

## SURGICAL TREATMENT OF SUPERIOR OBLIQUE PALSY\*

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### INTRODUCTION

Superior oblique palsy (SOP) is the most common isolated cranial nerve palsy seen by the strabismologist. Reports of several series, some of which have described more than 200 patients, were published between 1971 and 1986. Studies by Knapp,<sup>1</sup> Ellis and Helveston,<sup>2</sup> Scott and Kraft,<sup>3</sup> and von Noorden and associates<sup>4</sup> provided important information about classification, the influence of torsion, and the effects of specific muscle surgery in dealing with the complicated strabismus presented by superior oblique palsy.

In the past decade, new information has appeared describing additional characteristics of the superior oblique muscle and tendon and uncovering newly understood characteristics of patients with superior oblique palsy.<sup>5-12</sup> The anatomy and physiology of the trochlea is now well understood, particularly with regard to the limited excursion of the superior oblique tendon through the trochlea, which in turn limits the amount of superior oblique tendon that can be tucked safely without creating a troublesome Brown syndrome.<sup>7,13</sup> Beginning with a description of absence of the superior oblique tendon in a small number of otherwise normal patients with superior oblique palsy, a systematic classification of tendon anomalies in patients with congenital SOP has evolved.<sup>5-7</sup> This has been followed by detailed study of the superior oblique muscle utilizing magnetic resonance imaging (MRI), both static and cine, and by a resurgence in interest in computer modeling as a means of studying the superior oblique in the physiologic and diseased states.<sup>9,10,14</sup>

The superior oblique traction test, both to assess tightness of the superior oblique muscle after tucking and to assess laxity before perform-

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ing surgery, has provided important information about the behavior of the superior oblique muscle, particularly its behavior distal to its passage through the trochlea.<sup>15-17</sup>

The association of facial asymmetry with the head tilt occurring in patients with congenital superior oblique palsy has aided in establishing the etiology as congenital in many cases, which may have been placed in the "unknown" category in earlier studies.<sup>2,11,12</sup>

This report describes our experience in the last 5 years with diagnosis and treatment of superior oblique palsy. We used new information about variations in the superior oblique tendon to aid in diagnosis and planning of treatment. We used a surgical scheme tailored specifically to the needs of each patient, employing inferior oblique weakening in most patients, performing vertical and horizontal recession as needed, and limiting strengthening surgery of the superior oblique to lax tendons, and the Harada Ito procedure to patients whose main symptom was torsion. The results of our diagnosis and treatment in this series are presented and compared selectively with previous studies.

#### METHODS

This study represents a retrospective review of 190 patients surgically treated by us for the diagnosis of superior oblique palsy between January 1990 and December 1995. All patients who underwent one or more surgical procedures for SOP were included in the study population. The study population consisted of 105 males and 85 females with ages ranging from 6 months to 79 years (mean, 28.8 years). Of these, 181 patients were available for postoperative evaluation (95.3%).

A history was recorded for all patients included in the study. The symptoms that prompted the patient to seek medical attention were recorded and included diplopia, strabismus, head tilt or turn, and asthenopia. The duration of the symptoms and probable age at onset were noted. If available, photographic evidence of long-standing head posture was recorded. Nine patients had undergone surgical procedures elsewhere before presentation to us, and in all cases a description of the procedure(s) performed was obtained.

A complete eye examination was performed for all patients with recording of visual acuity, refractive error, and stereo acuity testing (Titmus stereo test), when possible. The presence of any prismatic correction incorporated into the patient's spectacles was recorded. Patients were also examined for the presence and direction of any anomalous head tilt or face turn as well as any notable facial asymmetry. Facial asymmetry was judged to be present if the distance between the corner of the mouth

and the lateral canthus appeared to be asymmetric from left to right and associated with other features of midfacial hypoplasia.<sup>12</sup>

An ocular motility examination and determination of the deviation in primary position and the cardinal positions of gaze were obtained in all cooperative patients using hand-held prisms and cover testing at 33 cm and 6 m. Estimates of the manifest deviation were performed in patients unable to cooperate with cover testing. On the basis of these data, a Knapp classification was assigned to each patient with the addition of class VIII, which represented a comitant vertical deviation with a positive Bielschowsky head tilt test. Double Maddox rod testing was performed in all cooperative patients using a red vertical Maddox rod before the right eye and a white vertical Maddox rod before the left eye in a trial frame.<sup>18</sup> The patient was allowed to adjust the orientation of the Maddox rod to make the lines parallel, and degrees of torsion in the primary position were read directly from the trial frame.

The operative reports and surgeon's notes were reviewed, and all procedures for each patient were recorded. When tested, the results of forced duction testing of the obliques (FDTO) were recorded.<sup>17</sup> Tendon laxity was recorded on a scale of normal to -4 as follows: -1, mild laxity; -2, moderate laxity; -3, severe laxity but tendon present; and -4, no tendon felt on FDTO. Evidence supporting the classification of a case as congenital included clinical history of a head tilt or symptoms dating to early childhood, photographic documentation of the tilt, facial asymmetry, and tendon laxity during FDTO. Acquired cases were supported by the lack of the above findings, normal SO tendons on FDTO, and a clear-cut history of an antecedent event related to the onset of symptoms.

The ophthalmic examination and orthoptic measurements were repeated at each postoperative visit. A cure was defined as resolution of the symptoms that initially prompted the patient to seek medical attention plus elimination of strabismus or head tilt, or reduction of these findings to an insignificant level without the creation of new symptoms.<sup>4</sup> Nine patients were unavailable for postoperative follow-up. The preoperative and operative data for these patients were included in the preoperative patient data but were omitted from the final analysis of outcome.

Data collection was performed in a retrospective manner by 2 observers not responsible for the initial surgery. Standard statistical analysis was performed on all data.

## RESULTS

One hundred ninety patients were included in this series, and preoperative data are given on all these cases; postoperative data were available for

181. The mean follow-up period was 9.7/16.8 months.

Table I presents the patient demographics and some of the preoperative data along with the results of intraoperative superior oblique traction testing. Of the patients with facial asymmetry, all but 5 were classified as having a congenital palsy. Of those for whom the facial symmetry status was documented, 56% of congenital palsy cases had facial asymmetry and only 15% of acquired cases had this finding.

Table II lists the surgical procedures performed and includes all muscles operated on irrespective of whether they were dealt with in 1 operating session or separate sessions.

Table III compares the preoperative and postoperative data on our series of patients. Where it is significant, the number of patients with documented results for the particular test are noted.

Preoperatively, 44 of 190 patients (23%) wore a corrective prism lens in their glasses. Postoperatively only 9 of 181 (5%) required prisms. Moreover, for those needing prism, the mean power was reduced from 7.8 to 2.2 diopters. Three patients required horizontal prism correction both before and after surgery, but the mean power was reduced from 6.3 to 2.7 diopters.

Vertical prism and cover testing showed that preoperatively only 14 of 191 (7%) had no vertical deviation in the primary position, while postoperatively that figure improved to 138 of 181 (76%). The mean and median hyperopia of those who had deviations was reduced.

The number of patients with a horizontal deviation was halved from 69 to 35. Of the 35 with a postoperative horizontal deviation, 12 developed over the period of treatment, while 23 were residual deviations from those existing preoperatively. Of the 46 patients whose preoperative horizontal deviation resolved, 8 occurred after horizontal muscle surgery. Fifty-five percent (38/69) of patients had their preoperative horizontal deviation resolve "spontaneously" (ie, after having treatment for only the superior oblique palsy). Forty-five percent of esotropias (9/20) and 75% of exotropias (37/49) resolved spontaneously. Preoperative esotropia was present in 42% of bilateral palsies (8/19) and only 7% of unilateral palsies (12/171).

Preoperatively, 28 of the 123 patients who could be tested for torsion (23%) had no excyclotorsion. Postoperatively, 56% of those tested had no torsion. Many patients did not have their postoperative torsion documented, especially those without complaints.

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**TABLE I: PATIENT DEMOGRAPHICS AND PREOPERATIVE DATA FOR 190 CASES OF SUPERIOR OBLIQUE PALSY**


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Sex	Male: 105		
	Female: 85		
Age	Range: 6 mo-79 yr		
	Mean: 28.8+/-22.2 yr		
	Mean for congenital group: 24.1+/-21.1 yr		
	Mean for acquired group: 40.9+/-20.5 yr		
Refraction	Mean: -0.49+/-3.04 diopters		
Visual Acuity	Mean: 20/25		
	Median: 20/20		
Congenital/acquired	Congenital: 137		
	Acquired: 53		
	Origin: Trauma 29		
	Iatrogenic 12		
	Vascular 7		
	Tumor 5		
Knapp Classifications	Class I: 28		
	Class II: 13		
	Class III: 65		
	Class IV: 53		
	Class V: 5		
	Class VI: 19		
	Class VII: 1		
	Class VIII: 6*		
Laterality	Right: 92		
	Left: 79		
	Bilateral: 19		
Facial asymmetry	Present: 56	51 congenital	5 acquired
	Absent: 69	40 congenital	29 acquired
	Unknown: 65	46 congenital	19 acquired
Abnormal head posture:	Right tilt: 55		
	Left tilt: 70		
	Others (eg, head turn, chin down): 10		
	No abnormal head posture: 39		
	Unknown: 16		
Forced duction tests	Tests performed: 161		
	Tendon laxity: 95	83 congenital	12 acquired
	No tendon laxity: 66	37 congenital	29 acquired

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\*Type VIII = comitant vertical deviation

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**TABLE II: SURGICAL PROCEDURES PERFORMED\***


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Inferior oblique surgery: 177
Myectomies and recessions: 175
Anterior transpositions: 2
Contralateral inferior rectus recession: 36
Ipsilateral superior rectus recession: 32
Superior oblique surgery: 50
Superior oblique tuck: 26
Superior oblique resection: 9
Harada Ito procedure: 15
Horizontal muscle surgery: 19
Horizontal shifts of vertical recti or vertical shifts of horizontal recti: 3
Number of surgeries: †
Mean: 1.26+/-0.61
Range: 1-6

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\* When procedures were performed bilaterally, they have been noted only once.

† Number of times the patient went to the operating room, rather than number of different strabismus procedures performed.

#### DISCUSSION

In this study, the incidence of superior oblique palsy in males is slightly higher than in females (55% versus 45%, respectively). This compares with 63% in males in the study of von Noorden and associates<sup>4</sup> and 68% in the study of Ellis and Helveston.<sup>2</sup> This may reflect the smaller number of patients diagnosed as having acquired traumatic SOP in the present series, 53 of 190 (28%), compared with 137 of 190 (72%) with congenital SOP. Since it has been generally agreed that males are more prone to trauma of the kind that could produce SOP, having fewer patients in the acquired category should result in a relative reduction in males being affected.

The range in age of patients treated covers 8 decades, from 6 months to 79 years, indicating that there is a lifelong potential for this condition occurring and requiring treatment.

In previous studies, a large proportion of cases have been labeled idiopathic: 23% in the study of von Noorden and associates<sup>4</sup> and 48% in the

TABLE III: A COMPARISON OF PREOPERATIVE AND POSTOPERATIVE DATA

	PREOPERATIVELY	POSTOPERATIVELY
Vertical prism correction in spectacles	Present in 44 Range: 2-21 diopters Mean prism: 7.8 diopters	Present in 9 Range: 1-4 diopters Mean prism: 2.2 diopters
Horizontal prism correction in spectacles	Present in 3 Range: 1-12 Mean prism: 6.3 diopters	Present in 3 Range: 2-4 Mean prism: 2.7 diopters
Vertical prism cover test in primary	176 with vertical deviation 14 with no vertical deviation Mean of those with: 13.4 diopters Median of those with: 12 diopters	43 with deviation 138 with none Mean: 5.5 diopters Median: 5 diopters
Horizontal prism cover test in primary	69 with deviation 20 esotropias 49 exotropias	35 with deviation 20 esotropias 15 exotropias
Stereo acuity	155 tested Mean: 420 seconds Median: 140 seconds	131 tested Mean: 340 seconds Median: 50 seconds

TABLE III: (CONTINUED)

Exocyclotorsion	123 tested 28 with no exocyclotorsion Range: bilateral 0-17° unilateral 0-15° Mean: bilateral 11.3° congenital 7.5° acquired 12.1° unilateral 4.6°	57 tested 32 with no exocyclotorsion Range: 0-13° Mean: 4.5°
Abnormal head posture	Present: 135 Absent: 39 Unknown: 16	Present: 39 Absent: 111 Unknown: 31
Principal symptoms	Diplopia: 104 Strabismus: 55 Head tilt: 25 Asthenopia: 6	Diplopia: 5 Strabismus: 1 Head tilt: 4 Asthenopia: 1 Downgaze diplopia: 3 DVD: 1 Asymptomatic: 166



study of Ellis and Helveston.<sup>2</sup> In this series, all patients were given a diagnosis, arbitrarily ruling out the category of "unknown." The 7 cases diagnosed as having a vascular cause consisted of elderly persons who had no features of a congenital palsy and no history of trauma, surgical or otherwise, but who had a history of cardiovascular disease or diabetes. Those patients with acquired SOP from other neurologic causes had a clear-cut supportive history plus a documented sudden onset. Since patients were treated on the basis of clinical findings only rather than etiology, this aspect, while of interest regarding other medical issues, played no role in surgical planning.

We documented the facial asymmetry status in 125 (66%) of our patients.<sup>11,12</sup> Facial asymmetry is characterized by the appearance of a fuller midface on the side away from the head tilt. When the head is tilted, the dependent side of the face becomes smaller, with a reduced distance between the lateral canthus and the corner of the mouth. This facial asymmetry can occur in other cases of head tilt occurring early in life. We believe facial asymmetry is a *result* of the head tilt and not associated with the cause of SOP, because we have seen it disappear when treatment of the strabismus or other causes of head tilt was successful in eliminating the tilt.

In the 65 patients for whom no comment was made regarding facial asymmetry, it is likely that the majority had no asymmetry, as we were aware of the significance of this sign and, presumably, would have noted its presence. If this is the case, then the percentage of cases having asymmetry might be as low as 37% for the congenital group and 10% for the acquired group. This figure is reasonable given our belief that facial asymmetry is a result, not a cause, of superior oblique palsy. The etiology data are biased by the fact that facial asymmetry was one of the criteria we used to classify cases as congenital. One would therefore expect the prevalence of asymmetry to be higher in this group.

Of the 5 acquired palsy patients with facial asymmetry, 3 had a clear history of acute onset following blunt head trauma, 1 occurred following the development of a medulloblastoma, and 1 occurred after orbital surgery. These patients were presumed to have facial asymmetry that was unrelated to their motility problems. The asymmetry did match the side of the muscle palsy, and it is possible, of course, that the other problems caused the breakdown of a preexisting congenital palsy. However, without corroborating evidence of tendon laxity or photographic proof of a long-standing head tilt, we were obliged to classify these cases as acquired superior oblique palsies with coincidental facial asymmetry.

It is apparent that many patients with congenital superior oblique

palsy do not have facial asymmetry, and furthermore, facial asymmetry can coexist coincidentally with acquired palsies. While facial asymmetry has a relatively low sensitivity as a diagnostic tool for congenital superior oblique palsy, it has a high predictive value (51/56, or 91%).

Tendon laxity was demonstrated in 75% of congenital and 30% of acquired palsies.<sup>8</sup> Of the 11 acquired cases, 4 were felt to be bilaterally and symmetrically lax in the face of a unilateral palsy. The other 7 were graded with only mild laxity. One must question whether the apparent laxity was due to variations in orbital anatomy, allowing increased rotation of the eyes during traction testing, rather than being due to a truly redundant tendon. Of 18 unilateral congenital palsies with bilateral tendon laxity, 9 were asymmetric, 2 markedly so, and may have had redundant tendon. However, 9 others were symmetrically lax and perhaps should not have been considered to produce a diagnostically lax traction test. One must always consider the possibility of a masked bilateral palsy, but in the presence of a clearly unilateral case, it may be useful to look for significant asymmetry as well as laxity when performing diagnostic traction tests.<sup>17,19</sup>

The principal role of intraoperative traction testing is to establish whether a superior oblique tuck can be usefully and safely performed or, conversely, should be avoided.<sup>13,16,17</sup> Unless asymmetry is present in addition to laxity in a unilateral case, it is probably not necessary or indeed wise to tuck the tendon.

A concomitant horizontal deviation was present in 36% of superior oblique palsies. Esotropia was particularly common in bilateral palsies, occurring in 42% of cases. Of the unilateral palsies with a horizontal deviation, 75% were exotropias. Seventy-five percent of exotropias and 45% of esotropias resolved "spontaneously," in the sense that the patients had vertical muscle surgery only and yet the horizontal deviation was not present postoperatively. Therefore, the presence of an esotropia in bilateral SOP or any horizontal deviation in unilateral SOP should not be a definite indication for horizontal rectus surgery. If the horizontal deviation is small to moderate, it may be reasonable to perform only vertical muscle surgery with a view to correcting any residual horizontal deviation at a second procedure.

Of the patients with an abnormal head posture postoperatively, 11 were considered slight tilts, while 23 were considered still definitely present and noticeable by us even if they had improved. Four patients reversed the direction of their tilt postoperatively. Two of these patients had superior oblique tucks, and 2 had only inferior oblique myectomies. Interestingly, only 4 of the 23 patients with a persistent head tilt postoperatively still considered it a problem. Typically, these patients presented

initially with diplopia and, having had that problem resolved, they were content to put up with an abnormal head posture without complaint.

The surgical results in this series demonstrate a definite improvement in most patients as measured by several parameters. Postoperatively, 76% of patients showed no vertical deviation in the primary position with cover testing, 79% (111/150 documented) had no abnormal head posture, and 96% had no diplopia in either the primary or reading positions. The median stereo acuity improved from 140 seconds to 50 seconds. Most important, 166 patients (92%) had resolution of the symptoms that led them to present for diagnosis and treatment in the first place.

We introduced into our Knapp classification a separate class for those who had a comitant vertical deviation in all gaze positions due to spread of comitance, plus a positive Bielschowsky head tilt test. We have called this a class VIII palsy.<sup>14</sup> We wondered whether, in retrospect, surgery should have been tailored specifically for this group. All 6 cases had inferior oblique weakening, with 3 having this alone. In addition to inferior oblique weakening, 1 congenital case with a very lax tendon had a superior oblique tuck, 1 case had an ipsilateral superior rectus recession, and another had a contralateral inferior rectus recession. Three patients (2 with inferior oblique surgery only and 1 with an additional superior rectus recession) had a residual hypertropia of <4 diopters, but all 6 had complete resolution of their symptoms. It would seem, as with so many other SOP cases, that in the absence of a lax tendon or a tight superior rectus, a small to moderate primary position deviation as occurred in these class VIII patients can be successfully treated with an inferior oblique weakening alone.

One hundred seventy-one patients of the 190 treated surgically by us (90%) had inferior oblique weakening as part of the initial procedure (6 had inferior oblique weakening before being seen by us) and 50 (26%) had no other muscles operated on. Inferior oblique myectomy is relatively safe, producing only 2 postoperative Brown-type syndromes, and is very effective. Ipsilateral superior rectus weakening is used for treating the Knapp type IV or V palsies with apparent overaction of the contralateral superior oblique and underaction of the ipsilateral inferior rectus due to restriction of the antagonist superior rectus.<sup>20,21</sup> Contralateral inferior rectus recession is helpful when the primary position deviation is greater than 15 diopters in cases with underaction of the ipsilateral superior oblique, especially when the tendon is not lax and a tuck cannot be done safely. Superior oblique tuck should be used only when there is marked tendon laxity, when the palsy is clearly unilateral, and when there is significant asymmetry of the forced duction test. A forced duction test should be

repeated after a tuck to ensure that the tendon is not too tight and therefore likely to cause a postoperative Brown syndrome.<sup>13,16</sup> A Harada Ito procedure is useful if torsional diplopia is the patient's principal complaint.<sup>18</sup> Since Knapp had suggested that in class IV and V superior oblique palsy the contralateral superior oblique could be weakened with a tenectomy, it should be now emphasized that in view of longer follow-up of this procedure, iatrogenic bilateral SOP can result. Because of this complication, there is now no indication for a contralateral superior oblique weakening when this muscle is apparently overacting. In the case of a class IV Knapp SOP, the recommendation of Souza-Diaz<sup>20</sup> and Jampolsky,<sup>21</sup> ipsilateral superior rectus weakening, should be heeded.

Review of these data suggests that no single feature compels one to label a superior oblique palsy as congenital. Characteristic features singly or in combination led us to diagnose congenital SOP in those cases with typical features usually associated with congenital SOP provided there was no supporting evidence for a diagnosis of acquired SOP. The presence of facial asymmetry was particularly helpful in making the diagnosis of congenital SOP, but it was not completely reliable. A history of early onset and/or knowledge of any long-standing head tilt, preferably with photographic evidence, are keys to the diagnosis of congenital SOP. An asymmetrically lax tendon also contributes to the diagnosis of congenital SOP, but mild tendon laxity can also be seen in some acquired palsies. Sudden onset of symptoms after head trauma, diagnosed or suspected vascular disease, and CNS surgery were the main supporting factors in diagnosis of acquired SOP.

The single most important feature both in diagnosis and in planning the surgical approach in SOP is the status of the superior oblique tendon; that is, lax or taut (normal), which is confirmed by the traction test. Since knowledge of the status of the superior oblique tendon is vital to surgical planning, the traction test should be performed on the superior oblique (both sides) in all cases of superior oblique palsy while the patient is under general anesthesia. Use of information from superior oblique traction testing and of all other parameters of evaluation can lead to effective surgical planning for treatment of SOP. This should include inferior oblique weakening in nearly all cases and superior oblique surgery only for lax tendons and those cases with torsion as the chief complaint. With this scheme, patients with superior oblique palsy can, in a very high proportion of cases (92% in this series), be successfully managed and made symptom-free.

**SUMMARY**

Reports of several large series of patients with superior oblique palsy (SOP) published in 1986 or before set forth important guidelines for both diagnosis and treatment of this condition.<sup>1-4</sup> Newer information about the anatomy, physiology, and pathophysiology of the superior oblique has accrued over the past decade.<sup>5-12</sup> This paper reviews our experience with diagnosis and treatment of SOP over the past 5 years in light of this new information. Charts of patients treated for SOP over 5 years (1990 to 1995) were reviewed for male or female sex, age, symptoms, refraction, vision, stereo acuity, head posture, facial asymmetry, intraoperative superior oblique traction test, diagnostic position prism and cover test, torsion, surgery performed, and results of treatment.

The charts of 190 patients were reviewed. In 181, postoperative examinations were performed by us. The etiology of the SOP was congenital in 137 and acquired in 53. Twenty-nine acquired cases were due to trauma and 24 arose from other causes. Fifty-six patients had facial asymmetry, 51 of whom had congenital SOP.<sup>11,12</sup> Ninety-five had a lax tendon, 83 (87%) of whom had congenital SOP.<sup>8</sup> Sixty-six had a normal tendon, 29 (44%) of whom had acquired SOP. Seventy-seven percent of patients had Knapp class I, III, or IV palsy.<sup>1</sup> An average of 1.26 surgeries was performed per patient. Inferior oblique weakening was performed in 177 (93%), while 68 vertical rectus recessions were done. Thirty-five patients had superior oblique tuck or resection, all on lax tendons, and 15 had Harada Ito procedures for torsion. Six patients had mild Brown syndrome postoperatively, none of which required a takedown.<sup>13</sup> A cure, defined as relief of symptoms or elimination of strabismus and head tilt, was achieved in 166 of 181 (92%) of patients.<sup>4</sup>

Successful treatment of SOP can be accomplished in the majority of cases by selective surgery usually beginning with inferior oblique weakening plus additional vertical rectus and horizontal surgery as needed, with superior oblique strengthening used only for lax tendons or when torsion is the main problem.

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#### DISCUSSION

DR MALCOLM R. ING. It is an honor to be selected by the program committee to discuss this very extensive review of experience with the diagnosis and treatment of superior oblique palsy by a group of notable investigators in the field of strabismus.

Dr Helveston and coauthors present data from their management of 190 cases. They have presented data to show that *facial asymmetry and laxity of the superior oblique tendon* as tested under general anesthesia aid in the differentiation between congenital and acquired superior oblique palsy. The authors claim that these 2 clinical features do not necessarily exclusively rule out acquired palsy by their presence, but are found sufficiently often enough that the clinician can modify surgical management to

obtain a better clinical result.

Overall cure, as defined by relief of symptoms plus elimination or marked reduction in strabismus and head tilt, was achieved in 166 (92%) of 181 patients. This is a very impressive figure, and by these standards, the authors are to be congratulated for their success rate!

Using the historical Knapp classification and recommendations for types of superior oblique surgery, the authors modified their surgical plan according to the results of the tests for tendon laxity. Have the authors really proved their conclusion that this modification was necessary for their high success rate? Perhaps, especially if their recommendation to avoid tucking the tendon if it is *not* found to be lax is followed. All of us can remember some cases with an induced Brown's syndrome (ie, inability to elevate in adduction) from previously tucked superior oblique tendon. Perhaps at least part of the authors' successes would come from avoiding the use of the tuck procedure in cases that do not have lax tendons.

The authors report the results of the forced duction test being positive (ie, lax) in 95 patients. It would be instructive to see if the finding of a lax tendon altered the preoperative plan based on the Knapp classification alone. For example, there were 13 cases classified as type 2 (greater deviation in opposite down gaze). By the Knapp recommendations, all of these patients would have received a tuck procedure. I would want to know if all these Knapp type II patients did, indeed, have a *lax tendon* and therefore received a tuck or resection.

Conversely, did the authors decide *not* to tuck the tendon when, despite the Knapp classification (eg, type II), the tendon was *not* lax and therefore it was not warranted to tuck the tendon by their new surgical approach to the problem of superior oblique palsy?

The authors report that there were 95 lax tendons. Yet, only 35 of this group received tendon tucks or resection. What was the decision-making process that made them decide not to tuck the other 60 patients with lax tendons?

Again, I believe there is a wealth of data in this paper, and I believe the authors are making a significant contribution to our knowledge about the management of superior oblique palsy. However, I would recommend that the data be analyzed to address specifically the question of whether or not the finding of a lax tendon played a significant role in changing the surgical plan from what it would have been using the older guidelines.

Thank you for allowing me to review this very thought-provoking paper.

SUZANNE VÉRONNEAU-TROUTMAN, M.D. FRCS(C) FACS. Dr. Helveston, I want to congratulate you on your excellent paper. My approach is similar to yours in that I myectomize the inferior oblique and tuck the superior oblique. As you do not like to have a Brown's Syndrome after a tuck, my question is, if you want to avoid an undercorrection, how much of a Brown's syndrome will you accept temporarily? I have sometimes had to wait up to a year before a recovery. How long do you wait for a reversal before attempting a surgical correction?

My next question concerns your diagnostic approach. I follow Dr. Marshall Parks' three-step examination technique, but I have found that a fibrosis of the superior rectus can give the same response as a superior oblique palsy. Have you had any such cases?

My last question is, did you use the microscope for your surgery?

EDWARD RAAB, M.D. I enjoyed that a lot, Gene. I have one question regarding the Harada Ito procedure. That operation was advocated for superior oblique palsies that are, as I understand it, almost exclusively torsional in nature. I have had many that have been more concerned with symptoms referable to the torsional misalignment than the vertical. But never have I seen a pure torsional misalignment. Therefore, I have had little occasion to do the Harada Ito procedure because I find that where they have the coexisting vertical deviation, and they typically do, surgical choices involving the vertical deviation seem to work pretty well for the torsional problem also.

You have a tremendous experience in this and I would like to hear your comments.

MARSHALL M. PARKS, M.D. Gene, I did appreciate your excellent report of such an extensive study. However, your report was essentially on surgical treatment of unilateral superior oblique palsy. I heard very little about bilateral superior oblique palsy which, of course, is a very broad subject in itself. Perhaps the program time restraint prevented the discussion of this aspect of the subject on superior oblique palsy it deserves.

The issue I wish to speak about is the facial deformity secondary to the torticollis which you so aptly described. However, the fact that you still advise that the torticollis secondary to congenital superior oblique palsy be treated initially by an inferior oblique weakening procedure is what prompts my comment. But first, allow me to comment about reversing the facial deformity in congenital superior oblique palsy which results from a large 25 to 40 degrees torticollis. The torticollis becomes obvious as soon as the baby can control the head, which is usually by 3 months of



age. The facial disfigurement quickly becomes permanent and it is unsightly. Since I heard no comment about when you would address this issue, I will present my view. I advocate surgically correcting the torticollis as promptly after its onset as possible, assuming the diagnosis of the palsied superior oblique muscle is proven by a positive Bielschowsky head tilt test. If the patient with congenital superior oblique palsy has torticollis at three months of age, then I do the surgery at that age.

But worse than facial deformity is the fact that torticollis eventually causes pain in the neck and back, beginning usually in the third to fourth decade of life. Ophthalmologists seem to be less aware of this than our orthoptic colleagues. Ophthalmologists tend to delay correcting the torticollis until an arbitrary older age when the chance for the best result has passed.

I inferred from your presentation that regardless of the nature of the superior oblique palsy, you conventionally start with weakening the ipsilateral inferior oblique in most cases and/or recessing the contralateral inferior rectus in some fewer cases, which I agree with except in the infants with severe torticollis. My experience in infants with proven congenital superior oblique palsy and severe torticollis is that the conventional approach of first weakening the ipsilateral inferior oblique or recessing the yoke inferior rectus invariably fails to eliminate the torticollis. In these cases I have had to resort to the tuck of the palsied superior oblique which, unfortunately, produces a Brown's syndrome. But I accept the Brown's syndrome as a cheap price to pay for the simple surgical procedure that invariably brings the head upright, eliminates the facial deformity, and prevents neck and back pain in the latter part of the patient's life.

THOMAS HEDGES, JR., M.D. I would like to congratulate Dr. Helveston on his contribution to this very important and relatively common condition. I stand here as a general ophthalmologist and neuro-ophthalmologist who has seen many non-traumatic fourth nerve palsies. The patients that I see, not being a pediatric ophthalmologist or "strabismologist", are usually people in their mid-life, usually men. They have an insidious onset of diplopia. Often they have very subtle complaints, such as headache, difficulty driving long distances, and when long standing can go on to scoliosis as well, when they have had it over many years. The important thing about facial asymetry is that you are reporting a younger subset of patients than I refer to and, therefore, I am not addressing that issue at all. It is a very important observation.

I would like to point out two things that I think you brought out in your paper. One is that the majority of the older patients we see are uni-

lateral, idiopathic, superior oblique palsies that were not complicated and do not have a superior oblique laxity at surgery. Second, those patients always invariably respond well to simple inferior oblique recession.

What is most distressing to me is to see patients with typical symptoms and signs of isolated superior oblique palsy who are subjected to MR or CT. This is wasting resources and it is only because the doctor that is referring the patient is insecure and has to cover his tracks.

I should like to ask, should we be doing superior oblique traction tests on all of these patients?

Again, I would like to compliment you on this paper and on the work that you wrote up several years ago. Hopefully we will not continue to have patients referred to us with their MR and CT in their hands.

Thank you.

EUGENE HELVESTON, M.D. First of all I would like to thank Dr. Malcolm Ing for his thoughtful discussion of this paper. The answer to Dr. Ing's first question is that we honor the Knapp classification diagnostically. We also respect the Knapp classification therapeutically but modify it depending on the status of the superior oblique tendon. We tuck or resect the tendon only when it is lax or if it is absent we employ some other means to treat the appropriate muscle in the necessary field of action. We reported 95 lax tendons but we did a strengthening procedure, either Harada Ito, tuck or resection in only SO or approximately half of the lax tendons. We graded lax tendons according to the response to the forced traction test in the operating room. Some of these lax tendons were -1, -1 symmetrical lax tendons. These may have simply been variations of normal. Nothing was done in these cases. We therefore may have over diagnosed lax tendons in our series. We strengthened only those lax tendons which presented asymmetrically always doing a strengthening procedure to the looser tendon.

With regard to Brown's syndrome, I am sensitized to the problems associated with postoperative Brown's syndrome because I have seen many patients with only a slight limitation of elevation in adduction due to an acquired Brown's syndrome who spent an inordinant amount of there time looking up and therefore complaining about the double vision. Because of this experience, I am adverse to creating a Brown's syndrome in any patient. My bias therefore is to avoid doing superior oblique strengthening procedures in patients with a normal or taut tendon. Instead we do inferior oblique weakening and appropriate vertical rectus surgery to treat the vertical deviation.

In answer to the question of Dr. Véronneau-Troutman about the microscope, we used the microscope to study the histologic specimens

shown here. We did not use the microscope for surgical procedures.

As far as a tight superior rectus is concerned, I am not sure that I can address the questions specifically other than I am sure that the ipsilateral superior rectus is tight in many cases of longstanding superior oblique palsy. We recently presented a poster at the AAPOS meeting demonstrating three cases of iatrogenic bilateral superior oblique palsy where a surgeon did a tenectomy of the "overacting" normal superior oblique. These cases were "almost" fixed regarding the original superior oblique palsy but the bilateral superior oblique palsy that was created simply did not go away. We strongly disavow weakening the overacting fellow superior oblique in unilateral superior oblique palsy.

Dr. Rabb asked about "pure torsion" in the patients who were treated with a Harada Ito. We use this procedure primarily in bilateral cases where torsion is the primary complaint and/or finding. These bilateral superior oblique palsy patients usually have a "V" pattern, a chin down head posture, and a reversing Bielschowsky or a very small vertical deviation with the head tilted to either side and a minimal vertical deviation in the primary position.

Dr. Parks makes an excellent point. We did not specifically talk about bilateral superior oblique palsy. However, we reported in this series 19 patients who were class VI which is bilateral superior oblique palsy. We included the bilaterals in the overall series almost as if they were 2 unilaterals. But obviously these patients were more difficult to treat than the unilateral superior oblique palsy patients. We treated selected bilateral superior oblique palsy patients surgically with a bilateral Harada Ito procedure. Others were treated with medial rectus recession with down shift or inferior rectus recession with nasal shift.

I question Dr. Parks' comment about abnormal posture in a 3-month old patient. I believe the neurologic process leading to the head tilt test is not activated when the patient is supine or prone. So I do not know why a patient would develop a head tilt from superior oblique palsy before he/she has learned to sit up and assume the upright or vertical position.

While we have operated on a patient as young as 6 months of age, the head tilting began after the patient learned to sit up. I know how difficult it is to get rid of big head tilts in infants, but I still avoid a large superior oblique tuck on a normal tendon because I cannot see the value in creating a Brown's syndrome for the improvement of head posture. In such a case I prefer doing inferior oblique weakening and whatever else needs to be done to eliminate the vertical deviation and therefore hopefully eliminate the head tilt. Fortunately, many very young children with large head tilt have a lax tendon suitable for tuck or resection.

Finally, I totally agree with Dr. Hedges. The patient population makes all the difference with regard to a consultant's experience with a given disease. I think the adult who consults a neuro-ophthalmologist first might be more likely to have problems requiring a workup including neuroimaging. I remember Dr. Frank Walsh's Saturday morning conference at the Wilmer Institute at a time when the only way to look "inside of the head" was to do cerebral arteriography, a test with a significant morbidity. Dr. Walsh said that it was necessary to have two signs, not just a sign and a symptom, before doing this test. I still have that advice in the back of my mind so I may over compensate toward not doing an MRI or CT scan. With regard to surgery, I would also like to reassure Dr. Hedges that our primary surgical procedure for the treatment of superior oblique palsy is inferior oblique myectomy. We operate on other muscles as needed but in almost every case this is done after an initial inferior oblique weakening procedure.

My thanks to the American Ophthalmologic Society for allowing me to give this paper and my thanks to all of those who made thoughtful comments and posed significant questions.