Objectives: To determine if ocular torsion is a major cause of A and V patterns and oblique muscle overaction or merely a contributing factor.

Methods: Three separate investigations were conducted. (1) The trajectory of eyes with oblique muscle overaction was plotted across the horizontal field of gaze from videographs to determine if it was linear or curvilinear. (2) The effect of successful Harada-Ito surgery to reduce extorsion on overelevation in adduction in patients with fourth cranial nerve palsy was studied. (3) The effect of successful surgery to treat pattern strabismus in the form of vertical transposition of the horizontal rectus muscles on objective torsion was studied.

Results: (1) Three eyes with inferior oblique muscle overaction and 2 with superior oblique muscle overaction had a curvilinear rise or fall (respectively) as they moved into adduction. (2) Surgery that successfully decreased extorsion had a negligible effect on overelevation in adduction in 2 patients. (3) Horizontal rectus muscle transposition that was uniformly successful in eliminating A or V patterns consistently caused an increase in objective torsion in all 5 patients studied.

Conclusion: Ocular torsion may contribute to A or V patterns and overelevation or overdepression in adduction, but it is probably not the major cause of these phenomena.


Estimates of the occurrence of A or V patterns with horizontal strabismus range between 12% and 50%, depending on the nature of the series from which the figure is derived.1-3 Over the years, various etiologies have been proposed for A and V patterns. These have included differing action of the horizontal rectus muscles in upgaze vs downgaze,1,6 weakness of the vertical rectus muscles,7 abnormal sagittalization of the oblique muscles,8 and more recently orbital pulley abnormalities.9 However, the most popular theory, proposed by Knapp2 in 1959, implicates abnormal oblique muscle function as the cause of A and V patterns. Abduction is the tertiary action of the oblique muscles. If the inferior oblique muscles are overacting and the superior oblique muscles are underacting, there will be an excessive force vector for abduction in upgaze and a relatively deficient abducting force vector in downgaze, resulting in a V pattern. Conversely, superior oblique muscle overaction and inferior oblique muscle underaction will cause a relative divergence in downgaze compared with upgaze, producing an A pattern. This is the basis for the Knapp theory.

Based on some prior studies by Weiss10 and Morax et al11,12 in patients with craniofacial anomalies, I calculated the effect that ocular torsion may play in contributing to A and V patterns, as well as to the overelevation or overdepression seen in adduction in patients with oblique muscle overaction.13 For example, if a patient has a V pattern with inferior oblique muscle overaction, each eye will be extorted. This will rotate the insertions of the rectus muscles counterclockwise in the right eye and clockwise in the left eye. The change in force vector resulting from this rotation will create an abducting force from the superior rectus muscles and an adducting force from the inferior rectus muscles, which will contribute to the V pattern. In addition, the new positions of the insertions of the horizontal rectus muscles will make the medial rectus muscles partial elevators in adduction and the lateral rectus muscles partial depressors in abduction. This will accentuate the overelevation seen in adduction (Figure 1 and Figure 2). I calculated that

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for the average amount of torsion observed in a series of patients with A and V patterns, these new force vectors were substantial. The extorsion seen in patients with V patterns will cause the new force vector of each rectus muscle to be approximately equal to 24% of its original force, e.g., an abducting force of the superior rectus muscle, elevating force of the medial rectus muscle, and adduction for the inferior rectus muscle. The converse occurs with intorsion accompanying A patterns. Although these calculations were made before the recognition of orbital pulleys, and hence are probably not quantitatively accurate, they should be qualitatively valid. In a publication in 1994, Guyton and Weingarten carried this hypothesis an important step further by hypothesizing how the disruption of fusion that occurs with horizontal strabismus may result in a free-wheeling torsional drift of the eyes. This sets the stage for the development of A or V patterns as well as overelevation or overdepression in adduction. Later, Miller and Guyton confirmed this hypothesis in patients who lost fusion after strabismus surgery. However, although I conceptualized that ocular torsion was merely a contributing factor to A and V patterns, as well as overelevation or overdepression in adduction, others describe this as the major causative factor. Which of these differing hypotheses is correct can have important practical clinical implications in choosing a surgical plan to treat some patients. Specifically, when addressing A or V patterns, should the surgical plan take into account the vertical and horizontal actions of the operated-on muscles, or is treating torsion sufficient?
The purpose of this study was to determine if ocular torsion is the primary cause, or merely a contributing factor, in the genesis of A and V patterns as well as overelevation or overdepression in adduction.

METHODS

This study consists of a series of 3 different investigations made from patients in my clinical practice. The study was approved by the University of Wisconsin institutional review board and was compliant with the Health Insurance Portability and Accountability Act.

INVESTIGATION 1: TRAJECTORY OF OBLIQUE MUSCLE OVERACTION

In my oral discussion of the Guyton and Weingarten presentation on the effect of torsion on pattern strabismus and oblique muscle overaction, I suggested that the elevation of an eye with inferior oblique overaction as it moved from abduction to adduction should be approximately linear, if the force vector changes of the rectus muscles from torsion as depicted in Figure 1 and Figure 2 were responsible for the apparent overaction (B.J.K., unpublished oral discussion of “Sensory Torsion as the Cause of Primary Oblique Muscle Overaction/Underaction and A and V” patterns by Guyton and Weingarten at the 19th annual scientific meeting of the American Association for Pediatric Ophthalmology and Strabismus, Palm Springs, California, April 20, 1993). The same should be true for the depression of an eye with superior oblique muscle overaction as it moves into adduction. However, if the elevation or depression were caused by an increase in the vertical torque vector of the respective oblique muscle, the elevation or depression should follow a relatively curvilinear course, which is more consistent with the change in vertical force vector of the respective muscle in adduction.

For this investigation, the motility of patients with primary inferior or superior oblique muscle overaction was videotaped. Videography was performed while fixation was maintained with the same eye across the horizontal plane from far abduction to far adduction. Thus, the nonfixing eye was mov-
ing from abduction to adduction, and its vertical position across the horizontal plane was analyzed. Individual still frames from the videos were captured at intervals of approximately 0.20 second and saved as still pictures. The position of the center of the pupil of the nonfixing eye was then graphed as a function of horizontal movement to determine its trajectory as the eye rotated from abduction to adduction across the horizontal plane. To ensure adequate excursion of the nonfixing eye into abduction, I only included patients with 20 prism diopters (PD) or less of esotropia or exotropia in the primary position. No specific mechanical device (eg, bite bar, forehead rest) was used to control head position during videotaping. However, the still frames were analyzed by drawing a line between the outer canthi of each eye. The orientation of the line was compared in selected images across the horizontal gaze field to ensure there was no inadvertent tilting of the head.

INVESTIGATION 2: EFFECT OF REDUCING THE EXCYLOTOPIA ON OVERELEVATION IN ADDUCTION IN PATIENTS WITH INFERIOR OBLIQUE MUSCLE OVERACTION

If the excyclotropia that accompanies inferior oblique muscle overaction is primarily responsible for the overelevation in adduction, reducing the torsion should reduce the overelevation. This investigation involved studying the effect of purely torsional surgery on the overelevation in adduction in patients with inferior oblique muscle overaction secondary to fourth cranial nerve palsy. Torsion was measured subjectively before and after surgery using the double Maddox rod test in the previously described manner and objectively with fundus photography. Measurements of the angle of strabismus were made at 6 m with appropriate spectacle correction in place using the prism and alternating cover test. Oblique muscle function was subjectively graded using a 9-point scale (−4 to +4). The patients in this investigation had been previously described in a report of surgical procedures to treat torsion, and their records were retrospectively reviewed for this study.

INVESTIGATION 3: EFFECT OF VERTICAL TRANPOSITION OF HORIZONTAL RECTUS MUSCLES ON BOTH THE TORSION AND ALPHABET PATTERNS

If ocular torsion is primarily responsible for A and V patterns, surgery that corrects a pattern should decrease torsion, and surgery that worsens torsion should worsen an A or V pattern. Alphabet patterns are often treated with vertical transposition of the horizontal rectus muscles. When a rectus muscle is transposed, 3 separate changes in force vector occur. The muscle’s primary action is lessened when the eye is rotated in the direction of the transposition and increased when the eye is rotated in the opposite direction (Figure 3A). This is the basis for using horizontal rectus muscle transpositions to treat A or V patterns. However, at the same time, a vertical force vector is created in the direction the muscle is moved. This is the principle behind using muscle transposition surgery to treat paralytic strabismus such as sixth cranial nerve palsy or monocular elevation deficiency. Finally, a torsional force vector is created in the direction from which the muscle was moved (Figure 3B). Consequently, transposition of the horizontal rectus muscles to treat pattern strabismus should theoretically create a torsional change in the opposite direction from what is desired. For example, a V pattern esotropia is often associated with inferior oblique muscle overaction and objective extorsion. However, successful treatment of a V pattern with only minimal inferior oblique muscle overaction can involve medial rectus muscle recessions with infraplacement, which should in theory worsen the extorsion. This investigation studied the change in objective torsion and the magnitude of A or V patterns in patients undergoing surgery consisting of vertical transposition of horizontal rectus muscles to treat the patterns. Transposition surgery was accomplished by keeping the superior and inferior poles of the new insertion of the transposed muscle a consistent distance from the limbus. For example, if a muscle was initially inserted 5 mm from the limbus, and the recession was 5 mm, the entire length of the new insertion was 10 mm from the limbus. Fundus torsion was documented photographically before and after surgery and graded in the manner previously described. This grading system, which is a modification of that described by Guyton, allows for a quantitative assessment of torsion on a scale of 0 to +4 as well as a calculation of the change in torsion between different examinations in actual degrees. Strabismus measurements were all made at 6 m with appropriate optical correction in place using the prism and alternating cover test.

For investigations 2 and 3, the outcome examination was approximately 6 weeks after surgery.

INVESTIGATION 1: TRAJECTORY OF OBLIQUE MUSCLE OVERACTION

This investigation included 3 patients with bilateral primary inferior oblique muscle overaction and 2 with bilateral primary superior oblique muscle overaction. All 5 patients had decompensated partly accommodative esotropia, had not undergone prior surgery, and did not have dissociated vertical divergence. For all 5 patients, the elevation or depression of the eye was curvilinear as the eye moved from abduction to adduction. Figure 4 shows the analysis of 1 patient with inferior oblique muscle overaction that was graded as +4 bilaterally. The plot of eye position for the other 2 patients with inferior oblique muscle overaction was similar; for the 2 patients with superior oblique muscle overaction, the trajectories were inverted but similar. This suggests
that the change in vertical force vector of the oblique muscle between abduction and adduction is the primary cause of the overelevation or overdepression in patients with primary oblique muscle overaction. On the contrary, if ocular torsion were the primary cause for the overelevation or overdepression seen in adduction in these patients, one would expect the rise or fall should be approximately linear.

INVESTIGATION 2: EFFECT OF REDUCING THE EXCYLOTROPIA ON OVERAGELEVATION IN ADDUCTION IN PATIENTS WITH INFERIOR OBLIQUE MUSCLE OVERACTION

This investigation involved 2 patients with unilateral acquired superior oblique muscle palsy for which each had undergone contralateral inferior rectus muscle recession by other ophthalmologists. This corrected the hypertropia in the primary position but left them with residual bothersome diplopia due to persistent exyclotropias of 8° and 9°, respectively. They each had +3 unilateral inferior oblique muscle overaction of the affected eye. Although they had undergone contralateral inferior rectus muscle recession, neither had objective torsion of the contralateral eye as determined by fundus photography, although in both patients the position of the fovea was closer to the border for intorsion than extorsion. Because they had no hypertropia in the primary position, they each underwent a modified Harada-Ito procedure. After surgery, they each reported no torsion with the double Maddox rod test; however, the inferior oblique muscle overaction was unchanged in 1 patient and only decreased from +3 to +2 in the other. Before surgery, the hypertropia of the affected eye measured 12 PD and 10 PD in adduction in the 2 patients, respectively; and after surgery, it was only reduced to 10 and 6 PD, respectively. They had a V pattern reduction of 2 and 4 PD, respectively, which was identical to the reduction of the hypertropia in adduction. Neither had a hypertropia in the primary position postoperatively.

INVESTIGATION 3: EFFECT OF VERTICAL TRANSPosition OF HORIZONTAL RECTUS MUSCLES ON BOTH THE TORSION AND ALPHABET PATTERNS

For this investigation, 5 patients with horizontal strabismus associated with an alphabet pattern underwent surgery in the form of bilateral horizontal rectus recessions with one-half or three-quarter tendon width vertical transposition. Three had V patterns with uncompensated partly accommodative esotropia, and 2 had A patterns with intermittent exotropia. All had mild oblique muscle overaction (inferior oblique muscle for the V patterns and superior oblique muscle for the A patterns), and all had objective extorsion or intorsion, respectively. In all patients, the pattern was effectively collapsed with the surgery, and in all patients, there was a small increase in the fundus torsion after surgery (mean sum of right and left eyes for the 5 patients was 6.4°; range, 5°-7°). No patient experienced an increase in the oblique muscle overaction of either eye, and 2 patients each had a mild decrease in 1 eye. The details of these patients are presented in the Table.

This study consisted of 3 investigations to assess different observations that could address the issue of whether ocular torsion is the primary cause of pattern strabismus or apparent oblique muscle overaction, or if it is merely contributory. All 3 suggested that ocular torsion was not primarily responsible for either.

Prior investigations support this hypothesis. In my oral discussion of the Guyton and Weingarten presentation on the effect of torsion on pattern strabismus and oblique muscle overaction (B.J.K., unpublished oral discussion of “Sensory Torsion as the Cause of Primary Oblique Muscle Overaction/Underaction and A and V” patterns by Guyton and Weingarten at the 19th annual
Scientific meeting of the American Association for Pediatric Ophthalmology and Strabismus, Palm Springs, April 20, 1993), I stated that I had the impression that objective torsion is often present in children with infantile esotropia, long before they develop overelevation in adduction. I reasoned that if torsion is the cause of overelevation in adduction in patients with inferior oblique muscle overaction, the 2 findings should theoretically occur approximately concurrently. Subsequently, in 1996, Eustis and Nussdorfer published a prospective study that nicely addressed the relative timing of the onset of primary inferior oblique overaction and the appearance of objective extorsion. They found that the occurrence of fundus extorsion was highly predictive of subsequent development of overelevation in adduction in patients with infantile esotropia, but the former often preceded the latter by as much as several years.

Investigation 1 confirmed that the vertical trajectory of an eye with superior or inferior oblique muscle overaction is curvilinear as the eyes move across the horizontal field. The altered force vectors of the horizontal rectus muscles in a torted, nonfixing eye as depicted in Figure 1 should produce an approximately linear elevation or depression of the nonfixing, torted eye as the eyes move across the horizontal plane, if their force vectors were the main factors responsible for the vertical excursion. In fact, the behavior of the fixing eye should also play a role. Consider, for example, a patient with bilateral superior muscle overaction and bilateral extorsion. When the fixing eye is in the abducted position, there will need to be innervation to elevate the eye to overcome the depression that would be driven by the overacting superior oblique muscle and/or the depression caused by the incyclorotated position of the medial rectus muscle insertion to maintain the eye in the fixing position on the horizontal plane. Although the field of greatest elevation of the inferior oblique muscle is in adduction, it has been shown that the superior rectus is still a greater elevator across the entire horizontal plane than the inferior oblique muscle.26–29 The converse is true for depression that primarily comes from the inferior rectus muscle across the horizontal gaze fields even though the superior oblique muscle has its greatest vector for depression in adduction. Thus, in the example of a patient with bilateral superior oblique muscle overaction, the elevating force vector to overcome the downward pull from the overacting superior oblique muscle and the incyclorotated position of the medial rectus insertion would come primarily from the superior rectus muscle in the fixing eye. This would be matched with further input to the superior rectus muscle in the nonfixing eye, thus increasing its vertical excursion. The vertical force vectors of the vertical rectus muscles do not vary as much between adduction and abduction as do the force vectors of the oblique muscles. Consequently, the influence of the vertical muscles in the fixing eye would also tend to result in an approximately linear vertical trajectory of the nonfixing eye if the overelevation or overdepression was primarily caused by ocular torsion. The fact that the trajectory is curvilinear suggests that torsion is not the primary cause.

Investigation 3, which studied the effect of horizontal rectus transposition on both the alphabet pattern and ocular torsion, is probably the most compelling. At first glance, it would seem conclusive that if increasing torsion still resulted in a decrease in the pattern that torsion could not be the primary cause of the pattern. To fully analyze the implications of these findings, however, the effect of the transposition on other affected muscles must be analyzed. Consider a patient with V pattern esotropia, mild bilateral inferior oblique muscle overaction, and bilateral extorsion. If the medial rectus muscles are surgically infrapaced, they might be moving from an abnormally elevated position preoperatively (due to the extorsion) to a more normal position, depending on whether they in fact were abnormally elevated initially. This would decrease the vertical force vector of preoperatively abnormally elevated medial rectus muscle and increase its horizontal force vector. The lateral rectus muscle, which might have been abnormally down preoperatively due to the extorsion, would be moved down even further when the surgery increases the extorsion. This exacerbation of the downward force vector of the lateral rectus muscle would cause an apparent increase in the inferior oblique muscle overaction as the abducting eye would be lower than the adducting eye. Similarly, the increased extorsion will increase the adducting force of the inferior rectus muscle and the adducting force of the superior rectus muscle. Thus, the increase in extorsion caused by infrapacing the medial rectus muscles should worsen the overelevation in adduction and also worsen the V pattern. In fact, vertical transposition of the horizontal rectus muscles should decrease the pattern in all patients. No patient experienced an increase in overelevation or overdepression in adduction, and some showed a slight decrease. Thus, even when taking into account the effect of rectus muscle transposition on unoperated-on muscles, it appears that torsion cannot be the main cause of either pattern strabismus or overelevation or overdepression in adduction.

Prior studies on induced torsional changes after transposition of the horizontal rectus muscle to treat A or V patterns only looked at subjective torsion using the double Maddox rods, Maddox wing, or an amblyoscope and found essentially no torsional change.30 However, we now know that subjective torsion does not correlate with the actual anatomical torsional position of the eyes in patients without bifoveal fusion or if the strabismus had its onset in childhood.16,22 Studying the actual anatomical change in torsion (objective torsion) would be a more useful way of determining if torsion is the cause of pattern strabismus or oblique muscle overaction. More recently, Sharma and coworkers31 studied the effect of horizontal rectus muscle transposition for treating A and V patterns on both objective and subjective torsion. Although they only assessed subjective torsion quantitatively and looked at objective torsion qualitatively, they also found that rectus muscle transposition routinely worsened the existing torsion.

Some authors have suggested that ocular torsion is the main cause of alphabet patterns and overelevation and overdepression in adduction,14 whereas I have felt it is merely contributory. This difference can have important clinical implications. If torsion were the main cause of alphabet patterns, vertical transpositions of the horizontal rectus muscles would be a counterproductive treatment modal-
ity because of its adverse affect on torsion. Surgery primarily designed to correct torsion would be needed. If oblique muscle surgery has either already been performed, or is not clinically indicated, one would need to transpose rectus muscles in the opposite direction than that which would be indicated by the principles shown in Figure 3A. In fact, more 50 years of experience has shown that rectus muscle transposition in the direction that appears to worsen torsion is indeed effective for treating alphabet patterns.2,18 Similarly, if a patient has overelevation or overdepression in adduction, a surgical procedure that is purely aimed at correcting the accompanying torsion may not be effective, as evidenced by the patients in investigation 2 of this study.

The importance of orbital pulley abnormalities is an evolving area in the understanding of alphabet patterns. Although there are patients in whom surgery to address the orbital pulley abnormalities has been successful in treating alphabet patterns, the relative incidence of pulley problems as a cause for pattern strabismus is unclear.9,32 The long and proven history of treating alphabet patterns with standard oblique muscle surgery or horizontal rectus muscle transpositions suggests that pulley problems are not uniformly the cause of pattern strabismus.

It appears that although ocular torsion may contribute to alphabet patterns and overelevation or overdepression in adduction, it is probably not the major cause of these phenomena.

Submitted for Publication: August 25, 2009; final revision received November 3, 2009; accepted November 4, 2009.

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Financial Disclosure: None reported.

Funding/Sponsor: This work was supported in part by an unrestricted grant from Research to Prevent Blindness, Inc, New York, New York.

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