also can explain why spreading the insertion of the transposed inferior oblique out laterally or placing it anterior to the inferior rectus muscle insertion can contribute to the development of the anti-elevation syndrome.

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