When and how to strengthen the superior oblique muscle

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PURPOSE
To review the history of procedures used to strengthen the action of the superior oblique (SO) muscle and methods of quantifying surgical dosage and to determine the relationship between congenital onset and tendon laxity measured at the time of surgery.

METHODS
We reviewed medical records over a 10-year period of 30 patients who had undergone SO tendon tuck for SO muscle palsy using intraoperative assessment of forced ductions to determine surgical dosage. We also designed and tested a modified Bishop tucker, which can simultaneously measure tendon shortening and the force required. This allowed development of length tension curves between 0 and 200 g for individual SO tendons in patients with and without evidence of muscle palsy.

RESULTS
In distinction to most other procedures on the extraocular muscles, intraoperative forced ductions are used to determine appropriate surgical dosage. Patients undergoing SO tendon tuck using a uniform and repeatable forced duction method receive a greater amount of tuck (mean, 3 mm) when there is known congenital onset. Patients with congenital SO muscle palsy received a mean tuck of 10.8 mm (range, 8-16 mm), whereas patients with equivocal or known adult-onset SO palsy received a mean of 7.8 mm (range, 4-12 mm; \( p = 0.002 \)).

CONCLUSIONS
Patients with congenital SO muscle palsy have increased tendon laxity when measured directly during SO tendon tuck. The excursion of presumed normal SO tendons through the trochlea is variable and may be less than previously thought. Modifying the Bishop tucker to provide length and tension data provides information that may be useful in determining surgical dosage. (J AAPOS 2009;13:430-437)

Surgery to weaken or strengthen the action of the superior oblique (SO) muscle has been performed routinely for nearly 50 years and remains one of the more difficult and controversial aspects of strabismus surgery. There are several reported methods of strengthening the SO muscle, all of which rely on operating on the reflected tendon (e-Supplement 1, available at jaapos.org). Because of the orientation of the trochlea medial and anterior to the SO tendon insertion, the relative proportion of vertical, torsional, and horizontal forces induced on the globe are complex and depend greatly on the direction of gaze. Strengthening procedures have multiple effects on ocular rotations which can be modified depending on exactly how the SO tendon is repositioned. Finally, shortening the SO tendon may produce an unacceptable limitation of elevation in adduction (iatrogenic Brown syndrome) in a substantial proportion of cases.1,2 For this reason many surgeons have avoided SO tendon strengthening procedures altogether. It is this unusual anatomy, complex physiology, and unpredictable surgical outcome that undoubtedly led von Graefe to his famous and often repeated admonition (“Noli me tangere”) that surgery on the SO muscle should not be attempted.3

Even as late as 1928, Banister4 stated, “Owing to the anatomical relations of the superior and inferior oblique muscles to the eyeball, their deep location in the orbit, and their attachments to the globe in the neighborhood of the equator… advancement or shortening by tucking, is out of the question … and the ophthalmic surgeon must make use of other muscles.” However, by that time Wheeler2 had already shown that direct surgery on the SO tendon could be performed to strengthen the action of an underacting SO muscle. Wheeler’s operation, described as a tendon advancement, was actually more of a plication (folding), with a tendon loop anchored to the sclera temporal and posterior to the insertion, and was performed after disinsertion.
of the superior rectus (SR) muscle. In 1946 Foster\textsuperscript{5} reported a tendon “reefing” procedure, a plication attached at the tendon insertion, which he suggested was more physiologic. McLean modified these techniques by sewing the tendon to itself as well as the sclera,\textsuperscript{6} describing what amounts to a combined SO tendon tuck and plication. He importantly advocated a temporal approach to the tendon, thereby avoiding disinsertion of the SR muscle. McGuire reported success using both SO resection and tuck,\textsuperscript{7,8} but apparently preferred the former procedure.\textsuperscript{8}

In 1966 Dyer\textsuperscript{9} described and illustrated the SO tuck procedure, essentially as it is performed today, using a single, nonabsorbable suture to fold the tendon near the insertion. Despite the above reports, prior to 1970 about the only time an ophthalmologist saw the SO tendon was during an enucleation (Eugene Helveston, personal communication, October 14, 2008).

As skepticism regarding SO muscle strengthening receded, the role of SO tendon tuck became better established, at least in certain clinical situations.\textsuperscript{10-18} The tuck has proved to be convenient to perform and generally reversible simply by removing the nonabsorbable suture. Most importantly, it leaves the scleral insertion of the SO tendon unmolested, with the theoretic advantage that any increased forces applied to the globe from tendon tucking would be generated in the same proportion they were lost due to the paresis. Thus the vertical, torsional, and horizontal correction would occur as needed, and the only required decisions would relate to surgical dosing and whether to operate additional extraocular muscles.

SO tendon tuck figured prominently in Knapp’s treatment paradigm in his well-known classification and treatment recommendations for SO muscle palsy, presented in 1970 as the First Richard C. Scobee Memorial Lecture, “Diagnosis and surgical treatment of hypertropia,” to the American Association of Certified Orthoptists in Las Vegas, NV.\textsuperscript{11} Variations have been described by Harada and Ito\textsuperscript{16} and others\textsuperscript{10-22} to increase the relative torsional correction by redirecting and advancing the anterior tendon fibers. However, SO tuck performed temporal to the SR muscle remains the simplest and most useful operation in the surgeon’s armamentarium to strengthen the action of an underacting SO muscle, particularly in the context of SO muscle palsy. Despite better accessibility, tendon shortening \textit{medial} to the SR muscle has not generally been advocated, perhaps because of proximity to the trochlea and the risk of producing iatrogenic Brown syndrome postoperatively. Our knowledge has been greatly advanced by anatomic and clinical studies by Parks,\textsuperscript{5} Helveston and coworkers,\textsuperscript{10,23,24} and others.\textsuperscript{25-32}

\textbf{Subjects and Methods}

\textbf{The Lax Tendon and Congenital Onset}

A retrospective database review was performed over two 5-year periods (1980–1985 and 2003–2008) for patients undergoing SO tendon tuck for SO muscle palsy at the Medical University of South Carolina. These time periods were chosen because of the availability of clinical data, which had been lost for the intervening years. Patients with onset of strabismus or torticollis during early childhood and no apparent cause, or those with characteristic plagiocephaly\textsuperscript{32} and later onset of symptoms, were deemed to be of “congenital” etiology. The remaining patients were classified as either “posttraumatic” or “undetermined,” depending on clinical circumstances. The presence or absence of a clinically lax tendon at the time of surgery was not used in assigning etiology. The amount of tendon tuck was determined in a similar manner as all cases. A Bishop tendon tucker was used to shorten the tendon after the entire SO tendon was isolated temporal to the SR on a Stevens muscle hook (e-Supplement 2, available at jaapos.org). Once a provisional tuck was secured using a 5-0 braided Dacron suture, a silk traction suture was placed through the loop for easy retrieval, and the tendon released. Forced ductions were then performed in a manner previously described\textsuperscript{12,34} to assess the limitation of elevation in abduction generated by the shortened tendon. An adequate tuck was judged to have been achieved when the first resistance to elevation in abduction was noted as the inferior limbus crossed an imaginary line between the medial and lateral canthus (Figure 1). Tucks judged too tight or too loose were adjusted appropriately (Figure 2). Care was taken not to retropulse the globe when performing forced ductions, because this would put the tendon on stretch and alter the result. The final endpoint was sufficient to remove all tendon laxity and generate an approximately –2 elevation deficiency (scale, 0 to –4, where 0 = full elevation and –4 = no elevation) in abduction, postoperatively. Data were analyzed with regard to amount of tuck, congenital vs noncongenital onset, and preoperative hyperdeviation.

\textbf{Studies Involving a Modified Bishop Tucker}

In 2005 we modified a Bishop tucker to allow the addition of an externally mounted strain gauge. The new instrument allowed simultaneous determination of the amount of SO tendon shortening and the force required to achieve this shortening. Early prototypes proved impractical, because of instrument size (Figure 3) and the use of different materials (stainless steel and aluminum), which required different methods of sterilization. We subsequently enlisted the help of Bausch and Lomb (St. Louis, MO), who produced an improved instrument, similar in size to a currently marketed Bishop tucker but with an \textit{internal} strain gauge that was more practical to use. The final version had strain gauge calibration between 0 and 200 g, in 50 g increments, which could be read through a cutout in the center of the shaft (Figure 4). After obtaining approval from the institutional Human Research Office, selected patients undergoing surgery on the SO tendon (typically posterior tenectomy or tendon tuck) had length-tension measurements taken during the procedure from 0 g to 200 g of force, which was the limit of the instrument.

\textbf{Results}

Medical records from 30 patients were found complete and analyzed (e-Supplement 3, available at jaapos.org). All had a diagnosis of SO muscle palsy (supported using Parks 3-step
and had SO tendon tuck performed using a Bishop tucker as part of their surgical procedure. As a general rule, patients with $\geq 30^\circ$ of hypertropia in extreme lateral gaze opposite the side of paretic muscle, or with $\geq 20^\circ$ in primary gaze position, had a second muscle operated—usually a weakening of the ipsilateral inferior oblique (IO) or contralateral inferior rectus muscle. These were selected based on prism-and-cover measurements (or estimates) in the diagnostic gaze positions, although treatment strategies recommended by Knapp\textsuperscript{11} were not necessarily followed (eg, contralateral SO tendon tenotomy in class 5 [so-called “double-depressor”] SO muscle palsy).

The 15 patients with congenital SO muscle palsy received a mean tuck of 10.8 mm (range, 8-16 mm), while the 15 patients with equivocal or known adult-onset SO palsy received a mean tuck of 7.8 mm (range, 4-12 mm) ($p = 0.002$), indicating greater tendon laxity in the congenital group. Among these 30 patients, the largest tuck performed was 16 mm; the smallest, 4 mm. A modest correlation was found between the amount of tuck and the preoperative hyperdeviation, both in primary gaze position ($r = 0.30$) and in greatest measured deviation in any diagnostic gaze position ($r = 0.32$). There were no complications referable to using the modified tucker or measurements of tendon laxity.

Data from 12 subjects from our institution were collected during SO muscle surgery to test the feasibility of generating length-tension curves for the SO muscle, with and without SO palsy, using the modified Bishop tucker.

![FIG 1.](image1) Drawing of the intraoperative traction test for titrating a superior oblique tendon tuck (left eye shown). A. The eye is grasped near the limbus at approximately 5:30 o’clock with toothed forceps. B. The globe is positioned in 30°-40° adduction. C. The globe is then elevated against the superior oblique tendon without retropulsion. The first resistance to elevation should be felt as the inferior limbus crosses an imaginary line (dashed line) between the medial and lateral canthus. 

![FIG 2.](image2) Simple technique of adjusting a tuck judged too loose or too tight. A. An excessive tuck can be loosened by pulling the looped tendon apart with a muscle hook while simultaneously working the provisionally tied nonabsorbable (eg, 5-0 braided Dacron) suture away from the sclera with a needle driver. Once adjusted, the tendon is then secured tightly with a second wrap of suture around and through the tendon. A silk traction suture is shown through the tendon loop and allows easy release and recovery from the orbit. B. An inadequate tuck is tightened by placing a second wrap of suture around and through the tendon closer to the sclera. The original suture is not removed. Drawings courtesy of Kevin Rockwell.
To stabilize the globe during measurements, it was found helpful to use forceps to position the globe in depression, with the superior limbus at the margin of the lower eyelid. Progressively greater forces could be demonstrated in all cases as the tucker tightened the tendon. This progression occurred regardless of whether the patient had mild SO.

**FIG 3.** Prototype instrument consisting of a modified Bishop tucker attached to an aluminum hand piece. A commercially manufactured spring-scale connects to the tucker shaft, which has been modified to allow free movement of the tendon hook. A, Component parts. B, Assembled instrument.

**FIG 4.** A, Integrated surgical instrument based on a commercially available Bishop tucker (Storz Instruments, St. Louis, MO). B, The amount of tendon shortening (in mm) is indicated in the usual fashion on the side of the shaft. However, an internal strain gauge, read through the window, provides the amount of force (g) required to displace the tendon.
muscle overaction or SO muscle palsy (Figure 5). Measurements were reasonably repeatable. Retesting, when performed, always yielded a tendon laxity ± 2 mm of the original value at a given amount of force, 0, 50, 100, 150, and 200 g.

Discussion

While patients with the clinical appearance of SO palsy may have multiple causes, most appear associated with dysfunction of the fourth (Trochlear) cranial nerve. With few exceptions, the lax tendon observed clinically and muscle atrophy with reduced contractility reported on orbital imaging can adequately explain the pathophysiology of this disorder. Alternative hypotheses, such as those involving alteration in orbital “pulleys,” cannot be used to explain posttraumatic or surgically induced cases and would have to account for the greater tendon laxity we typically find in presumed congenital onset. It remains clinically reasonable, therefore, that surgical interventions be designed assuming we are treating strabismus produced by weakness of the SO muscle in the majority of cases. While increased tendon laxity appears to occur in cases of congenital onset SO muscle palsy, and a clinical grading system described, more rigorous documentation has been lacking.

We have shown that patients with congenital onset SO muscle palsy do have, on average, greater tendon laxity than those of acquired or undetermined onset by approximately 3 mm. However, the variation in tendon tuck was large (8–16 mm) and some patients with known posttraumatic SO muscle palsy received tucks as large as 12 mm. While very redundant or otherwise anomalous (including absent) tendons will occasionally be encountered, such cases do not necessarily support the concept that excessive tendon laxity per se is ever the primary disorder in these patients.

Surgery to correct hypertropia and torticollis in patients with congenital SO muscle palsy is the most common indication for SO muscle strengthening. Other less common indications are acquired SO muscle palsy with marked underdepression in adduction, or torsional diplopia, typically found in acquired, bilateral SO muscle palsy. SO muscle strengthening may occasionally be appropriate in the treatment of residual IO overaction in patients with a V-pattern strabismus of presumed nonparetic etiology after IO weakening. However, little is known about its effectiveness in this clinical context.

Congenital SO muscle palsy responds well to SO muscle strengthening and SO tendon tuck is the easiest way to do it. A tuck is occasionally performed as an isolated procedure but is most useful when the hypertropia is large and combined with ipsilateral IO muscle weakening. Congenital palsies are characterized by excellent vertical vergence amplitudes, which may render phoric a 20Δ or 25Δ (or greater) hyperdeviation in primary gaze position. Acquired SO muscle palsy, in contrast, often presents with a hypertropia of lesser magnitude, typically with poor vertical vergence amplitudes. Barring profound underdepression in adduction, these cases are preferably managed by single-muscle surgery not involving the SO muscle. Surgery to weaken the ipsilateral IO, contralateral inferior rectus, or, occasionally, the ipsilateral SR muscle, is reasonably predictable and may be suited to adjustable suture techniques. Importantly, they do not require the more difficult titration of a SO tendon tuck in a “non-lax” tendon. Ironically, the only tuckakedown in my 30 years of clinical practice (with well over 100 SO tucks under multiple clinical circumstances) was in a patient with the least amount of tuck I ever performed (4 mm). This patient had unilateral posttraumatic SO palsy. Her surgical overcorrection and postoperative Brown syndrome may have been related to shortening.

a relatively tight tendon, even in the context of known muscle paresis. Also contributing to her poor result was the decision to perform simultaneously an ipsilateral IO recession. This was done despite the fact that her greatest hypertropia in any gaze position did not exceed 25°. I would manage such a patient differently today.

Only 2 other patients have had clinically important overcorrections following SO tendon tuck during this 3-decade period. One had congenital SO muscle palsy with 40° of sidegaze hypertropia preoperatively. An initial 15° of hypertropia in primary gaze position postoperatively spontaneously improved to orthophoria over several years and the patient became asymptomatic. The other patient had postruclamatic SO muscle palsy and was left with −3 elevation deficiency (due to Brown Syndrome) and diplopia in adduction postoperatively, which persisted for 7 years. However, he was well-aligned in primary gaze position and tuck takedown was repeatedly refused.

Because of the difficulty in quantitating surgical dosage, I reserve SO strengthening procedures directed primarily at torsional correction (such as the Harada-Ito procedure) to cases where hypertropia is small and there is symptomatic extorsion (typically ≥15°). However, a detailed discussion of this technique is a separate subject and beyond the scope of this article.

Surgery to strengthen the SO muscle is unique in that surgical dosing is determined almost exclusively on the basis of intraoperative findings. Previous authors have found no relationship between the amount of tuck performed and correction of vertical deviation in primary gaze position. This is true regardless of whether SO tendon tuck is performed in isolation or combined with surgery on other extraocular muscles. However, the amount of tuck appears to correlate with the greatest hypertropia measured preoperatively in sidegaze opposite the paretic muscle, at least in patients with congenital, large-angle vertical deviations. When using a resistance endpoint, the surgical dose presumably serves as a proxy for tendon laxity. Relying on reported mean dosages as a treatment guide in individual patients will therefore produce under- and overcorrection if tendon laxity is not taken into consideration.

It is also not widely appreciated that the best guide in selecting a surgical dosage (eg, 1 vs 2 muscles) may be the diagnostic position of gaze with greatest hypertropia, rather than primary gaze position. Knapp used 25° as the dividing point in recommending 2-muscle surgery. However, he did not specify whether this was primary position deviation or the greatest measured deviation. In a subsequent publication Knapp and Moore alluded to using “large deviations, over 30° in the paretic field” as an indication for multiple muscle surgery but did not explicitly state this principle. Even with fusion suspended, primary position hyperdeviations in congenital palsies may occasionally be small, especially at near viewing, while sidegaze hypertropia commonly exceed 30° or occasionally 40° (e-Supplement 3). In our experience, surgical treatment will not generally be curative in such cases unless 2-muscle surgery is performed. Conversely, patients achieving satisfactory postoperative alignment in sidegaze will almost always have their hypertropia resolved in primary gaze position as well. Alternatively stated, if you target the surgery to fix the sidegaze hypertropia, primary position will take care of itself.

The desirability of using intraoperative assessment when performing SO tendon tuck was actually suggested by Knapp in his 1970 Scobee lecture. After engaging the SO tendon temporal to the SR muscle, he recommended the Bishop tucker be tightened until “snug” prior to placement of the suture. I observed him perform SO tendon tucks several times as an ophthalmology resident during the mid-1970s, but was never clear on exactly how he decided on final surgical dosage. Scott subsequently recommended achieving a “moderate” Brown syndrome, although further guidance was again not provided. Helveston and Ellis used an estimate of tendon laxity to determine the amount of tuck performed, but they experienced a 17% rate of overcorrection requiring tuck takedown. Presumably, there were additional patients who were overcorrected, but not sufficiently to warrant additional surgery. The mean amount of tuck in this series of 59 patients with both congenital and acquired SO muscle palsy was 12 mm, which is similar to the mean amount of tuck we performed in our congenital onset patients (10.8 mm).

An alternative method advocated for unilateral cases with SO tendon laxity is to perform sufficient tuck to equalize the forced ductions with the presumed normal eye (David Plager, personal communication, November 11, 2002), provided ipsilateral IO weakening is also performed. However, surgical success rates have not been reported using this “matching” technique. One might speculate it could lead to frequent undercorrection if the SO tendon were not under sufficient tension to augment the action of the paretic SO muscle. Tendon tucking can be also performed with a Fink tucker or just a Stevens muscle hook but is more difficult to titrate.

In 1985 we described a technique to make endpoint determination more uniform based on position of the inferior limbus during intraoperative forced duction testing. Tendon tightness (Brown syndrome) is assessed as the globe is moved into an elevated position in adduction. The desired tuck is produced when the first resistance to elevation occurs as the inferior limbus crosses an imaginary line drawn between the medial and lateral canthus (Figures 1 and 2). This technique is easy to perform and reliable and has been successfully employed by others in both congenital and acquired SO muscle palsy.

While the choice and number of muscles to be operated is best guided by the magnitude of the hypertropia (particularly in the field of greatest hyperdeviation), the amount of SO tendon shortening is not. This near-total reliance on traction testing is an anomaly in modern strabismus surgery, where our textbooks are replete with dosage tables to guide weakening or strengthening of the horizontal rectus
muscles, vertical rectus muscles, and to some degree the IO muscle. Understanding this is necessary for avoiding poor outcomes. The reason for this difference in technique is not entirely clear, but it probably relates to (1) the presence of paresis (as opposed to paralysis) in most cases of clinically diagnosed SO palsy and (2) the unique structure of the trochlea, which rapidly becomes unyielding when the SO tendon is excessively shortened. Getting the best surgical outcome is therefore something of an art—perhaps more so than is true of more routine strabismus surgery. It is interesting, however, that the mean amount of tuck we performed in noncongenital cases (7.8 mm) correlates closely with the estimated upgaze excursion (8 mm) in a normal trochlea.24 Because our method yields a modest upgaze restriction in adduction (Brown syndrome) at the conclusion of surgery, the forward excursion of the tendon through the trochlea may be less than previously thought. In this group of presumed normal tendons, tucks ranged from 4 to 12 mm; 6 of 15 (40%) were less than 8 mm, indicating the forward excursion of the tendon through the trochlea may often be less than mean values. This further substantiates the need for tuck titration in this group of patients.

It is also worth noting that mild-to-moderate postoperative Brown syndrome is not undesired. Rather, it appears necessary in most cases to achieve satisfactory long-term outcome. Most patients have some permanent deficit in elevation after SO tuck but are minimally symptomatic and report diplopia only in extreme upgaze. Unless worse than –2, this should not generally be viewed as a complication. The need for temporary overcorrection in other forms of strabismus surgery, such as primary intermittent exotropia, is also well recognized.19,20 Except when the SO tuck is truly excessive, the postoperative Brown syndrome is clinically innocuous and improves or resolves with time (e-Supplement 4, available at jaapos.org). However, in cases where profound IO weakness (such as myectomy) is also performed, some surgeons (David Plager, personal communication, November 16, 2008) prefer to reduce slightly the amount of tuck to lessen the Brown syndrome postoperatively. Such an approach is clinically reasonable, because long-term undercorrection is easier to manage than long-term overcorrection, which invariably requires reoperation.

Our modified Bishop tucker provides a tool to further study properties of the SO muscle and tendon, both in presumed normal individuals and in patients with SO muscle palsy. Because the former group had no more than mild SO “overaction,” we believe these data will be useful to establish normal or near normal values for SO tendon laxity. We plan to report additional findings in the future.

Finally, this or similar instruments may also be useful in determining surgical dosage when SO tuck is performed. While intraoperative forced ductions seem effective and can be titrated to a defined endpoint, this is still a subjective assessment that many surgeons do not feel comfortable making. While I have experienced almost no long-term overcorrections, long-term undercorrections are not uncommon. It is possible that shortening the SO tendon to a predetermined tension endpoint may be more successful than using a forced duction end point. The target tension might prove to be less when there is simultaneous weakening of the IO muscle or more when SO tendon tuck is performed alone. It may also be possible to predict, based on tension measurements, which patients have increased risk for long-term surgical failure or unacceptable postoperative Brown syndrome.

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References