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Bilateral superior oblique palsies

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SUMMARY Eighteen consecutive cases of bilateral superior oblique palsy have been studied retrospectively. The patients were typically adults with a history of severe head trauma. All patients complained of diplopia, either vertical, torsional, or both. Other neurological sequelae were common. Central disruption of fusion was seen in three patients. Eight patients underwent surgery for torsional or vertical diplopia, and the results are briefly discussed.

Bilateral superior oblique palsy is not a common ocular motility problem.[1-3] It is nearly always acquired, often following closed head trauma, as in a road traffic accident. It is not unusual for the period of unconsciousness to be rather prolonged, and other neurological complications may occur. These patients complain of vertical diplopia, often with a torsional component, in the lower fields of vision, and are often orthophoric in the primary position. Fells and Waddell[4] have emphasised that this may cause delay in diagnosis and a tendency to label patients as 'compensation neurosis.' Careful examination, however, will reveal the defect, although several tests may prove necessary. A number of authors have emphasised that these palsies are often asymmetrical and may be misdiagnosed as unilateral acquired superior oblique palsy, and managed accordingly.

Patients and methods

Patient selection. The charts of private patients attending the Children's and Strabismus Clinic at the Bascom Palmer Eye Institute between April 1974 and February 1983 were reviewed. Eighteen patients were found to have a diagnosis of bilateral superior oblique palsy and data from them were analysed. Of the 18 patients 13 were males and 5 females. The age range of the males was 19 to 71 years, mean 40, and of the females 24 to 74, mean 39.

Aetiology. Sixteen patients had suffered severe head trauma with loss of consciousness. Fourteen had been involved in a road traffic accident, one fell off a horse, and one was involved in an aeroplane accident. One patient had myasthenia gravis with a positive edrophonium test, but a poor response to oral pyridostigmine. One patient had probably had a long-standing right superior oblique palsy of the congenital type, on which was superimposed an acquired left superior oblique palsy of probable vascular aetiology. In the cases with closed head trauma the duration of unconsciousness, where known, was from half an hour to one month. There was a higher incidence of other traumatic neurological symptoms in patients whose coma lasted for two weeks or more. The duration of time between the initial trauma and being seen at the Bascom Palmer Eye Institute was known in 16 cases and ranged from two to 60 months, mean 22·5 months.

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Results

Clinical findings (Tables 1 and 2). All the patients complained of diplopia, either vertical, torsional, or in combination. Additional complaints included abnormal head position and headaches. Corrected visual acuities were good, the worst recorded being 6/12, and in no patient was there more than one Snellen line difference between the two eyes.

Ocular deviation. In four patients there was orthophoria in the primary position, and in two more the primary position deviation was purely horizontal. In the remaining 12 patients there was a vertical deviation in primary position. In five patients this was purely vertical, in one there was a coexistent esotropia, and in six there was an exotropia. In downgaze all patients showed an increase in the vertical deviation. In depression to either side nine showed a classical pattern of hypertropia of the adducted eye with diplopia reversing between left and right depression. In two cases this information is not available in the patient's record. In the remaining

Table 1 *Preoperative data*

Case	*Age, sex*	*Corrected VA*		*Presenting complaint*	*Aetiology*	*Other neurological problem*
		R	*L*			
1	31 M	6/3	6/3	Vertical diplopia	Myasthenia gravis	Positive edrophonium test
2	51 M	6/9	6/12	Torsional diplopia	Airplane accident Oct 1973. Head trauma	Amnesia
3	34 M	6/4	6/4	Vertical diplopia	Car accident Mar 1972. Head trauma	L hemiparesis. R facial palsy. L XI n palsy
4	43 M	6/6	6/6	Oblique diplopia	Car accident 1973. Cranioplasty 9 mo later	Field defect. Retraction convergence nystagmus
5	26 F	6/9	6/12	Torsional diplopia	Car accident 1978. Head trauma	None
6	36 F	6/6	6/6	Headache. Vertical diplopia	Fell off horse 9 months before. Head trauma	Memory loss. Depressed skull fracture R temporal area
7	47 M	6/6	6/6	Vertical diplopia	Car accident June 1977. Head trauma. Coma 'days'	R hemiparesis. Hearing loss. Expressive aphasia
8	62 M	6/4	6/6	Vertical diplopia	Prob. 'congenital' RSO palsy+acquired LSO palsy 2° to hypertension	Meningioma of R convexity removed 1975
9	24 F	6/6	6/6	Vertical diplopia	Car accident Nov 1979. Head trauma. Coma 3 days	None
10	74 F	6/9	6/9	Vertical diplopia	Car accident June 1980. Head trauma	None
11	24 M	6/6	6/6	Oblique diplopia	Car accident 1976. Coma 1 month	Weakness R arm. Slurred speech. L facial palsy
12	26 M	6/6	6/6	Torsional and vert. diplopia. Head tilt L	Car accident Apr 1981. Head trauma. Coma 2–3 wks.	L hemiparesis. Poor memory. On phenytoin. Abnormal speech
13	32 M	6/4	6/4	Oblique diplopia	Motorcycle accident July 1980. Head trauma. Coma 3 wks	R hemiparesis. Dysphasia
14	71 M	6/6	6/6	Vertical diplopia	Car accident Mar 1982. Head trauma. Coma 'days'	None
15	39 M	6/6	6/6	Oblique diplopia	Car accident Nov 1978. Head trauma. Coma 2 wks	Poor balance. Poor memory. Nerve damage R foot
16	38 F	6/4	6/4	Vertical diplopia. Chin depression	Car accident May 1982. Head trauma. Coma ½ h	None
17	19 M	6/4	6/4	Vertical and torsional diplopia	Car accident Oct 1982. Head trauma. Coma 48 h	None
18	25 M	6/4	6/4	Oblique and torsional diplopia	Car accident Oct 1978. Head trauma. Coma 16 h	None

RSO=right superior oblique. LSO=left superior oblique.

seven cases the paresis was judged to be asymmetrical, with one eye being constantly hypertropic, maximally in the opposite inferior field to the palsy.

Head-tilt test. The Bielschowsky head-tilt test was performed and recorded in 15 cases. In nine cases it was bilaterally positive, with upshoot of the eye on the side of the head-tilt. In six cases it was positive only on tilt to the more hypertropic eye, in all six cases the right eye.

Double Maddox rod test. The double Maddox rod test was used to measure subjective cyclodeviation. In 12 cases there was subjective excyclophoria which could be elicited on fixation with each eye. In three cases there was detectable excylophoria of only one eye, in each case the less hypertropic eye being the eye with the perceived cyclodeviation. In three cases the test was not performed.

Ocular rotations were assessed. In nine cases there was detectable underaction of both superior oblique muscles, often one more than the other. In seven cases only one superior oblique could be seen to underact, with overaction of the ipsilateral inferior oblique. In two cases the rotations were not recorded.

V pattern. Several authorities have emphasised the importance of a marked V pattern as an indicator of bilaterality in superior oblique palsies. In this series the size of the V pattern ranged from 30 prism dioptres to a small A pattern of two prism dioptres. In addition to the latter patient three others had no

Table 2 *Preoperative data*

Case	Deviation primary position	Deviation in downgaze and R and L depression			'V' pattern	Bielschowsky head-tilt test	Cyclo-deviation	Rotations	Fusion?
1	Ortho	8 LHT 4 LET	2 LHT 2 LET	2 RHT 4 ET	8	Positive to R and L	Bilateral excyclophoria	U/A LSO O/A LIO	Yes
2	Ortho	4 RHT 10 ET	20 ET	8 LHT 8 ET	20	Positive to L and R	5° Ex L 5° Ex R		Yes
3	45 LHT 10 XT	55 LHT 7 LXT	40 LHT 10 XT		15		20° Ex R 5° Ex L	U/A LSO O/A LIO	Dubious prob. absent.
4	18 RHT 10 ET	8 LHT 20 ET	18 RHT 20 ET	35 RHT 10 ET	30			U/A both SO	No
5	3–4 RHT 12 XT	22 LHT	6 LHT (fixR) 4 RHT (fixL)	14 RHT	8	Positive to L and R	15° Ex L and R	U/A both SO	Yes
6	14 RHT	10 RHT	18 RHT	30 RHT	0	Positive to R	10° Ex R 5° Ex L	U/A RSO O/A RIO	Yes
7	10 ET	12 ET 20 LHT	16 ET 4 LHT	12 ET 10 RHT	16		10° Ex R 12° Ex L	U/A both SO	Weak but present
8	2–3 RHT		10 RHT		8	Positive to R	5° Ex R 5° Ex L	Minimal U/A both SO	Yes
9	4–6 LHT	18 LHT	8 LHT	Ortho	0	Positive to L more than R	5° Ex R	U/A LSO O/A LIO	Yes
10	2–3 RHT 4 XT	1–2 LHT	15 ET 7 RHT	4–6 RHT	15	Positive to R more than L	Marked ex R and L	U/A both SO	Yes
11	30 RHT 7 XT		35 RHT 16 XT		−2 ('A' pattern)	Positive to R		U/A RSO U/A RLR	Unclear
12	5 RHT 3 XT	6 RHT	10 RHT 7 ET	12 RHT	19	Positive to R and L	10° Ex R 5° Ex L	O/A RIO	Yes
13	4 XT Trace LHT	2 XT 2 LHT	4 XT 2 RHT	4 XT 3RHT	0	Positive to R			Yes
14	6 RHT	3 RHT	12 RHT	18 RHT	8	Positive to R	10° Ex R 5° Ex L	U/A SO L more than R	Yes
15	Ortho	5 LHT 16 ET	3 LHT 20 ET	7 RHT 14 ET	20	Positive to R and L	4° Ex R 7° Ex L	U/A both SO	Yes
16	5 RHT 1 XT	8 RHT 3 ET	10 RHT 1 ET	8 RHT 3 ET	2	Positive to R and L	11° Ex L	U/A RSO	Yes
17	Ortho	2 RHT 8 RET	5 RHT 10 RET	6 RHT 6 RET	10	Positive to R	5° Ex L	U/A both SO	Yes
18	1–2 RHT 3 XT	8 LHT 12 ET	8 ET	16 RHT 12 ET	15	Positive to R and L	5° Ex R 15° Ex L	U/A both SO O/A both IO R more than L	Yes

ET=esotropia, convergent squint. XT=exotropia, divergent squint. HT=Hypertropia. All measurements are in prism dioptres except where otherwise indicated.

detectable V pattern. In the 14 cases with a V pattern the average was 18 prism dioptres, and only in three cases was it 20 or more prism dioptres.

Fusion was assessed by tests in free space and, where doubt existed, on the synoptophore. Fourteen patients had good fusion when corrected with prisms or on the synoptophore. One had poor fusion, two had no fusion, presumably owing to head trauma, and in one case the fusional status was unknown.

Other neurological problems. Eight patients had no neurological signs or symptoms (one had coincident hypothyroidism). One patient was myasthenic. Nine patients had significant sequelae of their head trauma (summarised in Table 1). These ranged from memory loss after temporal skull fracture to hemiparesis, dysphasia, and convergence-retraction nystagmus. As might be expected, all the cases with absent, poor, or doubtful fusion had major neurological problems.

MANAGEMENT

Non-surgical management. This was advised in seven cases. The reasons were various. One case (No. 1) had myasthenia gravis and only moderate symptoms. One case (No. 5) was already improving spontaneously when first seen and continued to do so. Two cases (Nos. 9 and 10) had small deviations which could be rendered tolerable by prisms or occulsion of one lens of reading glasses. Three cases (Nos. 4, 11, and 13) with major neurological problems were advised that surgery would probably not be of value in relieving their symptoms, and one of these was fitted with prisms.

Surgical management. Table 3 shows the surgery

Table 3 *Surgery and results*

Case	Date of surgery (interval from onset of palsy)	Surgery performed	Immediate postop. status	Final postop. status	Rotations	Success
2	17 May 74 (7 months)	Bilateral superior oblique tucks		6/6 both eyes. Diplopia in extreme gaze positions. RHT3–4 in up and R gaze. LHT2–3 in down and L gaze	Mild U/A LIO	Yes
3	(1) 13 Sept 73 (2) 15 July 74 (3) 21 Oct 74 (18 months)	(1) LIO Recession, RIR recession 6 mm. (2) Bilateral superior oblique tuck. (3) RLR recession 8·5 mm, LSR recession 3 mm, LIR resection 4 mm		(1978)-Still diplopia. 25 LHT distance, 8 RHT near. Poor vertical OKN	Limited upgaze R more than L	No
6	20 Aug 79 (1 year)	Right superior oblique tuck	12–14 LHT. 15° incyclo. Fusing in lower field of gaze	(1979)-Diplopia only on up and downgaze at distance. Primary position: 3 EP+1 LHT. Large field of single vision	U/A RIO	Yes
7		Bilateral superior oblique tucks 10 mm R, 12 mm L	Diplopia all fields of gaze. A exotropia. Bilateral incyclo 50° L, 20° R	(1982)-Intermittent diplopia-can control. 2 LHT near and dist. 5° incyclo. Bilateral 'Brown's'	U/A bo IO	Partial
12	26 Apr 82 (1 year)	Bilateral Harada-Ito procedures. R inferior oblique disinsertion	Incyclo 2° R. Primary 8 XT 7 LHT. Downgaze 8 XT 6 LHT	Small field of single vision with 2 prism dioptres BDR with 2 prism dioptres BU } L with 1 prism dioptres BO }		Partial
14	(1) 22 Nov 82 (2) 23 May 83 (8 months)	(1) R superior oblique tuck. (2) L inferior rectus adjustable recession	Improved. Still diplopic	Less diplopia. Primary-ortho 3 LHT----2 RHT---6 RHT 4 ET 3 ET	O/A RIR U/A LIR	Partial
16	14 Mar 83 (10 months)	R inferior oblique recession. L adjustable Harada-Ito	Better. Little incyclo			Yes

ET=esotropia, convergent squint. XT=exotropia, divergent squint. HT=Hypertropia. All measurements are in prism dioptres except where otherwise indicated.

performed and the results in seven patients. One patient is awaiting surgery elsewhere (No. 15), and three patients are awaiting surgery in Miami (Nos. 8, 17, and 18). Various surgical treatments have been given. In all cases some form of superior oblique strengthening operation was performed, either unilaterally or bilaterally. Two cases (3 and 14) required multiple surgical procedures. Few conclusions could be drawn from these data, though it is notable that the patients with the best results from surgery had relatively mild injuries as judged by the absence of other neurological problems. On the other hand the patients who had poor results in terms of freedom from diplopia had all suffered considerable trauma. All had had a hemiparesis and other serious neurological sequelae of their injury. Whether the poor result can be attributed directly to the fusion loss or to the motility defect is unclear.

Discussion

Bilateral superior oblique palsies are not very common. Rucker[1,2] in his two separate series of 1000 cases of third, fourth, and sixth nerve paralyses found incidences of 67 out of 1000 and 84 out of 1000 for acquired fourth nerve palsy, but did not examine for bilaterality. This was done in a very similar study by Rush and Younge,[3] also from the Mayo Clinic. They found 172 out of 1000 cases of fourth nerve palsy, of which 13 were bilateral, 12 being considered to be due to head trauma. Younge and Sutula[5] reported 52 cases of superior oblique palsy over a two-year period. This included all causes of superior oblique palsy, including congenital palsy. They found four bilateral cases, all traumatic. Burger *et al.*[6] reported on 33 patients of superior oblique palsy, four of whom were bilaterally affected. All were the result of severe head injury, with coma lasting for 16 hours to 13 days. One of their patients, a 7-year-old boy, improved spontaneously, but the other, adult, cases did not. Khawam *et al.*[7] reviewed 3000 consecutive cases of strabismus and found 40 cases of acquired superior oblique palsy, of which seven were bilateral. Sydnor *et al.*[8] reported 33 cases of traumatic superior oblique palsy over a 10-year period. Of these, 12 were thought to be bilateral.

Other authors have reported series of bilateral superior oblique palsy. Chapman *et al.*[9] reported six cases, all due to motor car accidents. Raskind[10] reported three cases, all post-traumatic. Hermann[11]

reported nine cases initially diagnosed as unilateral which turned out to be bilateral after the initial surgery was performed. Lyle[12] reported eight cases seen over a five-year period, all traumatic in origin. Mitchell and Parks[13] reported nine patients in a prospective study of superior oblique surgery. The largest reported series of which we are aware is that of Fells and Waddell,[4] who reported 31 patients, 25 of whom were the result of head trauma. The present 18 cases were found on review of approximately 5500 patient case notes and covered a nine-year period.

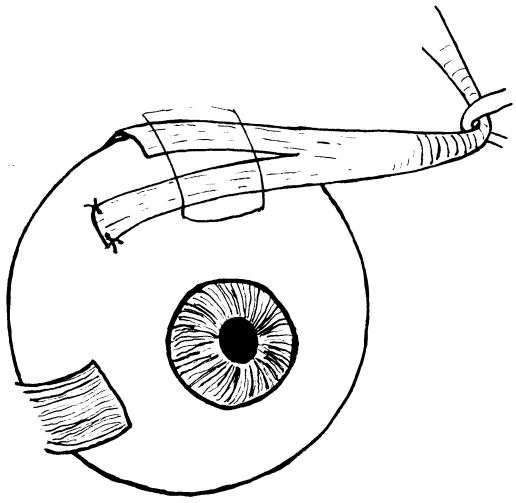
Many authors emphasise the necessity of suspecting bilaterality in all cases of traumatic superior oblique palsy. This is touched upon by Khawam *et al.*[7] and Sydnor *et al.*[8] Hermann[11] devotes his entire paper to nine cases initially thought to be unilateral which showed paresis of the opposite side after the initial surgery. Various diagnostic criteria are advised; Sydnor *et al.*[8] suggested that a V pattern of more than 25 prism dioptres, excyclotorsion of more than 10°, or head trauma sufficient to cause loss of consciousness should suggest possible bilaterality. Our figures do not suggest that a V pattern should exceed 25 prism dioptres, our average being 18, with four patients showing no V pattern whatsoever. In addition the cases reported by Hermann[11] had an average V of 6·5 prism dioptres, one case having an A pattern. In a series of six patients reported on by Chapman *et al.*[9] only four had significant V patterns. None of Raskind's three patients had a V pattern.[10] All but one of Michell and Parks's patients had a V pattern, but the size is not given.[13] Fells and Waddell[4] do not report the incidence of V patterns in their series.

We seem to be on safer ground when examining cyclotorsion. A number of patients complained of torsional diplopia, and double Maddox rod testing revealed excyclophoria in almost all patients. Ellis and Helveston[14] and Fells[15] have suggested that a finding of excyclotorsion of more than 15° by this method is strongly suggestive of bilateral fourth paresis. The average in this series was 13° of excyclotorsion. In very asymmetical cases the excyclophoria was perceived as being in only one eye, interestingly enough the less hypertropic in each case. This would seem to accord with the findings of Olivier and Von Noorden[16] in which the subjective tilt is seen in the non-dominant eye irrespective of the paresis. The only test used to measure torsion was a double Maddox rod test. Ruttum and Von Noorden[17] have recently thrown doubt on the usefulness of this test, especially when adjustable sutures are used on the superior oblique muscle. Fells and Waddell[4] advocate the use of a T-piece wand with a Lees screen as being accurate and non-fatiguing. The technique is described by Dulley and Harden.[18]

We agree with Sydnor *et al.*[8] and Fells and Waddell[4] that there is an association of bilaterality with severe head injury and with other neurological sequelae. It is also noted by Burger *et al.*[6] On the other hand Khawam *et al.*[6] state, 'the apparent insignificance of the head injury was a striking feature in many of the cases.' They do not specify whether they are discussing unilateral or bilateral cases. All the cases reported by Raskin[10] and Chapman *et al.*[9] were rendered unconscious, in several instances for prolonged period of time. It seems clear that severe head injury is much the commonest precipitating cause. The mechanism is less clear. Most authors, on anatomical grounds, place the lesion in the anterior medullary velum, where the trochlear nerves decussate. An alternative explanation is suggested by the work of Heinze[19] on fatal road traffic accidents. He found that the third, fourth, and sixth nerve could be wholly or partially avulsed as they emerged as rootlets from the brain stem.

When positive in both directions of head tilt, the Bielschowsky head tilt test is diagnostic. It will be positive in only one direction in very asymmetric palsies, and should not be relied upon when performed in the supine patient, as pointed out by Sydnor *et al.*[8] We therefore suggest that bilaterality should be suspected in all severe head injuries, that a Bielschowsky head tilt test may be diagnostic, and that the diagnosis be made on the basis of the deviation in major gaze positions, cyclotorsion measurements, and assessment of the ocular rotations.

As regards the results of surgical treatment, it will be seen that a variety of procedures were used, making it difficult to draw conclusions. We believe that it is insufficient in these cases to operate solely on the overacting muscles—that is, the inferior obliques or inferior recti. However, Lyle[12] in 1964 reported on eight patients in whom a variety of surgical procedures, including inferior oblique myomectomy and repositioning of the inferior rectus insertions, gave good results. Five of his patients in addition had superior oblique plications. In our view some form of superior oblique strengthening procedure must be performed, either plication or the Harada-Ito anterior half tendon transposition procedure. A good description of this procedure (modified by Fells) is given by Mitchell and Parks,[13] and illustrated in Fig. 1. If the excyclotorsion can be eliminated by this means, further surgery or prisms may be used to deal with any residual deviation. In general the better demonstrable fusion preoperatively, the better the surgical result. Fells and Waddell[4] have advised against being too influenced by apparent poor fusion, as this may be due to torsional diplopia, with improvement of fusion after surgery.



ANTERIOR HALF TENDON TRANSPOSITION OF SUPERIOR OBLIQUE

Fig. 1 *Anterior and temporal transposition of the anterior portion of the superior oblique tendon improves the cyclotorsional action of the muscle.*

CONCLUSION

Bilateral superior oblique palsy is an uncommon but debilitating condition, usually a sequela of severe head trauma. Care in testing and a high index of suspicion are required to detect bilaterality in asymmetric cases. Loss of fusion may occur, but, where it is present, appropriate surgery with strengthening of the superior oblique muscles is the treatment of choice.

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