Traction Testing in Superior Oblique Palsy

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ABSTRACT
This report describes the use of superior oblique traction testing in the evaluation of superior oblique palsy. Four consecutive children presenting with signs and symptoms of superior oblique palsy were found to have markedly abnormal tendon laxity with traction testing. Surgical exploration revealed anomalies including three with elongated lax tendons, one of which had an anomalous insertion, and one absent tendon. This tendon laxity is not typically found in acquired superior oblique palsy. It is proposed that, in addition to the well known neurogenic paresis/paralysis, a second type of "palsy" due to anatomic abnormalities of the tendon may exist and be responsible for a number of congenital superior oblique palsy. The mechanical disadvantage of these markedly long tendons may explain why inferior oblique weakening procedures alone frequently fail to correct head tilts due to superior oblique palsy in infancy.

INTRODUCTION
The passive duction or traction test to determine the relative stiffness of ocular movement has been applied widely to the rectus muscles. Superior oblique tendon traction testing has been reported for evaluating Brown's syndrome,\(^1\) for confirming the effectiveness of superior oblique tenotomy,\(^2\) for grading degrees of superior oblique tightness in cases with overaction of the superior oblique,\(^3\) and in assessing intraoperative tendon tuck tightness.\(^4\) This report describes a new application for superior oblique traction testing which employs a modified oblique traction test. This test has significant implications in diagnosing tendon laxity in superior oblique palsy.

TECHNIQUE
The technique of superior oblique traction testing described here is a modification of the "exaggerated" test described by Guyton. Two forceps are used to grasp the perilimbal tissue obliquely, eg, at the 2 o'clock and 8 o'clock position on the left eye or 10 o'clock and 4 o'clock position right eye, as the initial maneuver (Fig 1). The eye is then rotated upward and inward while the globe is repelled (Fig 2). Special care must be taken to grasp episcleral tissue in adults as the thin conjunctiva is easily torn in older persons. Small-toothed forceps work well. The endpoint is reached when a restrictive band produced by the taut superior oblique tendon is felt (Fig 3). The eye can be rocked back and forth over this taut tendon, a maneuver which helps delineate the tendon (Fig 4). The test is carried out in both eyes and the relative tautness of the tendon and, therefore, the degree to which the eye can be repelled in the elevated, adducted position is compared. Experience gained by carrying out this test repeatedly on normal patients will give the examiner a "feel" for the normal tendon tension. Since a positive test (for lax tendon) is usually unilateral, the fellow eye serves as a control and therefore interpretation of the test is straightforward. Visual comparison is also possible since the cornea remains visible to varying degrees, but it is the feel of the tendon which is most significant.
FIGURE 1: Traction test demonstrated on Patient #3. Forceps placed obliquely at the limbus to begin traction test.

FIGURE 2: The eye is moved up, in and retropulsed.

FIGURE 3: Photo shows the difference can be great enough to be visually as well as tactilly significant (compare with Fig 6).

FIGURE 4: The endpoint comes when the taut band of the fully stretched tendon prevents further movement.

CASE REPORTS

Case 1, RT: This 11-month-old white male presented for evaluation of right head tilt and "left eye rolling up." Examination showed a large right head tilt, large left inferior oblique overaction, left superior oblique underaction and positive Bielschowsky head tilt. Refraction was +2.00 both eyes and the fundus was normal.

The right superior oblique traction test was normal, as were the traction tests of both inferior obliques. The superior oblique traction test of the left eye demonstrated marked laxity, in fact no definite tendon was felt.

At surgery, absence of any superior oblique tendon was diagnosed after meticulous exploration of the superior half of the globe from the superior rectus insertion back past the equator including medially and laterally to the superior borders of the horizontal recti. This was confirmed by a second experienced strabismologist. The superior rectus was detached during the course of exploration to facilitate exposure.

A left inferior oblique myectomy was done and in addition a 4 mm left superior rectus recession was performed instead of the planned left superior oblique tuck. At 8
months postoperative, the infant was orthophoric in primary position with a straight head position.

Case 2, KH: A 5-year-old white male had a history of the left eye "going up" since age 2. Visual acuity was OD 20/25, OS 20/30. The patient demonstrated 400 seconds of arc stereo acuity. There was marked left inferior oblique overaction and a positive Bielschowsky head tilt test. Left hypertropia in the primary position measured 14Δ at distance and near with the alternate cover test. Refraction and fundus exam were unremarkable except for exotropia of the left fundus.

The superior oblique traction test of the left eye revealed marked laxity as evidenced by absence of the usual superior oblique resistance. The left inferior oblique and right superior oblique traction tests were normal. Surgical findings demonstrated an abnormally long, limp superior oblique tendon which inserted anomalously on the nasal side of the superior rectus (Fig 5). The tendon was resected approximately 8 mm and advanced to the temporal side of the superior rectus, and the left inferior oblique was weakened. At 6 months postoperative, the patient had a 4Δ right hypertropia (RHT) in primary position, a mild Brown's of the left eye and a small left head tilt.

Case 3, JC: A 4-year-old white female was noted to have a left hypertropia for several years. Old photographs confirmed a left head tilt, however the parents stated that she had been intermittently giving up the head posture over the past several months.

Exam showed OD 20/30 vision, OS 20/40. There was a comitant 12Δ esotropia (ET) and a 16Δ right hypertropia which was slightly greater on left gaze and increased to 25Δ on right head tilt. A mild dissociated vertical deviation was noted in each eye, but greater OD. An overaction of +3 of the right inferior oblique was apparent.

Traction testing at surgery showed marked laxity of the right superior oblique compared to the normal resistance found on testing the left superior oblique (Fig 6). The right superior oblique was isolated and found to be unusually long and floppy (Fig 7), however it was not tucked. The right inferior oblique was recessed 14 mm.

When seen at 5 months postoperatively, a residual right hypertropia measured 5Δ and head tilt test was still markedly positive. A dissociated vertical deviation response of the left eye and the preoperative esotropia were still present.

Case 4, NS: A 4-year-old white male was evaluated for left head tilt which had been present since birth. He had undergone left inferior oblique myectomy 2 years previously. Exam showed RHT 14 and ET 16, a positive Bielschowsky head tilt test and mild underaction of the right superior oblique. There was no inferior oblique overaction. Visual acuity was 20/30 in each eye.

Traction testing at surgery showed marked laxity of the right superior oblique in contrast to the normal resistance of the left superior oblique. The right superior oblique was found to be abnormally long and was tucked approximately 18 mm, which made the traction test similar to the normal left superior oblique. This amount was chosen to prevent a postoperative Brown's syndrome. The left inferior rectus was recessed 4 mm. In addition, the left superior oblique was explored, and found to be normal, that is, lacking the redundancy found on the right.

Two months postoperatively, there was no vertical deviation, however the esotropia remained. Versions were normal including right superior oblique action.
DISCUSSION

When the examiner becomes familiar with the superior oblique traction test, all normal and tight superior oblique tendons can be easily felt. In each of the four cases presented here one eye could be rotated up and in and retroposited to a considerably greater degree than is possible in the normal situation. Each was markedly different than the fellow eye.

The superior oblique traction test has several important implications. First, and of prime clinical relevance, this test alerts the surgeon of the likelihood of encountering an anomalously inserted, lax or absent superior oblique. With a positive test for laxity, the surgical approach may be modified so as to maximize exposure of the tendon.

It has been the experience of several surgeons that infant head tilt due to superior oblique palsy frequently persists unless a tendon tuck (or resection) is done (Parks MM. January 1988. Personal communication). Reynolds, Biglan and Hiles reported that 6 of 13 infants with unilateral superior oblique palsy failed to respond adequately to an inferior oblique weakening procedure. Case 4 is an example of this. This child had a persistent head tilt after inferior oblique myectomy. Versions showed mild underaction of the right superior oblique with no overaction of the myectomized right inferior oblique. Traction testing and surgical exploration revealed the markedly lax right superior oblique.

It is proposed that the reason for this failure to correct early onset head tilts is that many patients with congenital superior oblique palsies have long, anomalous, or infrequently, absent superior oblique tendons. The longer trochlea to insertion distance of the redundant tendon places it at a mechanical disadvantage that, even if normally innervated, cannot be overcome by antagonist muscle weakening. A tendon shortening procedure is recommended for these infants with markedly long tendons as at least part of the surgical repair. This can be done objectively using the traction test as a guide, and can help overcome the inability to accurately quantify prism measurements in diagnostic positions and therefore yolk muscle surgery in the infant age group. Traction test findings may help determine which tendons will require a strengthening procedure.

This abnormal traction test finding is not present in all superior oblique palsy patients. It has been my experience that acquired cases of superior oblique palsy in adult patients typically show symmetric superior oblique tightness and tendon length (D.A.P., unpublished data, 1989). This is true even in long standing cases which makes it less likely that tendon laxity observed in congenital cases is caused by denervation as opposed to true anatomic abnormality.

This finding may explain—and help avoid the commonly reported complication of iatrogenic Brown's syndrome after superior oblique strengthening procedures. These reports and observations of others have demonstrated that some patients receiving large tendon tucks do not acquire a Brown's syndrome, while others undergoing relatively small tucks do suffer this complication. It is hypothesized that this apparent paradox is explained by the differing lengths of the "palsied" tendons. That is, placing even a small tuck in a normal length tendon may cause an iatrogenic Brown's syndrome. This can perhaps be avoided by noting the tendon length status with the traction test. In all but the very lax tendon cases, attention should instead be focused on antagonist or yoke muscle weakening.

A large group of superior oblique palsy patients fall between the extremes of clearly congenital and clearly acquired. Some of these patients are thought to have controlled mild congenital superior oblique palsies that gradually break down to symptomatic problems. Traction testing may help to determine the need for superior oblique exploration and possible shortening procedure in these patients, or just as importantly, the advisability of avoiding a tendon-shortening procedure.

More patient evaluations are needed, but early observations are that these patients with later onset symptoms of congenital superior oblique palsy have mild degrees of tendon laxity on traction testing, compared to the marked laxity found in earlier onset (infantile or early childhood) palsies.

It is hypothesized, though certainly not proven, that there may be two types of superior oblique palsy. One is the well-established neurogenic palsy due to trauma, compression, vascular compromise, or agenesis of the trochlear nerve. The second is "palsy" due to anatomic
anomaly of the superior oblique tendon which can range from abnormal tendon length with or without anomalous insertion to complete absence.

REFERENCES