OCULAR TORTICOLLIS*

BY
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INTRODUCTION

Tortus, the Latin word for twisted, and collum, for neck, are the roots of the word torticollis.[1] The etiology of torticollis may be orthopedic[2] (including osseous, ligamentous, and muscular), neurologic, or ocular. Orthopedic causes include acquired traumatic or inflammatory cervical myositis and congenital musculoskeletal abnormalities, such as shortening and fibrosis of the sternocleidomastoid muscle, Klippel-Feil syndrome, and occipitocervical stenosis.[3] Cervical spine subluxation[4] is associated with congenital muscular torticollis and craniofacial asymmetry. Abnormalities of the skull, such as plagiocephaly, or unilateral coronal suture stenosis, can result in underaction of the superior oblique, overaction of the ipsilateral inferior oblique, and ocular torticollis, with a positive Bielschowsky head tilt test.[5] Torticollis associated with hiatus hernia is described as Sandifer syndrome,[6-9] with varying movements of the head and neck, often with twisting of the neck from side to side. Torticollis may occur in association with deafness, especially when unilateral,[10-12] or functional or psychiatric disturbances.[4,11]

Torticollis with an ophthalmic etiology was first described by Cuignet in 1873.[13] Ocular torticollis is an abnormal head posture that may be adopted by a patient in order to maintain binocular vision, with a twist or turn of the neck. There may be an associated head tilt, face turn, chin elevation, chin depression, or a combination of these postures.[3,14,15] Scoliosis may result from compensation for extraocular muscle paresis.[13] Verzella and associates,[16] in their description of 8 patients, credited von Graefe for recognizing this mechanism in 1864. Ruedemann[17] and Dietrich and Slack[18] also described scoliosis and spine misalignment secondary to ocular paresis of one or more vertical eye muscles. According to Wesson,[19] the purpose of ocular torticollis is to assist the patient's vision in 1 or more of 6 ways:

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1. To improve vision.
2. To bring the field of vision into a central area by reducing nasal field occlusion.
3. To strengthen or to achieve single binocular vision.
4. To reduce eye discomfort (eg, presbyopia, diplopia).
5. To afford eye protection.
6. To offer relief from pain.

Nutt[10] has described the compensatory head posture as a reflex activity, the stimulus being the presence of diplopia, with the altered head position allowing stimulation of corresponding retinal areas to allow single binocular vision.

BACKGROUND FOR STUDY

In a 2-year prospective study of abnormal head postures based on ocular causes, Kushner[20] identified 8 basic mechanisms causing torticollis in 188 patients, in an academic center. In his study, Kushner included patients who did not demonstrate ocular torticollis but had conditions frequently associated with head posture abnormalities, attempting to attain single binocular vision or some degree of fusion. Kushner identified incomitance as the leading cause of torticollis in 118 patients (62.7%) and found 70 cases of vertical incomitance, including 46 with superior oblique palsy, 7 with inferior oblique palsy, 6 with Brown syndrome, 5 with blow-out fracture, 3 with double elevator palsy, and 3 with superior rectus palsy. The horizontal incomitance group consisted of 48 cases, including 31 with Duane syndrome, 7 with acquired horizontal incomitance, (4 after asymmetric surgery, and 3 with sixth nerve palsy), 4 with A or V pattern, and 6 with torsional incomitance.

Kushner[20] found nystagmus in 38 cases as the second leading cause of abnormal head posture (20.2%). Other groups included 12 cases with congenital esotropia with ocular posture (6.3%), 10 cases permitting foveal fixation (5.3%), 4 cases with cosmetic etiology (2.1%), 3 cases with ocular motor apraxia (1.6 %), 2 cases with spasmus nutans (l%), and 1 case with astigmatism (0.5%).

MATERIALS AND METHODS

The clinical impression of this author was that the data presented by Kushner[20] would be somewhat similar to the clinical experience in this private ophthalmology practice. What this author found, however, in comparing this study to that of Kushner, was that while most cases of torticol-

lis could be explained, there remained a category of patients with torticollis without any apparent reason. Because this latter group of patients was not addressed in Kushner's study, the intent of this author is to compare the known etiologies for ocular torticollis as described by Kushner, as well as to reveal the incidence of, and to attempt to define the etiologies of, unexplained ocular torticollis.

Beginning on December 1, l992, a 12-month prospective study of all patients examined by this ophthalmology group was undertaken to identify and categorize the various reasons for ocular torticollis. A total of 15,168 patient visits were recorded, and 630 patients were identified with measurable ocular torticollis. From this group, a subset of 25 patients were identified with ocular torticollis in association with medical or neurologic conditions too complex to be studied in this paper. The remaining 605 patients presented with torticollis with a presumed ocular etiology, without evidence of musculoskeletal or neurologic etiology.

Of the 15,168 recorded office visits during the year of the study, a number of patients were examined more than once. After deleting repeated office visits, a patient pool of 11,299 remained, resulting in an incidence of torticollis of 630 of 11,299 (5.6%). Each of the 630 patients had a demonstrable head posture and had no prior history or evidence of orthopedic or neurologic abnormalities. Those without head posture abnormalities were excluded, in contrast to Kushner's study, which included a number of patients without head tilt but with a condition associated with head tilt. Therefore, the percentage comparisons between the 2 studies is not exact.

Each patient received a complete eye examination, including age-appropriate visual acuity testing, with the notations C, S, M for central, steady, and maintained fixation for infants, to preferential looking with the Teller visual acuity cards, Mentor BVAT equipment using pictures, "E," "HOTV," and alphabet. Additional measurements included stereoscopic testing, Worth 4-dot test, convergence amplitudes, near point of convergence, pupil evaluation, anterior segment, eye rotations, cover testing, cycloplegic refraction, indirect ophthalmoscopy, observing for ocular torsion, and direct ophthalmoscopy. When possible, intraocular pressure by applanation or digitally, red filter, double Maddox rod test, Bagolini striated glasses, Hess screen, or the Lancaster red-green test were performed.

The torticollis of every patient in this study was measured with a goniometer, a protractor with extended, hinged arms, an instrument used for orthopedic measurements on x-rays. When measuring a face turn, one arm was directed at the fixation target, and the second arm was placed along the sagittal axis of the head. Measurements were read directly from the goniometer in degrees 1,14,21 (Figs 1 and 2). When a head tilt or chin

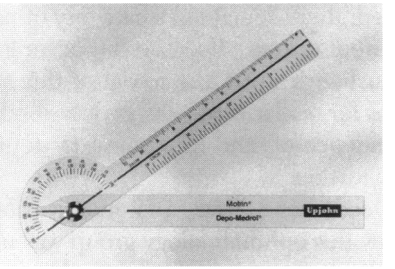

FIGURE 1
Orthopedic goniometer used for measurement of torticollis.

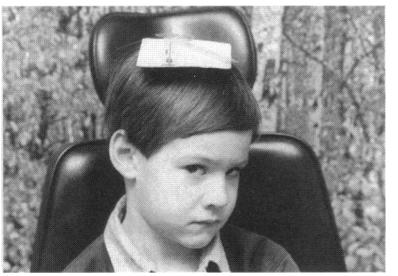

FIGURE 2
Patient with goniometer placed on head, with one arm directed at fixation target and one arm in direction of head turn. Head turn is measured directly in degrees.

tuck or chin up position was measured, the patient fixated on a distant target at eye level, while 1 arm of the goniometer was placed parallel to the axis of the face, and the second arm perpendicular to the floor. Again, the degrees of torticollis were read directly from the goniometer. (The goniometer is available without cost from the Upjohn Pharmaceutical Company, 7000 Portage Dr., Kalamazoo, MI 49001; 616-329-8244).

Other devices to measure torticollis have been described by various investigators. Cooper and Sandall[22] recommended using a perimeter; however, this is very cumbersome and impractical with young children. Schatz and Urist[23] described an apparatus that was strapped to the patient's head, and involves a protractor, a level, and a directional pointer. This instrument was never popularized, however, and is not available commercially. Young[24] proposed a photographic technique, which requires a linen headband, three hinged mirrors, and multiple photographs. This is also impractical and unrealistic when examining young patients. The handheld goniometer remains the instrument of choice for measuring face turns, head tilts and chin postures as the patient views age-appropriate distant and near fixation targets.

A number of patients presented with a face turn, head tilt, or chin elevation or depression only, while others presented with postures combining 2 or 3 of the head or face positions. To compare the measurements of torticollis for patients within each group, as well as for comparison from group to group, it was determined that the degrees of face turn, head tilt, and chin posture for each patient should be combined and listed as "total degrees" in each table. This method was deemed the most logical mechanism for reporting and for comparison purposes, not only of groups within this stud, but also for comparison with other studies as well.

RESULTS

Of the 630 patients with ocular torticollis, this author found 330 patients with incomitance (52.4%), 120 patients with nystagmus (19%), 69 patients with congenital esotropia with ocular posture (10.9%), and 27 patients with torticollis permitting foveal fixation (4.3%). Two patients had spasmus nutans (0.3%). No patients were found to have ocular torticollis relating to cosmetic reasons or ocular motor apraxia. Twenty-five patients were determined to have ocular torticollis in association with medical or neurologic conditions (4%). An unexpected high number, 57 patients (9%), were found with ocular torticollis without any apparent or obvious reason.

Table I summarizes the diagnostic groups described by Kushner[20] and gives comparison figures for this study. Kushner described a higher percentage of patients with incomitance and with permitting foveal fixation (eg, ptosis, fibrosis syndrome, Moebius syndrome). He noted a total of 8 patients with ocular torticollis as a result of cosmetic reasons, ocular motor apraxia, or astigmatism. This study revealed a similar incidence of nystagmus as an etiology for torticollis. In this study, however, a higher percentage of patients presented with congenital esotropia with ocular posture than described by Kushner.[20] The 57 patients with unexplained torticollis comprised 9% of the total group. A comparable group was not described in Kushner's study.

Table II summarizes those patients with ocular posture secondary to incomitance, with nearly l0% of the total group demonstrating superior oblique palsy. Acquired horizontal incomitance, including asymmetric surgery, A and V pattern, and 6th nerve palsy, accounts for nearly 30% of the torticollis pool. The groups of Duane and Brown syndrome combined for slightly more than 11% of the torticollis pool.

Several diagnostic categories were extracted from the total patient pool to allow for the calculation of incidence of each group. The office computer system in use at the time of this study did not allow for the recording of more than 3 diagnoses per office visit. This limits the accuracy of

TABLE I: ETIOLOGY OF TORTICOLLIS

FACTOR	KUSHNER STUDY[20] NO. OF PATIENTS (%)	PRESENT STUDY NO. OF PATIENTS (%)
Incomitance	118 (62.7)	330 (52.4)
Nystagmus	38 (20.2)	120 (19)
Congenital esotropia with ocular posture	12 (6.3)	69 (10.9)
Permitting foveal fixation	10 (5.3)	27 (4.3)
Cosmetic	4 (2.1)	0 (0)
Ocular motor apraxia	3 (1.6)	0 (0)
Spasmus nutans	2 (1)	2 (0.3)
Astigmatism	1 (0.5)	0 (0)
No apparent reason for torticollis	NA	57 (9)
Ocular torticollis with associated medical/neurologic conditions	NA	25 (4)

TABLE II: OCULAR POSTURE SECONDARY TO INCOMITANCE

INCOMITANCE	NO. OF PATIENTS	% OF 630 PATIENTS WITH TORTICOLLIS
Vertical		
Superior oblique palsy	59	9.4
Inferior oblique palsy	6	1.0
Brown syndrome	25	4.0
Double elevator palsy	9	1.4
Superior rectus palsy	1	0.2
Horizontal		
Duane syndrome	46	7.3
Acquired horizontal incomitance – asymmetric surgery	48	7.6
Acquired horizontal incomitance – A/V pattern	116	18.4
Acquired horizontal incomitance – sixth nerve palsy	1	0.2
Third nerve palsy	1	0.2
Torsional incomitance	4	0.6
Total	330	

the data to some extent, because there were frequently 4 or more diagnoses recorded for each visit. For example, a child with myopia, torticollis, nystagmus, esotropia, and amblyopia could not be categorized with every disorder. Therefore, the diagnoses in the central column in Table III are necessarily underreported, and the incidence of torticollis for each group may be slightly higher than would be calculated if all diagnoses were available.

TABLE III: TORTICOLLIS INCIDENCE*

	NO. OF PATIENTS		INCIDENCE OF TORTICOLLIS
Superior oblique palsy	59		
Inferior oblique palsy	6		
Double elevator palsy	9	PATIENTS WITH "EYE MUSCLE PALSY"	
Superior rectus palsy	1		
Sixth nerve palsy	15		
Third nerve palsy	1		
Total	**91**	**279**	**33%**
Brown syndrome	25	86	30%
Duane syndrome	46	128	36%
ET-A pattern	29	124	
ET-V pattern	40	213	
XT-A pattern	16	129	
XT-V pattern	31	144	
All patterns	**116**	**610**	**19%**
Nystagmus	120	290	41%
Infantile esotropia	69	812	8.5%
Ptosis	16	343	7%

*Total No. of office visits = 15,168
Total No. of patients seen = 11,299
Total No. of patients with torticollis = 630
Incidence of torticollis (630/11,299) = 5.6%

The classification "eye muscle palsy" was identified 279 times among 11,299 patients. A total of 91 patients presented with torticollis and eye muscle palsies, including 59 superior oblique palsies, 6 inferior oblique palsies, 9 double elevator palsies, 1 superior rectus palsy, 15 sixth nerve palsies, and 1 third nerve palsy. Therefore, the incidence of torticollis in the presence of eye palsy was 91 of 279, or 33%. Of the 86 patients examined with Brown syndrome, 25 had torticollis (30%). These findings were iden-

tical to Kushner's[20] group of patients with Brown syndrome. Nine of the 25 patients with Brown Syndrome demonstrated head tilt, higher than the 5% found by Kushner, and similar to a study by Urist.[25] Of the 128 patients with Duane syndrome, 46 had torticollis (36%). Of the 610 patients with A or V pattern, 116 demonstrated torticollis (19%). These results are higher than the 9% reported by Kushner[20] or the 11% reported by Campion.[26]

The presence of torticollis with nystagmus occurred in 120 patients of 290 with a diagnosis of nystagmus (41%). There were 812 patients diagnosed with congenital or infantile esotropia, and 69 diagnosed with congenital esotropia with ocular posture (8.5%). A number of infants in the A and V pattern category had infantile esotropia, but did not develop the full "syndrome" including dissociated vertical deviation, excyclotorsion, latent nystagmus, and torticollis, and therefore this category may be underreported. In his 1987 Edward Jackson Memorial Lecture, von Noorden[27] reported that only 26 (6%) of 406 patients with essential infantile esotropia demonstrated anomalous head posture, similar to the 8.5% incidence found in this study.

The major causes for ocular posture secondary to incomitance are summarized and explained in detail in subsequent sections of this study, followed by reviews of nystagmus, congenital esotropia with ocular posture, permitting foveal fixation, no apparent reason for torticollis, torticollis associated with medical and neurologic conditions, and superior rectus palsy, third nerve palsy, and spasmus nutans.

SUPERIOR OBLIQUE PALSY

Fourth nerve palsy is the most common isolated cyclovertical eye muscle palsy.[28-31] Congenital fourth nerve palsy occurs because of a defect in the nucleus or the motor portion of the nerve. Closed head trauma is the chief cause of acquired fourth nerve palsy, but other etiologic factors, including cerebrovascular accident, diabetes, intracranial tumors, ethmoiditis, mastoiditis, hematoma, aneurysm, and orbit surgery, have also been reported.[28-43] Plagiocephaly with superior oblique weakening, which is not a true palsy, but does exhibit a similar clinical presentation, has also been reported.[5] Despite extensive clinical and laboratory testing, the etiology of fourth nerve palsy may remain obscure.[30-34,41,42] In his study of 121 children ranging in age from birth to 16 years, Harley[42] was unable to determine the origin of fourth nerve palsy in 67% of his patients. Kodsi and Younge[43] found trauma to be the most common etiology for acquired cranial nerve palsies in a study of 160 pediatric patients at the Mayo Clinic. Trauma was the predominant etiology in the oculomotor (40%), trochlear (37%), and abducent palsy group (42%), as well as the multiple palsy group (56%).

The usual clinical presentation of superior oblique palsy is a head tilt

away from the side involved with the superior oblique palsy, along with a chin depression posture. Not all patients, however, demonstrate typical head posture. Some may present with absence of any discernable head tilt or chin depression, while others tilt toward the side with the superior oblique palsy.[31,44,45] Spontaneous absence of torticollis is most common in the presence of poor vision in one eye, where there is no need to develop a posture to restore binocular vision.[44]

The Bielschowsky head tilt test is of the utmost importance in diagnosis of fourth nerve palsy.[46] Parks[31,45] popularized his "three-step test," which includes the Bielschowsky head tilt test, to distinguish a paretic oblique muscle or vertical rectus muscle. The first step, which asks if there is a hypertropia in the primary position, immediately eliminates 4 of the 8 cyclovertical muscles if a hypertropia is present. Step 2 determines whether the vertical deviation increases in left or right gaze, thus eliminating 2 more possible muscles. After step 2, only 2 muscles remain, and they must both be either intorters or extorters, never 1 intorter and 1 extorter. It is in step 3 that the Bielschowsky head tilt test then differentiates which of the 2 remaining muscles is at fault.

The Parks three-step test is the standard for diagnosing isolated cyclovertical palsies. However, Kushner[47] has described conditions that may lead to diagnostic errors with the three-step test, such as contractures of the vertical rectus muscles, previous extraocular muscle surgery, myasthenia gravis, skew deviation, dissociated vertical divergence, paresis of more than 1 vertical muscle, and small nonparalytic vertical deviations seen in association with horizontal strabismus. The three-step test is also useful in the diagnosis of bilateral masked superior oblique palsy.[48]

Kraft and associates[49] analyzed compensatory head posture, before and after surgery, in a group of 381 patients with the diagnoses of lateral rectus paresis, superior oblique paresis, Duane syndrome, Brown syndrome, and congenital nystagmus. Superior oblique palsy was found in 139 patients. Ninety-nine patients (71%) presented with compensatory head posture. Robb[50] described 63 patients with idiopathic superior oblique palsies, all of whom presented with unilateral palsies. One of Robb's patients was found to have masked bilateral superior oblique palsy after surgery for an apparent unilateral palsy.

The superior oblique palsy group in the current study was composed of 59 patients, summarized in Table IV and Table XVIII. Thirty-one male and 28 female patients presented with an average torticollis of 13.9°. A face turn was measured in 39 patients, a head tilt in 46 patients, and a chin posture surprisingly in only 6 patients. The average age was 14 years and 6 months, with a range in age from 2 to 76 years. In all tables, age is recorded as years plus months.

TABLE IV: SUPERIOR OBLIQUE PALSY

CASE	INIT	SEX	AGE	TURN	TILT	CHIN	TOTAL (°)°	OTHER DIAGNOSIS	SURGERY	AGE
1	JA	F	18+9	5				5	Accommodative ET	
2	DB	M	9+2	4	6		10	ET		
3	AB	F	12+11	5	5		10	Excyclotorsion 4°		
4	GB	F	76+0	4			4			
5	JB	M	51+0	5	7		12		ET Surgery	0+3
								XT Surgery	0+4	
								XT Surgery	25+0	
								RE-REC RLR 5, RE-Resect RMR 6, REC RIO 12	47+6	
6	BB	F	2+8	20			20		REC RIO 14	1+4
								REC LIO 14	2+5	
7	MB	M	2+11		15		15			
8	LC	F	36+10		5		5	MVA	LSO Tuck	35+7
								REC RIR 8	36+6	
9	RG	M	4+4		8		8			
10	TC	M	5+6	8	8		16		REC RIO	1+7
								REC LIO 14	4+6	
11	RC	M	3+0		10		10			
12	AC	F	5+5		12		12			
13	RD	F	14+4		3		3	Excyclotorsion 4°		
14	JD	M	12+9	12			12	Amblyopia		
15	CP	F	14+3	20			20	XT		
16	JF	M	4+7	10	10		20		REC LIO 14	4+5
17	CF	F	2+0	10	12		22	ET	REC MROU 5.5, REC RIO 14	1+6
									REC LIO	2+0

TABLE IV (CONTINUED): SUPERIOR OBLIQUE PALSY

CASE	INIT	SEX	AGE	TURN	TILT	CHIN	TOTAL (°)°	OTHER DIAGNOSIS	SURGERY	AGE
18	AG	F	4+1	10			10			
19	RH	M	62+5	8	15		23	Excyclotorsion 10°		
20	JH	M	10+0	5	25		30		REC LIO 14	8+6
								REC RIO 12	9+4	
21	MH	M	6+1	8			8	Ptosis OD	Fasanella, REC RIO 14	3+11
								REC LIO 14	5+10	
22	MK	F	4+2	5	7		12			
23	PK	M	12+10	6	12		18	Noonan syndrome	REC LIO 10	12+2
24	SK	F	6+9	6	8		14		REC MROU 6	1+1
								REC IOOU	4+10	
25	ML	F	7+2	8	15		23	Rubinstein-Taybi syndrome		
26	AL	F	9+11	5	10		15		REC LIO 14	5+0
27	BM	M	8+10		8	10	18			
28	JM	F	17+1	6	6	4	16	VX(T)	REC RIO 12	16+9
29	KM	F	7+5		8		8	X(T)		
30	JM	M	62+11	8	8		16	MVA	Strabismus surgery 1966, '68	
								Bilateral Harada-Ito	62+1	
31	AM	M	4+2		15		15		REC MROU 6.5	0+9
32	MM	M	9+8		4	4	8	E(T)	REC RIO 12	0+11
								RSO Tuck	3+10	
33	MM	F	13+5		16		16			
34	CM	M	3+11		15		15			
35	JM	M	9+4	6	6		12	X(T)		
36	KM	F	13+1	10	5		15			
37	EM	F	68+9	10	5		15	Excyclotorsion 6°	RSO Tuck	68+5
38	SM	F	4+9	8	4		12		REC LIO 14	4+8

TABLE IV (CONTINUED): SUPERIOR OBLIQUE PALSY

CASE	INIT	SEX	AGE	TURN	TILT	CHIN	TOTAL (°)*	OTHER DIAGNOSIS	SURGERY	AGE
39	JN	M	34+6		7		7	XT,HT	Resect LLR 7, REC LIO 8	12+3
								REC RIO 14, Resect LMR 5, REC LLR 7		33+11
40	AO	F	19+11		25		25	ET and RHT		
41	JO	M	6+1	5	5		10	E(T)		
42	BP	F	4+11	20			20	Down syndrome	REC RIO	4+5
43	BQ	M	3+11		15		15			
44	LR	F	9+6		5		5	ET		
45	CR	F	3+5		10		10	LH(T)		
46	BS	M	10+8	10			10			
47	BS	M	1+0	5	10		15		REC LIO 14	1+6
48	AS	F	4+6	5	7		12			
49	BS	M	36+7		5		5	Bicycle accident	REC LIO 14	36+1
50	TS	M	3+3	6	6		12		REC MROU 6	1+5
								Resect LROU 7, REC IOOU 14	2+2	
51	ES	M	4+5	4	10		14		REC RIO 14	2+8
52	ES	F	3+8	6		5	11	X(T)		
53	NS	F	32+2			7	7		REC LSR 8, Resect LLR 5	32+2
54	ES	F	19+10	10	7		17	MVA, diplopia		
55	KT	M	5+6	15			15			
56	CW	M	17+1	5	6	3	14		REC RIO 14, REC LIR 3.5	16+5
57	SW	M	5+5	20			20	ET and ambylopia, OD		
58	AW	M	3+5		15		15		LSO Tuck	2+11
59	CW	M	3+5	20	20		40	Optic nerve hypoplasia OD Septo-optic dysplasia, VXT, Congenital 4th nerve palsy	REC LROU 6.5, REC IOOU 12	5+0

*Average degrees 13.9.

Not all patients in this group required surgery, either because the torticollis was not symptomatic or severe, or because the parents or the patient did not elect surgery. The procedures performed and age at surgery are compiled in Table IV. Of those patients selecting surgery, 27 inferior oblique recession procedures were performed, as were 4 superior oblique tucks, 2 inferior rectus recessions, and 1 superior rectus recession. One bilateral Harada-Ito procedure was performed, with an advancement of the anterior one half of each superior oblique tendon to a position at the superior border of the lateral rectus, 8 mm posterior to the insertion.

The etiology was obscure in most patients, with documentation of 2 motor vehicle accidents and 1 bicycle accident, which shattered the rider's helmet. Patient 19 sustained a head injury at work in another state, and although corrective surgery was recommended, the patient was lost to follow-up. The spectrum of superior oblique palsy classified by Knapp[28] was not apparent in reviewing this series of patients, hence the surgical options were limited to the above described procedures.

INFERIOR OBLIQUE PALSY

Inferior oblique palsy is the rarest of all extraocular muscle palsies.[51] In 1977, Scott and Nankin[52] reported the first series of patients with isolated inferior oblique paresis. An intrasheath tenotomy on the overacting ipsilateral superior oblique was performed on 6 patients, with excellent results. Olivier and von Noorden[51] performed tenectomy procedures on 6 patients, 3 of whom developed progressive paralysis of the tenectomized superior oblique muscle, with deterioration of binocular vision. Reese and Scott[53] performed a tenotomy on l6 patients. Six of their patients also required a contralateral superior rectus recession because of concerns with losing binocularity with the tenectomy procedure. Only 2 patients were classified as overcorrected with postoperative superior oblique palsy in a 5-year follow-up period. Pollard[54] described 25 patients with inferior oblique palsy, 2 of which were bilateral. Twenty patients in his group of 23 with unilateral palsy presented with a head tilt to the side of the paretic inferior oblique muscle. Nineteen required surgery, with superior oblique tenotomy or with contralateral superior rectus recession. Only 2 developed superior oblique palsy postoperatively, requiring subsequent surgery. Seventeen were able to fuse in the primary position, without head tilt, after surgery.

Kushner[20] reported that each of his 7 cases of vertical incomitance from inferior oblique palsy had significant head tilt toward the affected side. Five were iatrogenic, and 2 were presumed idiopathic inferior oblique palsy.

Tables V and XVIII summarize the inferior oblique palsy group found

TABLE V: INFERIOR OBLIQUE PALSY

NO.	INIT	SEX	AGE	TURN	TILT	CHIN	TOTAL(°)*	ETIOLOGY/SURGERY	AGE
1	CA	F	45+11			12	12	MVA, hypertropia	
2	AL	F	41+11	22			22	Arterial ligation (epistaxis)	
3	MM	F	13+4	3	15		18	RIO extirpation	8+11
4	GM	F	6+10	12			12	Unknown etiology	
5	CS	M	10+4	7			7	Hypertropia	
							ET surgery	0+6	
							Ptosis surgery	0+9	
6	MB	M	7+7	20	10		30	Premature, cerebral palsy	

*Average degrees 16.8.

in the present study, including 2 male and 4 female patients, ranging in age from 6 years 10 months to 45 years 11 months, with an average age of 21. The average torticollis was 16.8°. Only 2 of the 6 patients demonstrated a head turn to the affected side. Patient 3 was first examined at 8 years of age, with marked overaction of the right inferior oblique muscle. An extirpation procedure of the right inferior oblique resulted in marked underaction of the inferior oblique, with a 15° head tilt to the right. There was a right hypotropia of 25 diopters in left head tilt, and 25 seconds of stereoacuity on the random dot test in the position of torticollis.

Patient 2 had 2 emergency procedures for epistaxis, which could not be controlled in the conventional manner. After a ligation of the right internal maxillary artery, the bleeding persisted, requiring ligation of the anterior and posterior ethmoidal arteries. These procedures led to an underaction of the right inferior oblique, hypotropia in left gaze, and a marked face turn of 22° to the left, with gaze right preference. There was also a large area of facial anesthesia. Patient 6 had presumed congenital inferior oblique palsy and cerebral palsy and did not require surgery.

BROWN SYNDROME

Brown[55,56] is credited with the first description of the eye motility defect, known as Brown syndrome, demonstrated by an inability to raise the adducted eye above the midline horizontal plane. There is usually less restriction in the primary position, and little or no limitation of elevation in abduction of the affected eye. There is a slight downshoot of the adducting eye, simulating superior oblique muscle overaction. In association with the restriction of elevation is an occasional widening of the palpebral fissures in adduction. Exodeviation in upgaze in a V pattern[57,58] is present.

Initially, Brown[55,56] felt there was a simulated inferior oblique muscle palsy due to a deficit in innervation to the inferior oblique, with secondary contracture of the anterior sheath of the superior oblique tendon. This was proved erroneous by electromyography.[57] Brown[59] redefined the tendon sheath syndrome as being more complex than initially suspected and described the true sheath syndrome as congenital, permanent, and associated with a positive traction test in adduction. Most patients do not demonstrate a vertical misalignment in the primary position, but if hypotropia is present in the involved eye, there may be a compensatory chin up posture to allow fusion in downgaze.[57]

Various superior oblique surgical weakening procedures have been described, including removal of the superior oblique tendon sheath. The term "sheath," however, is a misnomer. There exists an intermuscular septum surrounding the superior oblique tendon, but the "sheath" is nonexistent. When the Tenon's capsule and intermuscular septum are

hooked without directly observing the superior oblique tendon, the thickened, redundant layers of Tenon's capsule and intermuscular septum on the tip of the muscle hook are interpreted as a "sheath."[57,60] The preferred surgical technique requires leaving Tenon's capsule intact and unopened, beyond 10 mm from the limbus, to avoid orbital fat. The intermuscular septum must remain intact except at the placement of the tenotomy. Superior oblique tenectomy temporarily improves the superior oblique tightness in Brown syndrome but frequently leads to a palsy of the tenectomized superior oblique, with opposite vertical tropia and subsequent torticollis.[57,60-65]

Parks and Eustis[65] recommended simultaneous superior oblique tenotomy and ipsilateral inferior oblique recession for true Brown syndrome. This was suggested to avoid reoperation for iatrogenic superior oblique palsy, which frequently occurred when the superior oblique tenotomy was the sole procedure. Underaction of the inferior oblique may persist in some patients. The silicone expander has been advocated by Wright and associates.[66] The silicone spacer is sutured to the cut ends of the superior oblique tendon, nasally to the superior rectus muscle, after a tenotomy procedure. This technique offers more predictable results, without requiring inferior oblique surgery. Major advantages of this technique include controlled elongation of the superior oblique tendon, preservation of fusion and stereopsis, which may be compromised when iatrogenic superior oblique palsy occurs, and preservation of inferior oblique function through avoiding surgery on that muscle.

Tables III, VI, and XVIII summarize the findings of 25 patients with Brown syndrome. Ten male and 15 female patients were found to have an average torticollis of 9.4°. The average age was 11 years, 3 months,with an age range of 2 to 49 years. Torticollis was found in 25 of 86 patients, or in 30% in the group diagnosed with Brown syndrome. The incidence of torticollis was higher than that reported by Kraft and associates,[49] who described a compensatory head posture in 6 of 35 patients with Brown syndrome (17%). In the current group of 25 studied, 16 patients experienced face turn, 9 had a head tilt, and 7 demonstrated chin postures. Of the 5 patients who elected surgery, all had tenotomy procedures and 3 had surgery before their inclusion in this study. Two patients received surgery after their inclusion in the study. The average degrees of torticollis of those patients who had surgery was 9.2°, with a range of 6 to 14°.

DOUBLE ELEVATOR PALSY

Third nerve palsy may be partial or complete.[31-34,39,43] Double elevator palsy occurs when both elevators of the eye are affected. There may be varying degrees of ptosis or pseudoptosis. Pseudoptosis occurs when the upper

TABLE VI: BROWN SYNDROME

NO.	INIT	SEX	AGE	TURN	TILT	CHIN	TOTAL (°)*	SURGERY	AGE
1	AB	F	4+6			6	6		
2	RC	F	2+0	10			10		
3	GD	F	2+5	15	5		20		
4	PD	M	7+9			10	10		
5	MD	F	12+1	5			5		
6	OF	F	4+7	4			4		
7	CF	F	12+4		7	5	12		
8	KH	F	5+8		6		6	RSO tenotomy	2+6
9	LH	F	2+3			5	5		
10	JH	F	11+11			10	10		
11	RJ	M	47+0			10	10	RSO tenotomy, REC LROU	47+2
12	ML	M	11+1	15			15		
13	AL	F	5+1	5			5		
14	KL	F	6+6		6		6		
15	DM	M	6+5	5			5		
16	MM	F	49+0	10	10		20		
17	JM	M	23+6	3			3	(Bilateral Brown syndrome)	
18	SM	F	7+1	6	2		8		
19	MP	M	10+8			12	12		
20	RQ	F	5+7	10			10		
21	RS	F	12+6	6			6	RSO tenotomy	6+0
22	GS	M	5+6	5			5		
23	JS	M	1+10	5	5		10	RSO tenotomy	1+4
24	JS	M	8+2	8	6		14	LSO tenotomy	7+5
25	CS	M	12+8	15	3		18		

*Average degrees 9.4.

eyelid follows the hypotropic eye, but when the hypotropic eye fixates, the pseudoptosis disappears. True ptosis may occur in association with pseudoptosis.

In double elevator palsy, the traction test is usually negative. In the case of a negative traction test, the treatment of choice is the Knapp procedure,[67,68] a full tendon transfer of the medial rectus and lateral rectus to the insertion of the superior rectus. When there is double depressor palsy, the horizontal rectus muscles are transferred to the insertion of the inferior rectus muscle. In double elevator palsy, the elevation of the eye is restored only to midline, but the hypotropia is improved or eliminated. When a full tendon transfer was performed for double elevator or double depressor palsy, Knapp[68] reported an average correction of 38 diopters in primary position, and movement of 25° in the field of action of the paretic muscles from primary position.

In the presence of a positive traction test with an absent or poor Bell's phenomenon, Scott and Jackson[69] advocate recession of the inferior rectus muscle as the initial procedure and recommend a full Knapp procedure if the traction test is negative.

Double elevator palsy results in the present study are summarized in Tables VII and XVIII. Three male and 6 female patients, ranging in age from 4 months to 73 years 6 months (average age, 19 years 1 month), were examined. The average torticollis found was 19.3°, with 7 of the 9 patients demonstrating a face turn, 4 with a head tilt, and 4 with a chin posture. Patient 4 presented with elevator palsy induced at cataract surgery. This might be more accurately categorized as a pseudo-double elevator palsy, induced by complications of periocular anesthetic injection to the extraocular muscles. Two patients required only inferior rectus recession for their palsy. A Knapp procedure and a Fasanella procedure for residual ptosis was performed in 1 patient, in addition to 2 other procedures for complicated strabismus.

DUANE SYNDROME

Duane syndrome, as described by Duane[70] in 1905, is more precisely designated the Stilling-Turk-Duane syndrome because of earlier descriptions compiled by Stilling[71] and Turk.[72] Duane emphasized retraction, an essential feature of the syndrome. Clinical presentation includes retraction of the globe and a horizontal motility defect. Huber[73] described three types of retraction syndromes based on electromyography.

Type I includes absent abduction, normal or restricted adduction with globe retraction, and palpebral fissure widening on attempted abduction. Electromyographic findings include absent electrical activity in the lateral rectus on abduction, but paradoxical activity on adduction.

TABLE VII: DOUBLE ELEVATOR PALSY

NO.	INIT	SEX	AGE	TURN	TILT	CHIN	TOTAL(°)*	SURGERY	AGE
1	BB	M	7+10		25		25		
2	NC	F	7+7	12		7	19		
3	DC	F	0+4	12		10	22		
4	EC	F	73+6	5			5	Iatrogenic—at cataract surgery	
5	DB	F	44+6	10		5	15	REC LIR 8	44+1
6	BL	M	4+11	20	8		28	Frontalis suspension, OD	1+1
7	AM	F	7+6			15	15	REC RIR 8	0+6
								Frontalis suspension, OD	0+8
8	CO	F	9+4	12	12		24	REC LROU 8.5, REC RIO 7	1+1
								Knapp OS	2+5
								Fasanella, OS	5+8
								RE-REC RLR 3.5, up 4mm.	6+7
								Resect RMR 8, up 2mm.	
9	MP	M	14+8	15	6		21	LMR and LLR	8+3
								REC LLR 7, Resect LMR 6	9+10

*Average degrees 19.3

Type II includes exotropia with restricted adduction and abduction, with retraction of the globe on adduction. Electromyographic findings include electrical activity with contraction of the lateral rectus on both abduction and adduction.

Type III includes severe restriction of both abduction and adduction, with orthophoria or minimal esophoria or exophoria, and retraction of the globe on adduction. Widening of the palpebral fissures occurs on abduction. The electromyographic findings reveal co-contraction of the horizontal rectus muscles on both abduction and adduction.

Clinical presentation is variable,[74] and observable retraction ranges from minimal to conspicuous. An upshoot, downshoot, or both may be present in the adducting eye, simulating oblique muscle overactions. Upshoot and downshoot of the affected eye have been described as the "leash" or "bridle" phenomenon and are caused by a tight lateral rectus slipping over or under the globe with subsequent anomalous vertical eye movement.[75-78]

Bloom and associates[75] described minimal vertical displacement of the lateral rectus in relation to the orbit, with magnetic resonance imaging in 2 patients with upshoot and downshoot, suggesting a modification of the bridle effect theory. Von Noorden[79] noted that the globe slips beneath or above the horizontal rectus muscles, which maintain their vertical position with reference to the orbit wall. Von Noorden believed that the magnetic resonance imaging (MRI) study provided further proof for the bridle theory. Therefore, with slight elevation or depression of the eye from primary position, a co-contraction occurs, with subsequent upshoot or downshoot of the globe.

Duane retraction syndrome may present bilaterally[80-86] and occurs more frequently in females.[81,82,84-86] Associated findings include the Klippel-Feil anomaly in 3% to 4% of patients[81-82] and the congenital labyrinthine deafness in 11% of patients.[81] The Wildervanck syndrome[87] includes Duane retraction syndrome, Klippel-Feil anomaly, and congenital labyrinthine deafness. A comprehensive list of associated findings, anomalies, and syndromes has been published.[74]

The majority of patients with Duane syndrome have straight eyes in the primary position during infancy and childhood. In time, however, some patients with type I Duane syndrome will develop an esodeviation in the primary position, with restricted abduction in the involved eye. A compensatory head posture develops, with a face turn toward the side of the involved eye, in order to maintain binocular vision. Surgery for Duane syndrome is not usually necessary unless a significant head posture has developed.[71] Because the surgery cannot correct the anomalous innervation, the purpose of surgery is to restore the eyes to a parallel alignment in

primary position, and to reduce the compensatory face turn. Surgery for Duane syndrome was first suggested by Nutt,[88] who recommended medial rectus recession in the involved eye with uncomplicated Duane syndrome and abduction limitation. A maximal recession of the tight medial rectus muscle has been recommended,[74] along with a Z-tenotomy when necessary. Lateral rectus resection is discouraged because of increased restriction following this procedure. If there is marked retraction of the globe, along with marked narrowing of the fissures on attempted adduction, a recession of the lateral rectus at the time of recession of the medial rectus can be performed.

Either partial or total vertical muscle transposition to the border of the lateral rectus muscle has been recommended,[89-91] but this has also been discouraged by others[74] because of further limitation to adduction. However, Molarte and Rosenbaum[92] demonstrated a 77% improvement in esotropia, and a 100% improvement in face turn in 13 patients who had full transposition of the superior and inferior rectus muscles to the lateral rectus insertion.

In patients with bilateral Duane syndrome, secondary exotropia has been known to occur after bilateral medial rectus recessions.[93] Simultaneous medial rectus and lateral rectus recessions may be required to reduce this complication. Despite large medial rectus muscle recessions, Pressman and Scott[94] did not report any significant overcorrections, nor did they gain significant increase in abduction. Nelson,[95] however, described severe adduction deficiency following large medial rectus recessions in 2 patients, 1 with a 7 mm medial rectus recession and a second with a 5 mm recession followed by a 2 mm re-recession.

Eisenbaum and Parks[96] addressed the leash effect of overelevation and overdepression of the involved eye in adduction, wherein the tight lateral rectus muscle slips over the surface of the eye in adduction, simulating inferior oblique overaction, or superior oblique overaction. The leash effect of the lateral rectus was successfully eliminated by placing a permanent posterior fixation suture 14 mm from the insertion, at the superior and inferior one third of the muscle. Von Noorden and Murray[97] also described success with posterior fixation of the horizontal rectus muscles in 5 patients with upshoot and downshoot in Duane retraction syndrome.

In discussing the Eisenbaum and Parks paper, Jampolsky[98] proposed a "Y" splitting of the lateral rectus tendon insertion to reduce the upshoot and downshoot in Duane syndrome. Rogers and Bremer[99] described a marked reduction in upshoot and downshoot in their series of patients with a "Y" splitting of the lateral rectus, with or without recession of the ipsilateral medial rectus muscle.

Duane syndrome is summarized in Tables III, VIII, and XVIII. Of

TABLE VIII: DUANE SYNDROME

NO.	INIT	SEX	AGE	TURN	TILT	CHIN	TOTAL(°)°	SURGERY	AGE
1	MA	F	27+7	12			12	REC LMR 8	26+2
2	BA	M	11+5	15	10		25		
3	KB	M	9+11	5			5		
4	DB	M	7+4	10	5		15		
5	CB	M	5+1	4			4		
6	SB	F	5+7	5			5	REC LMR 8	3+9
7	DC	M	7+10	10	22		32	REC LMR 8	1+7
8	CC	F	2+6	15			15		
9	HC	F	9+4	12		4	16		
10	MD	M	0+7	5			5		
11	FC	F	9+4	10			10		
12	SD	M	3+0	25			25		
13	RD	M	1+10		5		5		
14	AE	F	7+2	12		5	17	REC LMR 9	1+8
15	KF	M	6+0	10			10		
16	AF	F	3+8	12			12		
17	DF	F	3+7	5	4		9		
18	LH	F	2+11	10			10		
19	SH	F	8+1	13			13		
20	AI	F	3+11	25			25	REC MROU 7, REC LLR 6	3+11
21	RJ	F	9+5	15			15		
22	BK	F	9+9	7			7		
23	DJ	M	10+6	5			5		
24	TK	F	12+5	20		8	28		
25	BL	F	2+11	10		8	18	REC LMR 8	1+0
26	ML	M	15+10	8	10		18		

TABLE VIII (CONTINUED): DUANE SYNDROME

NO.	INIT	SEX	AGE	TURN	TILT	CHIN	TOTAL(°)*	SURGERY	AGE
27	KL	F	8+11	15			15		
28	CL	F	11+5	10		7	17	REC LMR 7	7+5
29	TL	M	5+6	20	8		28		
30	JM	M	14+9	15	10	5	30		
31	EM	F	10+1	10			10		
32	SM	F	4+7	10			10		
33	DM	M	5+11	15	5		20		
34	BN	F	17+10	5			5	REC LROU 7	10+0
35	DN	M	34+8	12			12	REC LMR 7.5	24+0
36	SS	M	4+9	7			7	REC MROU 7, RE-REC MROU	4+0
37	CS	M	11+1	15			15		
38	JS	M	10+1	5			5		
39	SS	M	7+5	5	7		22	REC LMR 8	3+4
40	MT	F	15+2	8			8		
41	WW	F	7+6	7			7		
42	JV	M	5+0	30			30		
43	AW	F	14+3	10			10		
44	RW	F	10+7	12	6	5	23	Resect LMR 6, Y-Split RLR Type 2 Duane syndrome	3+7
45	TW	M	1+8	5			5		
46	JY	M	17+3	20			20	Y-Split RLR 10, REC 3 Resect RMR 6, REC LLR 8 RE-REC RLR 10, LLR 5 RE-REC LLR 3, RE-Resect RMR 5 REC RSR 7	9+11 12+10 17+3

*Average degrees 14.3.

128 patients with the diagnosis of Duane syndrome, 46 presented with torticollis, resulting in a torticollis incidence of 36%. Kraft and associates[49] found compensatory head posture in 62 of 91 patients, or 68% of those with Duane syndrome. The current series reflects 22 male and 24 female patients, ranging in age from 7 months to 34 years 8 months, with an average age of 9 years 1 month. The average torticollis was 14.3°, and all 46 patients demonstrated a face turn. Eleven patients also demonstrated a head tilt, and 6 presented with a chin posture. Thirteen patients required surgery, including 2 who received a "Y" splitting procedure. Each of the 8 unilateral cases involved the left eye. The 3 remaining cases were bilateral. Patient 44 achieved a fair response to the "Y" splitting. Patient 46, with bilateral Duane syndrome and evidence of congenital aberrant innervation, required 3 procedures to provide satisfactory alignment. The face turn is evident at times, but the patient has markedly improved from his initial presentation.

ACQUIRED HORIZONTAL INCOMITANCE

Kushner[20] described 7 patients with acquired horizontal incomitance, 4 of whom had asymmetric surgery. Three patients had overcorrected exotropia, with esotropia in the primary position but experienced fusion with a compensatory head posture. The fourth patient had esotropia with a recession of the right medial rectus and an undercorrection, presenting with a compensatory head posture. After a recess-resect procedure on the fellow eye, the torticollis resolved, and stereoscopic vision was achieved.

Kushner described 48 patients with A or V patterns, but only 4 had a chin elevation or depression posture, which he reported as a 9% incidence of torticollis. Of the 44 with A or V pattern without torticollis, the deviation was so marked that fusion was not possible in 35 patients. The remaining 9 patients had deviations of less than 10 D but failed to demonstrate fusion. The 3 patients with sixth nerve palsy were listed in this study but were not discussed.

Acquired horizontal incomitance was included in this study, but because of the number of patients reviewed, 3 separate categories were necessary for tabulating acquired horizontal incomitance. Table IX includes data on acquired horizontal incomitance as well as asymmetric surgery. Table X includes A or V pattern, and Table XI includes sixth nerve palsy.

ACQUIRED HORIZONTAL INCOMITANCE/ASYMMETRIC SURGERY

This group of 48 patients (22 male and 26 female) is summarized in Tables IX and XVIII. Age range was 3 years 2 months to 82 years 7 months (average, 21 years 3 months). Average torticollis was 13.3°. A face turn

TABLE IX: ACQUIRED HORIZONTAL INCOMITANCE

NO.	INIT	SEX	AGE	OD	OS	TURN	TILT	CHIN	TOTAL(°)°	ETIOLOGY	AGE
1	JA	M	13+11			6	6		12	Unknown	
2	JA	M	75+2	20/40	20/125	5			5	Detached retina	71+6
3	EA	F	3+2	20/40	20/400	8			8	REC MROU 5, OS amblyopia	0+9
4	CB	F	14+8	20/70	20/60			3	3	REC MROU 5.5, resect LROU	1+0
			20/40	20/25						RE-REC RLR, resect RMR 6	13+8
5	LB	F	8+1	20/40	20/70		8		8	Unknown	
6	VB	M	7+6			24	16		40	REC MROU 5	4+0
										REC IOOU 14	5+11
7	VB	M	6+8	20/30	20/25		20		20	REC MROU 5	4+0
										REC RIO 14, LIO 10	6+0
8	JB	F	5+0	20/30	20/30		12		12	REC MROU 6	0+6
										REC IOOU 12, resect LROU 7	1+3
9	AB	F	4+1	20/40	20/40	7		7		REC MROU 5.5	4+0
										REC IOOU 14	
10	NB	M	6+9	20/30	20/40	15			15	REC MROU 5 (slipped MR)	0+7
										Advance LMR, REC RLR	1+6
										REC LLR 8, resect LMR 18	1+10
										REC LMR 5, REC LIO	2+9
										REC anteriorize LIO	
11	FB	M	13+7	20/30	20/40		10		10	REC MROU 6	1+0
										REC LROU 5, REC IOOU	10+6
12	EC	M	56+2	20/20	20/50	7			7	OS, detached retina	54+8
										OD XT, REC RLR 6	56+1
										Resect LMR 4	
13	AC	F	8+2	20/30	20/40	5			5	REC LROU	1+4
										Hydrocephalus/shunt	
14	SD	M	3+6	20/30	20/40	18	5		23	Unknown, XT right gaze	
15	SD	F	7+10			6	4		10	REC LROU 7	7+8
										XT right gaze	
16	DF	M	14+5	20/50	20/20	14			14	REC RMR 4.5, resect RLR 6	3+0

TABLE IX (CONTINUED): ACQUIRED HORIZONTAL INCOMITANCE

NO.	INIT	SEX	AGE	OD	OS	TURN	TILT	CHIN	TOTAL(°)*	ETIOLOGY	AGE
										REC MROU 4, LIO 14	8+0
										XT left eye	
17	EG	F	44+0			14	5		19	OS surgery	5+8
										OD surgery	28+0
				20/25	20/50					REC LLR 5, advance LMR 5	35+0
18	WH	M	3+3	20/40	20/40	6	15		21	REC MROU 7	0+5
										XT, HT left gaze and left tilt	
19	EH	F	4+5	20/20	20/70		13		13	REC LSR 8	1+2
										LHT Right Gaze	
20	PJ	M	10+6	20/20	20/30	6			6	REC LLR 6.5, RIO	
										extirpation	4+0
										RE-REC LLR 3, REC RLR 4,	
										REC LSR 5	10+4
21	PJ	M	9+6	20/50	20/50	7	10		17	LHT left gaze, Down	
										syndrome	
22	MK	M	11+5			12	15	6	33	REC MROU 5.5	0+9
										Resect LROU 7	1+10
										REC IOOU 14	3+9
										XT right gaze	
23	SK	F	5+10	20/50	20/50	20	10		30	REC MROU 6	1+2
										REC IOOU	4+10
										Cerebral palsy, secondary XT	
24	AK	F	9+3	20/20	20/20	7			7	ET surgery	0+6
										ET surgery	2+2
										HT left gaze	
25	LL	F	68+2	20/30	20/60	11			11	REC MROU	24
										Resect RMR, REC RLR, LLR	42
										RE-REC LLR, resect LMR	56
										Limit adduction OD	
26	TL	M	6+11	20/20	20/20	13	5		18	REC LROU 6	4+4

TABLE IX (CONTINUED): ACQUIRED HORIZONTAL INCOMITANCE

NO.	INIT	SEX	AGE	OD	OS	TURN	TILT	CHIN	TOTAL(°)*	ETIOLOGY	AGE
										XT left gaze	
27	CL	M	16+8	20/30	20/30	6	8		14	REC MROU 4+3	4+3
										Cerebral palsy, hydrocephalus	
										XT right gaze	
28	JL	M	15+0	20/30	20/20		12		12	REC LROU 7, S.O. tenotomy	7+3
										HT right gaze	
29	YM	F	16+11	20/30	20/30	10			10	REC LROU 3, S.O. tenotomy	5+10
										HT right gaze	
30	DM	M	16+9	20/160	20/40		7		7	REC MROU	4+7
										ET right gaze	
31	JM	M	8+8	20/25	20/60	10			10	REC MROU	0+5
										RHT, ET left gaze	
32	AP	F	8+9	20/20	20/30	8			8	XT, HT left gaze	
33	DP	F	69+3	20/60	20/40	10			10	REC MROU 4	67+9
										ET left gaze	
34	KQ	F	11+10	20/100	20/50	20			20	REC MROU 6	0+6
										Resect LROU	0+8
										REC RIO	3+1
										RE-resect LROU 4, REC LSR 5	6+10
35	KS	F	12+3	20/20	20/20	10			10	XT, HT left gaze, cerebral palsy	
36	HS	F	82+7	20/80	20/160	40		5	45	ET surgery	3+0
										ET surgery, slipped LMR	12+0
										REC LLR 10, resect LMR 16	82+6
37	AP	F	9+9	20/25	20/30	6			6	REC LLR, resect LMR	5+3
										XT right gaze	
38	NT	M	18+6	20/20	20/20	6			6	REC MROU 3.5	16+2
										ET right gaze	
39	VT	F	68+2	20/25	20/40	10			10	REC LSR 4	52+6
										RE-REC LSR4, REC RIR	59+7

TABLE IX (CONTINUED): ACQUIRED HORIZONTAL INCOMITANCE

NO.	INIT	SEX	AGE	OD	OS	TURN	TILT	CHIN	TOTAL(°)*	ETIOLOGY	AGE
										HT left gaze	
40	AV	F	21+10	20/60	20/40	6	12		18	REC LROU 4, S.O. tenotomy	8+2
										Cerebral palsy	
41	VD	F	13+5	20/25	20/25	10			10	REC MROU 6.5	4+0
										Cerebral palsy, hemianopsia	
42	CW	M	4+2	20/30	20/40	15			15	REC MROU 5, REC RIOOU 14	3+1
										HT left gaze	
43	RW	F	13+8	20/25	20/25	5			5	ET surgery	1+0
										REC LROU 9, IOOU	
										Extirpation	
44	MD	F	29+10	20/20	20/30	12			12	Lateral incomitance	
										Surgery 1965, '67, '86 Records NA	
45	RF	M	58+11	20/50	20/40	8			8	Esotropia	56+4
										REC RMR, resect	
46	ON	M	5+11	20/30	20/35	10			10	ET 95 preop	4+9
										REC MROU 7.5, Resect LLR 7	
47	DT	F	62+11	20/20	20/30	10			10	OS detachment	59+7
										Redetachment	59+7
										OS cataract extraction	60+11
										OD hypertropia, REC LSR 4	69+3
48	CS	F	36+4	20/50	20/20	4			4	ET 80 preop	36+1
										REC RMR 8, REC LMR 7.5,	
										Resect LLR 9	
										ET 10 right gaze	
										XT 8 left gaze	

*Average degrees 13.3.

TABLE X: ACQUIRED HORIZONTAL INCOMITANCE/A OR V PATTERN

NO.	INIT	SEX	A OR V	AGE	TURN	TILT	CHIN	TOTAL (°)*	STEREO	OVERACTION	OTHER DIAGNOSIS
1	CA	F	VXT	3+11	13	18		31	0	IOOU	
2	PA	M	VET	16+0	12	5		17	0	IOOU	
3	SA	M	VXT	6+10	5	10		15	20	IOOU	
4	JA	M	VET	3+8	12		4	16	0	IOOU	
5	LA	F	VET	1+10			5	5	?		
6	LB	M	VET	13+1	8		5	13	0	IOOU	
7	BA	F	AET	7+6			5	5	0	SOOU	
8	SB	M	VXT	13+3		5		5	0	RIO	
9	MB	F	VET	4+3			8	8	3000		Down syndrome
10	MB	M	VXT	9+11	8			8	0	IOOU	
11	MB	F	VET	5+2			7	7	0	IOOU	
12	AB	F	VET	3+11	10			10	3000	IOOU	
13	PB	F	AET	15+4			5	5	3000	OAIOOU	
14	TB	M	VET	7+10			5	5	0	RIO	
15	AB	F	VXT	3+5	8	16	8	32	100	IOOU	Brown OS
16	JC	M	VET	10+0	30	10	5	45	20		
17	CC	M	VET	13+4			10	10	0	IOOU	
18	MC	M	VET	7+6			20	20	0	IOOU	
19	SC	F	AET	18+4			10	10	400		
20	JC	M	AET	11+8	10			10	70		Cerebral palsy
21	AC	F	VET	5+5	12			12	0	IOOU	
22	TD	M	AXT	2+11	5	5	10	20	3000	SOOU	Tenotomy(2+11)
23	JD	M	VET	4+3	13			13	0	IOOU	
24	DD	F	AXT	10+7			12	12	0		Cerebral palsy
25	JD	M	AET	7+8	10			10	0		Optic atrophy
26	RD	M	VXT	7+6	15	5		20	0	IOOU	
27	JD	F	VXT	65+3			8	8	70	RSO	
28	PD	M	VXT	11+3		15		15	200	LIO	
29	PD	M	VXT	11+3		8		8	0	RIO	
30	LD	F	VXT	7+10		8		8	0	IOOU	

TABLE X (CONTINUED): ACQUIRED HORIZONTAL INCOMITANCE/A OR V PATTERN

NO.	INIT	SEX	A OR V	AGE	TURN	TILT	CHIN	TOTAL (°)°	STEREO	OVERACTION	OTHER DIAGNOSIS
31	SE	F	VET	7+3			15	15	0	IOOU	
32	MF	F	AXT	7+11		8	4	12	0	SOOU	
33	TF	M	VET	8+4			10	10	0		Down syndrome
34	SF	F	AET	6+0	5	10	12	27	100	SOOU	
35	DF	M	AET	2+6			5	5	?		
36	AF	F	AET	1+10			15	15	?		Down syndrome
37	JG	M	AET	13+9		10		10	0	LSO	
38	EG	F	VET	1+4			10	10	?		
39	LG	M	VXT	57+0			20	20	140		
40	JH	F	VXT	3+4			8	8	0	IOOU	
41	GH	M	VET	10+11	7	10	10	27	0	IOOU	
42	NH	M	VET	13+6			5	5	50	RIO	
43	KH	M	AXT	5+0	15			15	0	SOOU	
44	SH	F	VET	3+0		5		5	0	IOOU	
45	DH	F	AET	7+11			8	8	0	SOOU	
46	DK	M	VET	15+8			8	8	25		
47	RK	F	VXT	7+0			8	8	0	IOOU	
48	DK	M	VET	3+0			10	10	0	IOOU	
49	LL	F	VXT	4+10	20			20	0	IOOU	
50	BL	M	VET	14+2	5	12		17	0		
51	JL	F	AET	7+10			10	10	0	SOOU	
52	LF	M	VXT	65+8		3		3	0	IOOU	
53	AL	M	AXT	3+0	15			15	400	SOOU	
54	SL	F	VXT	4+6		6		6	100	RIO	
55	TL	M	AXT	5+1	18			18	70	SOOU	
56	GM	M	VET	4+8			8	8	0	IOOU	
57	SM	M	VET	3+0			8	8	0	IOOU	
58	AM	F	VXT	11+4		6		6	70	IOOU	
59	MM	M	VXT	7+8		6		6	0	IOOU	
60	NM	F	VET	5+4	8	10		18	0	LIO	

TABLE X (CONTINUED): ACQUIRED HORIZONTAL INCOMITANCE/A OR V PATTERN

NO.	INIT	SEX	A OR V	AGE	TURN	TILT	CHIN	TOTAL (°)°	STEREO	OVERACTION	OTHER DIAGNOSIS
61	SM	M	VET	7+8	8		6	14	0	IOOU	
62	EM	M	AET	1+0			10	10	?		
63	SM	F	AET	1+1			5	5	?		Cerebral palsy
64	BM	M	VXT	19+1		9	8	17	0	SOOU	Cerebral palsy
65	SM	F	AXT	7+0			5	5	3000	SOOU	Hydrocephalus
66	TN	M	AXT	3+4			20	20	400	SOOU	
67	BO	M	VXT	7+7			5	5	0	IOOU	
68	CO	M	VET	4+11	10	7	12	29	0	IOOU	
69	AK	F	VET	17+4		7		7	25	OAIOOU	
70	KP	M	AET	6+6			15	15	0	UAIOOU	
71	AP	M	AET	4+1	3			3	0	UASOOU	
72	RP	F	AET	50+7	4	6		10	70	LSO	
73	MP	M	VXT	8+3			5	5	0	IOOU	Down syndrome
74	EP	F	AET	11+3			4	4	200	OASOOU	Myelomeningocele/ shunt
75	JP	M	AET	11+4	8			8	200	RIO	
76	BP	M	VET	16+5			4	4	40	RIO	
77	AR	M	VXT	6+0		20		20	0	LIO	
78	ER	M	AET	1+2			5	5	?		Down syndrome
79	RR	M	VXT	3+8		8		8	400		
80	SR	M	AET	1+11	5			5	3000	UAIOOU	
81	SR	M	VET	2+10	6	8		14	0	OAIOOU	
82	NR	M	AET	10+6	20	6	3	29	0		Down syndrome
83	MR	M	AXT	10+8		5		5	70	OASOOU	
84	DS	F	VXT	54+4	3	6		9	3000	RIO	
85	AS	F	VET	1+7		10	20	30	?		
86	AS	F	VET	2+0			5	5	?	IOOU	
87	KS	F	VET	4+7	20		15	35	3000	RIO	Down syndrome
88	JS	M	AET	18+1	10			10	0	SOOU	

TABLE X (CONTINUED): ACQUIRED HORIZONTAL INCOMITANCE/A OR V PATTERN

NO.	INIT	SEX	A OR V	AGE	TURN	TILT	CHIN	TOTAL (°)°	STEREO	OVERACTION	OTHER DIAGNOSIS
89	NS	M	AXT	6+8	8			8	3000	SOOU	
90	JS	M	AET	6+8			8	8	?		Down syndrome
91	JS	M	AXT	5+10	20			20	0	IOOU	
92	TS	M	AXT	7+9		6		6	50	SOOU	
93	JS	F	VET	2+5			8	8	?		
94	MS	M	VET	3+9	10			10	0	IOOU	
95	AS	F	VXT	7+10			8	8	20	IOOU	
96	AS	F	VET	2+3			25	25	?	IOOU	
97	KS	F	VXT	7+7			30	30	0	IOOU	ROP
98	GS	M	VXT	1+2			5	5	?	IOOU	
99	CS	M	VET	11+3	6	8		14	0	UASOOU	IOOU (7+0)
100	WT	F	VXT	43+4			7	7	30	OAIOOU	
101	ST	F	AXT	5+5	6	10		16	0	SOOU	
102	AT	F	VXT	20+11			8	8	0	OASOOU	Neurofibromatosis
103	MT	F	AXT	10+8	4			4	0		
104	EV	M	AET	87+6	12	10	5	27	3000		
105	FV	M	AET	7+8			5	5	0	OASOOU	Cerebral palsy
106	AV	M	VET	5+6	8			8	0	OAIOOU	
107	TV	M	VET	18+6	10			10	0	LIOOA	
108	HW	F	AET	9+5			12	12	0		
109	JW	F	AET	1+10			5	5	?		Myelomeningocele
110	AW	M	AET	8+10		12		12	200	RIO	
111	EW	F	AET	6+5			6	6	0	RSO	William syndrome
112	KW	F	VET	23+10	4	6		10	0	OAIOOU	
113	MW	F	AXT	5+2			8	8	0	RSOOA	
114	MY	F	VXT	12+10			5	5	25	IOOU	
115	PY	M	AXT	8+3		3		3	0	IOOU	
116	AZ	M	VXT	4+11			10	10	?	OASOOU	

°Average degrees 12.1.

TABLE XI: ACQUIRED HORIZONTAL INCOMITANCE/SIXTH NERVE PALSY

NO.	INIT	SEX	AGE	TURN	TILT	CHIN	TOTAL(°)*	SURGERY	AGE
1	MA	M	8+1	5	5		10		
2	DB	M	38+4	15	12		27		
3	KB	F	12+11	8			8	(Bilateral—no surgery)	
4	JB	M	12+8	4			4		
5	MC	M	1+2	25			25		
6	JH	M	81+0	20		15	35		
7	KJ	M	1+11	30			30		
8	EK	F	78+6	10		8	18	REC RMR 4.5	78+0
9	DP	F	40+0	8			8	REC RMR 7, resect RLR 9	28+0
								RE-REC RMR 3, transpose Temporal 1/2 SR+1R to LR	38+0
10	TP	F	31+0	10			10		
11	AR	F	14+2	6			6		
12	IS	F	84+0	15			15		
13	CT	F	72+9		8		8	REC LRM 4, resect LLR 6	72+0
								REC LMR 4, resect LLR 5	37+4
14	MV	F	9+8	18			18		
15	NW	F	8+1	5	5		10	REC MROU 5.5	4+8

*Average degrees 15.5.

occurred in 40 of the 48 patients, a head tilt in 19 patients, and a chin posture in only 4 patients. Only 3 patients 1, 5, and 14 did not have eye surgery. Of the 45 patients who had surgery, all had eye muscle surgery except for patient 2, who had retina detachment surgery only. The majority of the patients had symmetrical surgery, but all presented with incomitance and torticollis. Only 12 patients had differences in vision of 2 or more Snellen lines, and the torticollis can not be explained on the basis of vision alone.

A number of patients presented with lateral incomitance along with horizontal incomitance, contributing to torticollis in an attempt to maintain fusion. The Worth 4-dot responses ranged from fusion, to alternation, to suppression, and the random-dot stereoscopic testing ranged from 20 seconds to negative. On the basis of these sensory findings, it would have been possible to classify some of these patients in the group summarized in Table XVI with no apparent reason for torticollis. However, the asymmetry of the motility findings directed their placement to this group. The placement of a patient in any of these diagnostic groups was arbitrary, and the overlap of prior history, surgery, and preoperative or postoperative measurements created a redistribution of a number of patients during and at the completion of this 1-year prospective study.

ACQUIRED HORIZONTAL INCOMITANCE/A OR V PATTERN

The A and V patterns are characterized by a horizontal eye alignment change in midline upgaze and downgaze as the eyes shift from the primary position.[100] Vertical incomitance was first described by Duane,[101] but its significance and importance were not appreciated until the work by Urrets-Zavalia[102,103] and Urist.[104,105] Albert suggested the "A" syndrome and Jampolsky advocated the tent or teepee syndrome (A), with the A and V terminology gaining universal acceptance.[106]

In the primary position, orthophoria, esotropia, or exotropia in association with the A or V pattern may be present. By definition, an A or V pattern involves a change of 10 prism diopters in the horizontal alignment between upgaze and downgaze.[107] Urist suggested that the etiology of the A and V patterns was related to the horizontal rectus muscles. However, overactions of the inferior and superior oblique muscles are frequently associated with the A and V patterns. Surgery on the oblique muscles corrects these patterns, suggesting that the etiology of the A and V patterns is related to oblique muscle overactions.

Tables III, X, and XVIII summarize the findings of patients with A and V pattern with horizontal incomitance. In the current series, 116 patients presented with A or V patterns with torticollis, categorized by type in Table III. The diagnosis of A and V pattern was found in the office review

610 times, for an incidence of torticollis of 19%, almost double the percentage described by Kushner.[20] Sixty-seven male and 49 female patients ranging in age from 1 year to 87 years 6 months (average age, 10 years 9 months) presented with torticollis averaging 12.1°. Forty-four patients presented with a face turn averaging 10.2°, and 40 patients presented with a head tilt averaging 8.5°. Sixty-seven patients demonstrated a chin depression or elevation posture, yet the degree of chin posture averaged only 9.0°. The average degree of chin posture was lower than anticipated, since a chin posture with elevation or depression would be expected more frequently with oblique muscle overaction.

Overaction of 1 inferior oblique was noted in 17 patients but occurred bilaterally in 45 patients. Underaction was seen bilaterally in 2 patients. Overaction of the superior oblique muscles was noted to be unilateral in 3 patients but bilateral in 21. Three patients had underactive superior oblique muscles in both eyes. Stereoscopic vision could not be recorded in 15 patients, and therefore they were deleted from the group total of 116. There was no measurable stereoscopic vision in 63 patients. The incidence of absent stereoscopic vision was 63 of 101 patients, or 62%. From the group of 48 with A and V patterns, Kushner described 35 patients without torticollis, who had large deviations prohibiting fusion, including 9 patients with less than 10 D of deviation. Although Kushner's group lacked torticollis, it also lacked fusion, with an incidence of 73%, higher than in this study .

Eight patients with Down syndrome were included in the current group, only 1 of whom had oblique muscle overaction. This group of Down patients merits further investigation because of their apparent concentration in the group with A and V pattern, and because of the lack of oblique overactions reported in this study.

SIXTH NERVE PALSY

Sixth nerve palsy may be congenital,[32-34,39,42,43,108-110] though extremely rare, or may be acquired.[32-34,39,42,43,111-114] The long intracranial course predisposes the sixth nerve to increased intracranial pressure, trauma, edema of the meninges, inflammation, and brain stem displacement.[115] Sixth nerve paralysis has many etiologies, including trauma, neoplasm, inflammation, undetermined causes, and miscellaneous causes. An extensive summary of the literature concerning the etiology of sixth nerve palsy and many associated conditions has been described.[115]

Sixth nerve palsy clinically presents with esotropia in the primary position, with a compensatory head posture to maintain binocular vision. Recovery often occurs within 3 months of onset, and if contracture of the ipsilateral medial rectus has not occurred, then surgical intervention may

not be required. In a young child who does not assume a compensatory head posture, patching may be necessary to preserve vision and prevent amblyopia. Surgery may be necessary if normal function does not return after 6 months. A recession of the medial rectus or a resection of the palsied lateral rectus muscle may prove to be sufficient. If there is a negative forced duction test, a transposition procedure with a Hummelscheim or a Jensen operation may be required.

The acquired horizontal incomitance group, with sixth nerve palsy, is summarized in Tables XI and XVIII. Fifteen patients compose this group, including 6 males and 9 females ranging in age from 1 year 2 months to 81 years (average age, 32 years 11 months). The average torticollis was 15.5°. All except 1 patient demonstrated a face turn, and only 4 presented with head tilt and 2 with chin postures. Four patients required surgery, including patient 9, who had a severe sixth nerve palsy and contractures, eventually requiring three surgical procedures. Kraft and associates[49] found compensatory head posture in 26 of 93 patients (29%) in their study of 381 patients with compensatory head posture.

In the current series, patient 3 was comatose for a brief period after a motor vehicle accident at age 10. She presented with 75 D of esotropia, which spontaneously improved in slow increments, to E(T)' of 0-10 D. Stereo returned to 20 seconds of arc with the random-dot test . She was scheduled for surgery several times but was canceled each time owing to gradual improvement of the esotropia over 18 months. Her only significant eye motility finding at this time is E(T) of 6 to 8 D, only in right gaze, and a residual face turn of 8°.

TORSIONAL INCOMITANCE

A compensatory head posture to the right or left shoulder may be present with cyclovertical strabismus or in the presence of nystagmus. Conrad and deDecker[116] have combined a recess-resect procedure at the anterior poles of the oblique muscles, with transposition of their insertions toward the posterior-anterior pole. The procedure is directed at rotating the globes toward the side of the tilt position by shortening and by lengthening the anterior parts of the oblique muscles. Cyclotorsional diplopia has been described following retina detachment surgery, although the overall incidence is low.[117] A compensatory head tilt may be evident in the presence of cyclovertical strabismus in order to permit fusion.

Von Noorden[118] presented 5 patients, one of whom was lost to follow-up. The remaining 4 patients had horizontal transposition of the superior and inferior rectus muscles, which eliminated the head posture by rotating the eyes around the sagittal axis. The clinical assessment of ocular torsion, as described by Guyton,[119] has been a valuable objective test, not only

with torsional incomitance, but also with cyclovertical palsies. As Guyton has described, the clinical torsion noted with overacting inferior obliques up to 15° or 20° does not correlate with the double Maddox rod test, which invariably fails to demonstrate subjective torsion.

In a study of cyclovertical displacement of the blind spot, Locke[120] abandoned the double Maddox rod test because of a high incidence of negative results, while torsional deviation was revealed by blind spot plotting monocularly and binocularly. Von Noorden[121] has described limitations of the double Maddox rod test in that only the central portion of the visual field is being tested, where cyclodisparity is less significant than in the retinal periphery. Fusable visual material in the periphery of the visual field is not available when the red and white streaks are seen. Cyclofusion may compensate for a symptomatic cyclodeviation, although there may be several degrees of cyclotropia measurable on double Maddox rod testing. Although cyclovertical muscle anomalies occur with relative frequency, patients rarely complain of tilting of images, cyclodeviations seem to rarely play a role in head tilting, and most cyclodeviations are well tolerated.[119,121] Despite objective cyclotorsion of the globe in cyclotropia, subjective testing may yield negative results because of sensorial adaptations.[122,123]

In the current study, there were 4 patients with torsional incomitance, as summarized in Table XII. Patient 1 had stereo of 25 seconds with no obvious reason for torticollis, and intorsion of the fundi, which was not severe enough to require surgery. Patient 2 had a Harada-Ito procedure on both eyes, with a small residual degree of torsion, not significant enough to require further surgery. Patient 3 was lost to follow-up. Patient 4 has one nonseeing eye and ocular torsion with a marked head tilt; assessment for nystagmus has been difficult, even at high-power illumination in the slit-lamp microscope. He may be a candidate for the operative procedure described by von Noorden; however, the family is reluctant to allow surgical intervention on his only seeing eye.

TABLE XII: TORSIONAL INCOMITANCE

NO.	INIT	SEX	AGE	TURN	TILT	CHIN	TOTAL(°)*	SURGERY	AGE
1	PS	M	5+9	15			15		
2	SG	F	30+1		10		10	Harada-Ito OU 27+1	
3	HV	M	61+11		15	10	25		
4	IG	M	8+4		20		20	(Blind OD, OS excylotorsion)	

*Average degrees 17.5.

NYSTAGMUS

Kushner[20] described 38 patients in his group of 118 (20%) with nystagmus as an etiology for abnormal head posture. The results of the current study are summarized in Tables I, III, XIII, and XVIII. In the group of 630 patients with torticollis, 120 presented with nystagmus, for an incidence of nystagmus of 19%, compared to Kushner's study. In the current study, 290 patients presented with a diagnosis of nystagmus, for an incidence of torticollis of 41% (120/290). Seventy-five male and 45 female patients presented with torticollis and nystagmus; age range was 9 months to 65 years 6 months (average age, 9 years 2 months). The average torticollis was 21.1°. Stereoscopic vision was not measurable because of age or comprehension in 20 patients, was absent in 53 patients, and varied from 20 to 3,000 seconds of arc in 47 patients. Seventeen patients presented with albinism or macula hypoplasia, 13 with neurologic-related disorders and 7 with Down syndrome. Patient 16 was seen for the first time at 9 months of age, with recent onset of nystagmus and no other apparent problems. Results of her eye examination were normal except for the nystagmus. Neurologic testing and studies revealed a grade 3 astrocytoma requiring immediate therapy.

Eighteen patients have had a conventional Kestenbaum operation, and 4 have had a variation because of marked head tilt, face turn, or chin posture. The Kestenbaum modification includes recession of 2 vertical muscles to compensate for the chin posture, recessing the inferior rectus muscles or the superior rectus muscles from 7 to 9 mm, and using the Kestenbaum guidelines for recessing two horizontal muscles for a face turn (Fig 3).

In 1950, Metzger[124] recommended the use of eyeglasses with prisms,

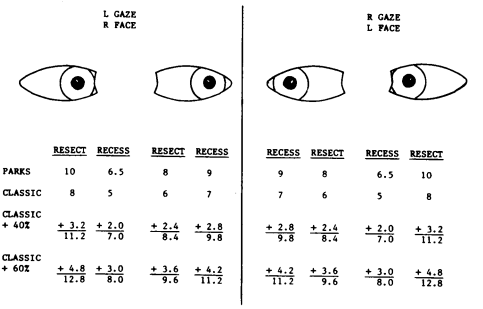

FIGURE 3

Kestenbaum surgical diagram placed in each patient's chart prior to surgery for notation of appropriate muscles for surgery.

TABLE XIII: NYSTAGMUS

NO.	INIT	SEX	AGE	NYST°	STEREO	TURN	TILT	CHIN	TOTAL°†	OD	OS	STRABISMUS	ADDITIONAL DIAGNOSIS	SURGERY (AGE)
1	AA	F	7+11	L	0	5			5	20/30	20/60	ET=20	Aphakia	OS Ocutome (0+2)
2	DA	M	5+0	RP	0		45		45	20/160	20/160	ET=8	Albinism	REC MROU 4.5 (2+6)
3	CA	M	8+3	HP	0	12			12	20/400	20/200	ET=20	Albinism	REC MROU 5 (5+3)
4	RB	M	9+11	HP	0	10	6	3	19	20/400	20/25	XT=18	Cataract	Epikeratophakia (0+1)
5	NB	M	12+10	HP	70	15			15	20/70	20/70	XT=16	Astigmatism	
6	SB	F	10+9	HP	20	8			8	20/30	20/35	EX=0		Kestenbaum 7,8.5,10,11 (4+10)
7	KB	M	6+6	HP	0	10	15		25	20/50	20/80	ET=6		REC RMR 6, LMR 7.5 (0+6) Kestenbaum 4,7,8,10 (4+8)
8	KB	F	18+9	HP	0	15	7	20	42	20/50	20/60	ET=10	Albinism	REC MROU 5 (4+0) REC SROU 10 (6+3) RES IROU 8 (9+2)
9	JB	M	42+0	R	200	5			5	20/30	20/30	35-0-35 ET	Moebius syndrome	
10	KB	F	6+1	HR	400	10		15	25	20/60	20/60	EX=0	Down syndrome	REC RLR 9, LMR 7, IROU 7 (5+11)
11	SB	M	3+4	HP	?	20			20	CS(M)	CS(M)	XT=50	Meningitis Cerebral clot Optic atrophy	
12	DC	M	6+3	HP	0			5	5	20/125	20/160	ET=12		
13	SC	M	8+4	HP	0	8	5	5	18	20/250	20/160	A=XT	Cerebral palsy	Kestenbaum 5,9.5,11,9 (7+3)
14	JC	M	12+7	HJ	3000	15		8	23	20/70	20/60	ET=5		Kestenbaum 7,8.5,10,11 (7+4)
15	DC	M	8+11	HR	0	3		5	8	20/25	20/60	ET=12	Tourette syndrome	REC LROU 8 (1+2) REC LSR 6 (6+9)
16	CC	F	0+9	HR	?	35			35	?CSM	CSM	EX=0	Astrocytoma,	

TABLE XIII (CONTINUED): NYSTAGMUS

NO.	INIT	SEX	AGE	NYST[a]	STEREO	TURN	TILT	CHIN	TOTAL°†	OD	OS	STRABISMUS	ADDITIONAL DIAGNOSIS	SURGERY (AGE)
17	JC	M	3+7	PR	?	20			20	4/200	3/200	EX=0	grade 3 Optic nerve hypoplasia	
18	AC	F	22+5	HP	70			5	5	20/40	20/30	EX=0	-2+5.00X105, -2+5.00X75	Kestenbaum 7,8.5,10,11(19+4)
19	JC	F	11+2	HP	30	15		8	23	20/30	20/30	EX=0		
20	ED	F	10+0	HP	3000	25	12	4	41	20/50	20/30	LH=10		REC MROU 4.5, REC IOOU 14 (0+11) S.O. Tuck OU (3+7)
21	AD	F	13+3	HP	0	5		6	11	20/40	20/50	XT=10		Kestenbaum 6.5,7,8,10 (4+0) REC RMR 4.5 (5+6)
22	KD	M	15+4	HP	0	8			8	20/400	20/200	ET=25	Usher syndrome	
23	RD	F	3+9	HJ	0	15	28		43	20/30	20/25	ET=14		
24	JD	F	7+1	HR	0	10	12	8	30	20/50	20/40	AET	Albinism	
25	MD	M	4+0	HP	400	28			28	20/30	20/40	EX=0	Astigmatism +3.00	
26	MD	M	5+3	HP	100	35		15	50	20/25	20/25	EX=0	Albinism	Kestenbaum REC RLR (5+6) LMR 7, SROU 6
27	DD	M	13+10	HJ	70		12		12	20/30	20/30	X=4	Turner syndrome	
28	LD	M	10+10	HJ	100	10	5		15	20/30	20/30	ET=8		
29	AD	M	2+4	HR	3000	10	15		25	20/40	20/60	ET=25	Amblyopia	REC MROU 7 (0+6)
30	DD	F	6+0	RL	0	20			20	20/50	20/60	ET=12		REC MROU 6.5 (1+1)
31	JD	M	2+1	HP	0			5	5	20/200	20/400	ET=30	Acc. ET-Phospholine	
32	ED	F	14+0	HR	3000	5			5	20/100	20/100	EX=O	Albinism, JRA	REC LROU 6 (3+7)
33	ME	M	3+3	HR	?		10		10	CSM	CS(M)	ET=12		

TABLE XIII (CONTINUED): NYSTAGMUS

NO.	INIT	SEX	AGE	NYST°	STEREO	TURN	TILT	CHIN	TOTAL°†	OD	OS	STRABISMUS	ADDITIONAL DIAGNOSIS	SURGERY (AGE)
34	ME	M	1+6	HP	?			6	6	20/300	20/400	E(T)=8	"A" pattern	
35	RF	M	6+6	HR	0	25	8		33	20/30	20/30	XT=10		REC MROU 7.5 (7+6)
36	GF	M	6+1	HJ	0	20	15	25	60	20/60	NLP	XT=7	OS Microophthalmia	PHPV (0+3)
37	JF	M	1+6	HJ	?	10			10	NCSM	CSM	XT=45	Retinopathy of prematurity OU	
38	DF	F	65+6	DB										
39	AG	M	6+4	HJ	?	25			25	CSM	CSM	ET'=15	Down syndrome	
40	EG	M	2+6	HJ	?	10	15		25	20/50	20/40	ET=35		
41	EG	F	7+11	DB	0			15	15	20/30	20/30	ET=14	Neurological negative	
42	BG	M	5+5	HP	100	25	6		31	20/40	20/50	EX=0		
43	MG	M	10+5	HP	0	12	8		20	20/40	20/40	ET=16 HT=12		REC MROU 5 (4+10)
44	CG	M	6+2	HJ	0	20		7	27	20/40	4/200	LET=20	OS microop, choroid colob	
45	EG	M	11+6	HP	3000	6			6	20/60	20/70	E=5		
46	MG	M	14+9	HR	200	10			10	20/80	20/60	EX=O		
47	MG	F	1+2	HP	?	25			25	20/300	20/300	EX=O	Albinism	
48	SG	F	4+1	HR	0			11	11	CSM	CSM	E(T)=16		REC MROU 6, REC IOOU 14 (3+11)
49	WG	M	9+7	HJ	0	5	5	5	15	20/400	20/400		Down syndrome	
50	AG	M	8+9	HP	0	15			15	20/50	20/100	ET'=5		REC MROU 6 (4+7)
51	TG	M	10+1	HR	0	35	15		50	20/400	20/30	RXT=25	Retinopathy of prematurity	REC RSR 7 (6+1)

TABLE XIII (CONTINUED): NYSTAGMUS

NO.	INIT	SEX	AGE	NYST°	STEREO	TURN	TILT	CHIN	TOTAL°†	OD	OS	STRABISMUS	ADDITIONAL DIAGNOSIS	SURGERY (AGE)
													Monocular nystagmus	
52	EH	F	12+0	R	3000	13			13	20/20	20/30	ET=15		REC LROU 5 (5+0) REC MROU 3.5, REC RSR 6 (6+6)
53	EH	M	4+2	HJ	0	15		8	23	20/60	20/30	XT=8	Aphakia OU (0+2)	REC MROU 5.5 (0+5)
54	JH	M	9+9	R	0	15	5	15	35	20/70	20/70	ET=20	Albinism	REC RMR 7, REC LLR 5 SROU (9+10)
55	NH	M	4+7	HP	70	15			15	20/30	20/40	E'=2		
56	MH	M	14+4	HJ	25	12	9		21	20/25	20/25	EX=0		Kestenbaum 7,8.5,10,11 (5+8)
57	KH	M	23+2	HJ	0	15		5	20	20/40	20/50	LH=6		
58	KH	M	18+8	HP	0	25	7	4	36	20/60	20/60	XT=4	Seizures	REC LROU 7 (5+7)
59	MI	M	10+0	HP	200	15		10	25	20/100	20/100	EX=0		
60	RI	M	5+8	HR	400	20	8	12	40	20/60	20/125	X(T)'=10		
61	FJ	M	9+7	HJ	3000	5		7	12	20/70	20/50	ET=6		
62	MK	F	6+11	HP	0	20	10	6	36	20/50	20/160	ET=45	OS Amblyopia	
63	BK	M	9+10	HR	3000	6		5	11	20/30	20/70	HT=4	OAIOOU	
64	BK	M	7+0	HP	0			15	15	20/200	20/200	ET=20	Albinism	
65	AK	F	1+4	HP	?	30	8		38	C(SM)	C(SM)	XT=25	Optic nerve hypoplasia	
66	AL	F	15+6	HP	70	15			15	20/40	20/30	EX=0		Kestenbaum 8,9.5,11,13 (9+4)
67	GL	M	12+11	HP	70	45			45	20/70	20/50	EX=0	Left or right 45°	
68	VL	F	3+9	DB	0			12	12	CSM	CSM	EX=0	Neurological exam negative	

TABLE XIII (CONTINUED): NYSTAGMUS

NO.	INIT	SEX	AGE	NYST°	STEREO	TURN	TILT	CHIN	TOTAL°†	OD	OS	STRABISMUS	ADDITIONAL DIAGNOSIS	SURGERY (AGE)
69	AL	M	1+10	HJ	?	15			15	CNSM	CNSM	ET=30	Aniridia	REC LMR, Resect LLR
70	KL	F	7+1	HR	0	10		3	13	20/125	20/80	ET=6	Macula hypoplasia	REC LROU 8 (1+7)
71	SL	M	3+5	HJ	3000	40			40	20/60	20/50	ET=8	Developmental delay	
72	EL	F	5+11	HR	0			10	10	20/40	20/40	ET=18	Albinism	
73	BL	M	4+2	HJ	?	20	30	30	80	20/1200	20/1600	EX=0	Down syndrome	
74	WL	F	1+5	HP	?	10		10	20	20/200	20/200	XT=5	Albinism	
75	CL	F	16+2	HP	0	15			15	20/200	20/50	ET=6	Down syndrome, Cataract OU	REC MROU 6 (12+5)
76	DM	M	3+6	HR	0	25			25	20/25	20/25	ET=35		
77	SM	M	2+9	HP	0		6	5	11	CSM	C(SM)	ET=30		
78	CM	F	8+3	HR	40	15		10	25	20/70	20/40	EX=O		REC RLR 9, REC LMR 6.5 (8+6) REC IROU 5
79	NM	M	10+1	HP	3000			5	5	20/40	20/30	ET=35	Down syndrome, accom. ET	REC MROU 7 (5+0)
80	RM	M	16+3	HP	40	10			10	20/50	20/40	EX=0		Kestenbaum (4+0)
81	MM	F	16+10	HP	0	10			10	20/100	20/80	EX=0	Macula hypoplasia	Kestenbaum 6.5,8,9,10 (8+11)
82	CM	F	14+11	HP	100	10		4	14	20/50	20/70	X(T)=15	Macula hypoplasia	
83	AM	M	11+7	HP	70	10	4		14	20/60	20/50	E'=4		
84	SM	M	2+7	HP	?	6	12		18	20/200	20/150	EX=0	Down	Rec MROU 5.5, REC

TABLE XIII (CONTINUED): NYSTAGMUS

NO.	INIT	SEX	AGE	NYST°	STEREO	TURN	TILT	CHIN	TOTAL°†	OD	OS	STRABISMUS	ADDITIONAL DIAGNOSIS	SURGERY (AGE)
											syndrome	IOOU 12 (1+1)		
85	JM	M	2+8	HP	?	10		5	15	CNSM	CNSM	ET'=12		
86	TM	M	6+11	HP	400	35	15		50	20/50	20/100	EX=0	Macula hypoplasia	Kestenbaum (7+0)
87	AM	F	20+0	HP	0	10	6		16	20/25	4/200	XT=8	Left optic nerve anomaly	Kestenbaum 8,8.5,10,13 (3+9)
88	TM	M	2+11	HP	3000	10	20		30	20/400	5/200	EX=0	Albinism	
89	EN	F	2+8	HJ	?	10			10	HM	HM	XT=40	Microcephaly, cerebral palsy	
90	SO	M	3+2	HP	0			10	10	20/70	20/70	EX=0		
91	AO	F	37+7	HP	0	6		4	10	20/50	20/100	HT=14		Kestenbaum 6,9.6,11,10 (30+5)
92	TP	M	3+7	HP	0		15		15	20/25	10/200	ET=30	Amblyopia Congenital glaucoma	Goniotomy OU
93	JM	M	13+8	HP	400		15		15	20/20	20/25	HT=5		REC MROU 6.5 (0+8)
94	CP	F	12+5	HP	0		15		15	20/70	20/60	ET=4	Fetal alcohol syndrome High myopia	
95	LP	F	5+0	HJ	0	10			10	NCSM	NCSM	ET=15		REC MROU 7, REC LIO 14 (4+9)
96	LP	F	14+2	HP	0	5		4	9	20/160	20/250	EX=0	Aniridia	
97	JP	M	15+6	HP	25	5	15		20	20/25	20/25	EX=0		Kestenbaum (4+8)
98	JP	M	5+6	HP	0	10		5	15	20/70	20/40	ET=16	Accommodative ET +8.00 OU	REC MROU 6.5 (1+8)
99	AP	F	5+3	HP	0	10		20	30	20/40	20/60	ET=3		

TABLE XIII (CONTINUED): NYSTAGMUS

NO.	INIT	SEX	AGE	NYST°	STEREO	TURN	TILT	CHIN	TOTAL°†	OD	OS	STRABISMUS	ADDITIONAL DIAGNOSIS	SURGERY (AGE)
100	TR	F	5+6	DB	0	15		10	25	20/40	20/60	ET=8	Accommodative ET	
101	SR	F	1+7	HP	?	20		10	30	CSM	CSM	EX=0		
102	ER	F	6+9	HP	70	10		15	25	20/40	20/50	EX=O		Kestenbaum 8,9.5,11,13 (14+7)
103	BS	F	6+4	DB	0	6		15	21	20/30	20/40	ET=12	Neurological exam negative	
104	LS	M	9+8	HP	70			10	10	20/60	20/60	EX=0		REC RLR 9, REC LMR 6.5
105	ES	M	13+7	HP	50	5			5	20/60	20/100	EX=0	-2+6X90 OU	Kestenbaum (4+9)
106	BS	F	4+2	HP	0	40	15		55	CNSM	CNSM	ET=15	Head Banging	
107	AS	M	8+10	HP	3000		15		15	20/100	20/160	EX=O	Albinism, Hermansky-Pudlak's	
108	RS	F	2+1	HP	?	30			30	20/100	20/100	HT=10		
109	ES	M	6+7	HJ	0	20		30	50	20/200	20/200	XT=8	Optic nerve coloboma OU	
110	MS	M	16+11	HP	70	4	6	6	16	20/20	20/25	ET=2		Kestenbaum (4+6)
111	HS	M	13+10	HP	0	15			15	20/40	20/40	X(T')=18	Autism	
112	KS	F	5+0	HJ	0	20			20	20/60	20/50	ET'=4		
113	DT	M	6+5	HP	70	10	15	10	35	20/30	20/30	EX=0		
114	MV	M	11+6	HJ	70	10	10	5	25	20/30	20/30	EX=0		
115	EV	F	8+11	HP	20		10		10	20/25	20/30	EX=0		
116	MV	M	2+4	HJ	?	10			10	20/150	20/200	ET=40	Aphakia OD	
117	RW	M	6+1	HP	100		8		8	20/60	20/40	X(T)=6		
118	LW	F	5+5	HJ	70	10			10	20/60	20/60	EX=0	Macula hypoplasia	

TABLE XIII (CONTINUED): NYSTAGMUS

NO.	INIT	SEX	AGE	NYST°	STEREO	TURN	TILT	CHIN	TOTAL°†	OD	OS	STRABISMUS	ADDITIONAL DIAGNOSIS	SURGERY (AGE)
119	AZ	M	4+4	HJ	100	20	3		23	20/40	20/30	EX=0		
120	ZZ	M	2+3	HR	?			15	15	20/200	20/200	ET'=20	Down syndrome High myopia -12	REC MROU 6 (0+9)

° H, horizontal; J, jerk; L, latent; P, pendular; R, rotary.
† Average degrees 21.1.

with the apex directed toward the null point, in order to correct or control the torticollis, but subsequent patients adapted to the prisms and torticollis failed to be controlled. In 1953 and 1954, three independent reports advocated treatment for the torticollis. Kestenbaum[125] suggested shifting the eyes away from the null point by an equal amount of recession and resection, or controlled tenotomy on 1 eye, followed by a similar procedure on the fellow eye after a period of stabilization. Anderson[126] described recessing the horizontal rectus muscles to shift the eyes away from the null point, with the exclusion of resection procedures. Goto[127] advanced the horizontal rectus muscles to pull the eyes from the null position. Subsequently, many others have provided guidelines for surgical correction of torticollis caused by nystagmus. Cooper and Sandall[128] performed surgery on the fixating eye first, with a recess-resect procedure, followed by surgery on the nonfixating eye, with appropriate adjustment for the strabismic angle. This was felt to be especially sound advice in treating torticollis with strabismus. Pratt-Johnson[129] was the first to recommend equal amounts of surgery on all 4 rectus muscles but noted recurrence of the torticollis after a period.

Parks[130] was concerned that the pulling power varied between the medial and lateral rectus muscles and that identical amounts of recession and resection would turn the eye on the rotation center by different quantities. Parks modified the Kestenbaum procedure so that the 4 horizontal rectus muscles would receive the maximal amount of surgery without compromising the ductions of the eyes and would not induce strabismus when the pulling power was altered. The medial rectus was recessed 5 mm, and the lateral rectus of the same eye was resected 8 mm. On the fellow eye, the lateral rectus was recessed 7 mm and the medial rectus was resected 6 mm. This became known as the "classic maximum," as described by Calhoun and Harley,[131] who noted undercorrections within these guidelines for the Kestenbaum operation. Calhoun and Harley augmented these surgical guidelines by 40% and noted improved surgical responses. Subsequently, Nelson and associates[132] described a 60% augmentation and guidelines for torticollis surgery for nystagmus. For a face turn of 15°, surgery was not recommended. For a face turn of 30°, surgery was recommended with the classic maximum plus 40%, and for 45° of face turn, the classic maximum plus 60%.

Fig 3 describes the surgical guidelines for the Kestenbaum procedure. A copy of this diagram is placed in the chart of each patient requiring extraocular muscle surgery because of nystagmus and torticollis. The appropriate muscles are marked on the diagram to ensure accuracy in the surgical procedure.

Concern has been expressed that very large recessions and resections

may result in limitation of ductions. Any slight limitations in extreme gaze, however, must be compared with reduction in torticollis and marked improvement in appearance as well as frequent improvement in vision.[17,21]

When Calhoun and Harley's guidelines have been followed, the long-term results have proven to be promising. In a review of 79 patients[21] with an average follow-up of 5 years, the average face turn of nearly 40° preoperatively was reduced to less than 10° in the series of patients receiving horizontal surgery. Stereoscopic vision was not compromised in any patients who demonstrated stereoacuity preoperatively and was actually enhanced in several patients postoperatively. The visual acuity was the same or improved in all patients, and the vision was not compromised in any patient.

Most patients maintain a stable head posture after surgery. While some patients may reveal a tendency to gradually shift toward the preoperative state, the patients almost never return to the original head position.

CONGENITAL ESOTROPIA WITH OCULAR POSTURE

In a series of 58 patients with essential infantile esotropia, Lang[133] reported 38 patients (70%) with anomalous head posture. Dissociated vertical divergence was seen in 54 patients (93%), latent nystagmus in 29 patients (50%), and excyclorotation of the nonfixing eye in 35 patients (60%). Lang stressed that the head posture was not adopted to avoid diplopia. His patients demonstrated different combinations of these conditions, with some more pronounced than others. In his 1982 Costenbader Memorial Lecture, Lang[134] described his series of the congenital strabismus syndrome, totaling 82 cases, with 70% demonstrating abnormal head posture, 92% with dissociated vertical divergence, 57% with latent nystagmus, and 65% with excyclorotation of the nonfixating eye. In addition, 20% demonstrated A pattern, 17% had V pattern, and 15% had cerebral damage. He also found that when an abnormal head posture was present, the head was usually tilted toward the shoulder of the fixating eye, with the face turned to that side. Lang suggested that the congenital esotropia syndrome was due to an imbalance in the midbrain, between the geniculo-striate and the extra-geniculo-striate system, and he noted that a dominant feature of the syndrome was the latent nystagmus. Lang further speculated that the cause of the head tilt was related to a more fully developed vestibular system at birth, and that the vestibular system in the patient with congenital esotropia may have excessive dominance.[20,133]

In discussing the relationship between dissociated vertical divergence and head tilts, Bechtel and associates[135] reported an incidence of manifest head tilt of 35% in a series of patients with dissociated vertical divergence (DVD) associated with infantile esotropia. The DVD increased on forced

contralateral head tilting and decreased on ipsilateral tilting. The investigators suggest that some of the head tilts and anomalous head postures attributed to the congenital esotropia syndrome may actually be due to DVD and that DVD is a frequent cause of head tilts.

Early reports by Crone[136] on alternating hyperphoria, by Anderson[137] on alternating hyperphoria and latent nystagmus, and by Ciancia[138-140] on esotropia in infants with abduction limitation have contributed to this syndrome of congenital esotropia with ocular posture.

Tables III, XIV, and XVIII summarize this group of 69 patients with congenital esotropia and ocular torticollis. Table III notes that 812 patients were seen with infantile or congenital esotropia. The percentage of patients with torticollis and congenital esotropia is 69 of 812, or 8.5 %. This group consisted of 39 males and 30 females, ranging in age from 8 months to 32 years 1 month (average age, 7 years 4 months). The average torticollis was 14.1°. There were 119 operative procedures performed, for an average of 1.7 operations per patient. Dissociated vertical deviation was present in 61 of 69 patients (88%), and latent nystagmus was present in 18 of 67 (27%). This low incidence of nystagmus is more closely related to the study of infantile esotropia by von Noorden,[27] who found 15% with manifest nystagmus and 10% with manifest-latent nystagmus.

Comparison between these series is difficult because von Noorden described 408 patients with essential infantile esotropia, while the current series compares the findings of all patients with esotropia and torticollis. In his series of 408 patients with essential infantile esotropia, von Noorden found 26 of 408 (6%) with anomalous head posture. In comparing the 69 patients presenting with congenital esotropia or infantile esotropia of the 812 patients included in the current study, von Noorden's figure of 6% is comparable to the 8.5% with torticollis, noted in Table III.

The initial surgical procedure in this group was most commonly a recession of the medial rectus muscle of both eyes. With residual esotropia, the second procedure was usually a resection of the lateral rectus muscle of both eyes. There were 24 inferior oblique procedures and 11 procedures on vertical rectus muscles.

Of interest is the limited response to stereoscopic testing, with only 1 patient achieving 140 seconds and only 7 patients recognizing the Titmus stereo fly, at 3,000 seconds. Six patients were too young to comprehend the stereo testing. In agreement with Lang,[129,130] these limited binocular responses suggest that the anomalous head postures are not adopted and are not assumed for the purpose of achieving or maintaining binocular vision, or to avoid diplopia.

Because of the low incidence of positive stereoscopic responses postoperatively, a future study is planned to review the records of those

TABLE XIV: CONGENITAL ESOTROPIA WITH OCULAR POSTURE

NO.	INIT	SEX	AGE	STEREO	TURN	TILT	CHIN	TOTAL (°)°	SURGERY	AGE	DVD	LATENT NYSTAGMUS
1	AA	F	1+11	0		15		15	REC MROU 7	0+8	+	+
									REC LROU 3	2+5		
2	BA	M	14+2	0	5	7		12	REC MROU	1+3	+	-
									Resect LROU 7, REC IOOU	7+3		
3	KB	M	6+4	0	6	15		21	REC MROU 6	2+4	+	+
									REC LROU 7.5, REC IOOU 14	6+2		
4	GB	F	14+7	0	10	10		20	REC RMR 4.5, Resect RLR 6.5	3+5	+	+
									RE-REC RMR 3, REC LMR 6, IOOU X	4+5		
									Resect LROU 4, REC LSR 4	14+6		
5	JB	M	12+1	3000		10		10	REC MROU 6	0+10	+	-
6	CB	M	8+10	0	5	10		15	REC MROU 6.5	0+11	+	-
7	PB	M	7+2	3000?	7	5		12	REC MROU 4.5	1+8	+	-
8	AB	M	13+8	0		8		8	REC MROU, REC IOOU	0+10	+	+
									REC RSR 6, resect RLR 6	3+11		
9	SC	M	3+2	0	15			15	REC MROU 14.5, REC RIO 12	1+2	+	-
10	RC	F	5+3	0	15			15	REC MROU 7	0+6	+	+
									REC RSR 8.5, LSR 6	2+0		
									Resect RIR 7, LIR 3	6+2		
11	LC	F	5+8	0	5	5		10	REC MROU 7, REC IOOU 14	0+9	+	+
									REC RSR 4, REC LROU 5	3+11		
12	NC	M	16+1	0		6		6	REC MROU 5.5	0+10	+	-
									Resect LROU 8	1+0		
									RE-REC MROU, REC RIO	2+2		
									Laceration left cornea	8+1		
									REC LROU 7.5	11+1		
13	JC	M	6+9	0	5	9	4	18	REC MROU 7	0+7	+	-

TABLE XIV (CONTINUED): CONGENITAL ESOTROPIA WITH OCULAR POSTURE

NO.	INIT	SEX	AGE	STEREO	TURN	TILT	CHIN	TOTAL (°)°	SURGERY	AGE	DVD	LATENT NYSTAGMUS
									Resect LROU 7	0+11		
									REC IOOU and anteriorize	2+5		
14	CD	F	2+9	0	10	25		35	REC MROU 5	0+11	+	-
15	KD	F	2+8	?	18			18	REC MROU 4.5	2+9	+	-
16	ED	M	10+6	0	5	8		13	REC MROU 5, resect LRL 6.5	0+11	+	+
									REC LLR, vertical R+R OD	4+3		
									REC RSR 6, RLR 4	5+5		
									REC and anteriorize RIO	10+8		
17	KD	F	14+0	0		5		5	REC MROU 6	4+6	-	-
18	RD	M	0+8	?	15			15	REC MROU 7	0+6	-	-
19	TE	F	1+4	?		16		16	REC MROU 7	0+11	-	+
20	JF	M	13+8	0	8			8	REC MROU 6	0+6	+	+
									Resect LROU 4, REC RSR 6, LSR 4	4+11		
21	DF	M	12+5	140	3	7		10	REC MROU 7	3+11	+	-
									REC LIO 14			
22	EG	F	3+0	0	10			10	REC MROU 6.5	0+9	+	+
23	LH	M	1+1	?			10	10	REC MROU 6	1+0	-	-
24	KH	F	10+6	0		10		10	REC MROU 5.5, REC LIO 12	1+0	+	-
									REC RLR 6 and RIO extirpation	3+10		
25	SH	F	5+2	0		10		10	REC MROU 7	1+2	+	-
26	LI	M	4+8	0	15			15	REC MROU 6.5	2+11	+	-
27	MJ	M	2+4	0		5		5	REC MROU 5.5	1+8	+	-
28	AJ	M	9+5	0		14		14	REC RMR, resect RMR	4+0	+	-
29	BL	M	10+5	0		10	4	14	REC MROU 5	0+7	+	-
									Resect LROU 7	2+0		
									REC RSR 4.5	6+9		
30	AL	F	3+7	0	8			8	REC MROU 6	1+2	+	-
31	PL	F	4+4	0	6			6	REC MROU 6.5	0+8	+	-

TABLE XIV (CONTINUED): CONGENITAL ESOTROPIA WITH OCULAR POSTURE

NO.	INIT	SEX	AGE	STEREO	TURN	TILT	CHIN	TOTAL (°)°	SURGERY	AGE	DVD	LATENT NYSTAGMUS
									Resect LROU 7, REC IOOU 12	1+9		
32	MM	M	9+8	0		15		15	REC MROU 7	0+8	+	+
									REC LIO 14	1+2		
33	NM	M	3+4	0	5	8		13	REC MROU 5.5	0+7	+	-
									Resect LROU 8	2+0		
									RIO extirpation, REC RLR 4	2+8		
34	WM	M	9+3	0	15	12	5	32	REC MROU 6	0+6	+	+
35	EM	F	13+6	3000		8		8	REC MROU 6, REC IOOU 14	1+4	+	-
									Resect LROU 5	2+0		
36	JM	M	3+1	0	8	15		23	REC MROU 5	0+8	-	-
37	NN	M	4+2	0	6	8		14	REC MROU	0+8	+	+
38	KN	F	7+3	0	7	10	7	24	REC MROU 7	0+6	+	-
									REC LROU 4	1+7		
									Extirpation IOOU	2+11		
39	AN	F	1+9	0		15		15	REC MROU 6	0+9	+	-
40	CN	M	1+6	?		15	10	25	REC MROU 6.5	0+7	-	-
									Resect LROU 7	1+5		
41	MO	F	6+0	0		15		15	REC MROU	0+10	+	-
									Resect LROU	1+1		
42	AO	F	2+1	0			15	15	REC MROU 5	1+11	-	-
43	TP	F	8+9	0		15		15	REC MROU 6.5	1+2	+	-
44	JP	M	2+2	?		10		10	REC MROU	2+2	+	-
45	TR	F	4+11	0		5		5	REC MROU	0+6	+	+
									Resect RLR 5, LLR 6	0+11		
46	KR	F	9+3	0		8		8	REC MROU 6.5	0+6	+	-
									Resect LROU 8	1+0		
									REC IOOU 14	3+7		
									REC LROU 6	9+1		

TABLE XIV (CONTINUED): CONGENITAL ESOTROPIA WITH OCULAR POSTURE

NO.	INIT	SEX	AGE	STEREO	TURN	TILT	CHIN	TOTAL (°)°	SURGERY	AGE	DVD	LATENT NYSTAGMUS
47	DR	M	8+0	0	20			20	REC MROU 6	0+7	+	+
48	CS	F	9+4	0	5	12		17	REC MROU 6	0+7	+	-
									Resect LROU 6	2+8		
49	BS	M	7+2	0	6	8		14	REC MROU 7	0+6	+	-
50	MS	F	10+11	0	8	6		14	REC MROU 6.5	0+8	+	-
									REC LROU 4, REC IOOU 14	3+10		
51	HS	F	4+3	3000		10		10	REC MROU 6.5, REC IOOU 14	0+8	+	-
52	DS	M	9+7	0	6			6	REC MROU 5	1+8	-	-
									Resect LROU 5.5	3+2		
53	PS	F	7+11	0	10			10	REC MROU	0+8	+	-
54	NS	F	32+1	0			7	7	REC LMR, resect LLR	2?	+	-
55	JT	M	2+11	0	15	10		25	REC MROU 6	0+6	+	-
									Resect LROU 7	1+4		
									REC LIO 14	2+5		
56	ST	M	6+11	0	6	16	4	26	REC MROU 3.5	1+4	+	-
									REC LROU 5	3+1		
									RE-REC LROU 2.5	6+8		
57	CV	M	9+1	0	6	15		21	REC LMR, resect LMR	0+9	+	-
58	KW	F	4+5	0	15	5		20	REC MROU 6, REC RIO 14,	1+10	+	-
									LIO 12			
59	TW	F	15+3	0		8		8	REC MROU 5.5	0+6	+	+
									REC RLR 4, RSR 6, LSR 4	14+2		
60	AW	M	3+0	3000			5	5	REC MROU 5.5	0+6	+	-
61	CY	M	7+0	0	15	10		25	REC MROU 6	0+8	+	-
62	AZ	F	6+6	0		10	5	15	REC MROU 6	0+9	+	-
									Resect LROU 6	1+7		
63	BS	M	9+0	0			10	10	REC MROU 5	0+8	+	+
									Resect LROU 6, REC IOOU 12	3+7		

TABLE XIV (CONTINUED): CONGENITAL ESOTROPIA WITH OCULAR POSTURE

NO.	INIT	SEX	AGE	STEREO	TURN	TILT	CHIN	TOTAL (°)*	SURGERY	AGE	DVD	LATENT NYSTAGMUS
64	EF	M	13+1	0	5	12		17	REC MROU 6 REC LROU 6, RSR 5.4, LIO 14 RE-REC RLR 3, Advance RMR 3, IOOU X	0+9 3+9 5+7	+	+
65	AL	M	4+2	3000	5			5	REC MROU 6	1+5	+	-
66	MM	M	9+8	0		15		15	REC LIO 14 REC MROU 4, RE-REC LIO	0+8 1+2	+	-
67	JM	M	3+1	3000	8	15		23	REC MROU 5	0+8	+	-
68	AM	F	3+4	0	6	6		12	REC MROU 6	0+9	+	-
69	JP	M	3+1	0	5	10		15	REC MROU	2+2	+	-

*Average degrees 14.1.

patients with congenital esotropia who do not present with a head tilt. The group of patients in this study had early surgery for congenital esotropia, yet the sensory test results are not as successful as those reported by Ing and associates.[141,142]

In a retrospective study of 118 patients with the congenital esotropia syndrome, Pratt-Johnson[143] did not find any patient with central fusion, and only 24% of the total demonstrated measurable stereopsis from within the group that demonstrated peripheral fusion. Absence of central fusion is closely related to the sensory findings in this study and also merits further investigation.

PERMITTING FOVEAL FIXATION

Kushner[20] described 10 patients who adopted an abnormal head posture in order to achieve foveal fixation. In his study, he noted 3 patients with ptosis, 3 with ocular muscle fibrosis, 3 with Moebius syndrome, and 1 monocular patient with sixth nerve palsy. The current study included 27 patients, summarized in Tables XV and XVIII. There were 15 male and 12 female patients, ranging in age from 4 months to 18 years 1 month (average age, 5 years),with an average of 12.8° torticollis. Seven patients had observable face turns, and 4 had head tilts. Twenty of the 27 had chin postures, primarily chin up, with 16 patients demonstrating ptosis. Two patients presented with large chalazia occluding the visual axis. Marked ptosis was produced in patient 27 because of the chalazia. Patient 10 presented with a marked chin tuck in order to see over her incorrectly placed bifocal. Only one hemangioma was detected as a cause for ocular torticollis.

NO APPARENT REASON FOR TORTICOLLIS

A group with this title was not described by Kushner.[20] Tables XVI and XVIII summarize this group. There were 57 patients assigned to this group, which accounted for 9% of all patients seen with torticollis. Thirty-two male and 25 female patients, ranging in age from 5 months to 29 years 5 months, with an average age of 8 years 6 months, presented with torticollis averaging 14.1°. There were 28 patients with face turn, at an average of 12°. Thirty-five presented with head tilt, at an average of 11.7°, and 7 presented with chin postures, at an average of 11°. The turn, tilt, and chin posture measurements did not suggest any specific pattern or common denominator. Stereoscopic vision was present, ranging from 20 seconds, to 3,000 seconds in 34 patients. Stereoscopic vision was not measurable in 17 patients, and it could not be tested in 6 patients. The associated diagnoses are tabulated in Table XVI. The spectrum includes esotropia, exotropia, hypertropia, hypotropia, no surgery, multiple surgeries, amblyopia, retinal diseases, chromosome abnormalities, myopia,

TABLE XV: PERMITTING FOVEAL FIXATION

NO.	INIT	SEX	AGE	TURN	TILT	CHIN	TOTAL (°)*	DIAGNOSIS
1	MB	M	0+7			5	5	Ptosis
2	JB	M	2+6			5	5	Ptosis
3	HB	F	0+9			5	5	Ptosis
4	BC	M	3+7			5	5	Ptosis, jaw wink
5	DD	F	0+7			25	25	Ptosis, nystagmus
6	CT	M	6+0	3	10		13	ROP, dragged fundi
7	BR	M	5+3	10			10	ROP
8	MF	M	5+9	12	8		20	Ptosis, Noonan syndrome
9	EH	M	2+3			5	5	Chalazion
10	CH	F	12+5			15	15	Bifocal too high
11	LI	M	0+8			8	8	Ptosis
12	NL	F	4+1			5	5	Hypotropia
13	RM	M	6+10			10	10	Ptosis
14	JN	M	1+2	15			15	? Visual field defect
15	SP	M	0+7			12	12	Ptosis
16	JP	F	0+8			5	5	Hemangioma
17	SP	F	0+4			30	30	Ptosis
18	AR	F	4+10		15		15	Ptosis
19	TR	M	1+3			15	15	Moebius syndrome
20	FS	M	2+4			10	10	Ptosis
21	SS	F	3+5			10	10	Ptosis, hypotropia
22	HS	F	3+10			10	10	Traumatic ptosis
23	JS	F	9+9	7	5		12	Ptosis
24	MS	M	18+1			10	10	Fibrosis syndrome
25	JV	M	12+9	10			10	Moebius syndrome
26	RT	F	13+4			5	5	Ptosis
27	WB	F	10+2	50		6	56	Chalazion/ptosis

*Average degrees 12.8.

hyperopia, astigmatism, nystagmus, "normal" eyes, macula scar, plagiocephaly without superior oblique compromise, cataract, binocular vision, absent binocular vision, limited cooperation because of age or developmental status, or excellent cooperation.

Campos[12] recommended an occlusion test to rule out nonocular torticollis. Each eye is occluded separately while a fixation target is observed. If there is no change in the torticollis, then the etiology is considered nonocular. If the head remains straight after the occlusion of either eye, or if there is a shift in head posture with only one eye occluded, then the torticollis is considered to be ocular in origin. This test should be performed as part of the total evaluation of a patient with torticollis, especially when the etiology is obscure. A change in the fixation pattern may suggest a source of the torticollis. Fine latent nystagmus may be revealed with

TABLE XVI: NO APPARENT REASON FOR TORTICOLLIS

NO.	INIT	SEX	AGE	STEREO	TURN	TILT	CHIN	TOTAL (°)*	DIAGNOSIS
1	CA	M	7+6	0	15	10		25	ET, HT, XT'
2	SB	M	12+11	0		6		6	ET, HT, amblyopia
3	CB	M	15+1	0			10	10	V ET
4	AB	M	3+0	0	4	5		9	Accommodative ET
5	SB	F	7+7	140	20			20	Recess MROU
6	LB	M	3+3	200	5			5	X'
7	RC	M	17+5	70		6		6	-5.00 OU
8	NC	M	4+11	100		10		10	Anisocoria
9	AC	M	5+8	0	8			8	EOM surgery x2
10	RC	M	10+2	70		10		10	Herpes encephalitis
11	MC	M	7+5	100			20	20	Chin tuck, 1+ OA IOOU
12	KC	M	0+5	N.A.	10			10	Infant posture
13	SC	F	1+10	N.A.	10	10		20	X(T)
14	PC	M	12+9	50		12		12	Overactive RIO/ET' =2
15	CD	F	7+6	0		8		8	Optic nerve hypoplasia
16	ED	F	10+2	30	10	25		35	Accommodative ET
17	SD	F	2+1	N.A.	30			30	X'
18	RD	F	4+5	3000	10	10		20	HT
19	CE	M	5+6	70	15			15	Amblyopia
20	MF	M	10+4	40	5			5	ET
21	JG	M	12+1	0	20			20	Recess MROU, IOOU
22	AG	F	11+3	20		8		8	X, X'
23	RG	F	24+5	0		12		12	ET, HT
24	GH	M	29+5	0	5	15		20	ET, HT
25	BH	F	11+3	50		10		10	X'
26	JJ	M	3+3	200		30		30	X'
27	CJ	M	10+1	20	5			5	E'
28	MJ	F	0+11	N.A.		8		8	Plagiocephaly, infant posture
29	LL	F	14+6	0	8	8	5	21	Chromosome 5 deletion
30	ML	F	6+0	0			5	5	Macula scar left
31	EL	F	3+2	3000			5	5	Hypotropia
32	SL	F	14+0	20		8		8	Normal
33	JL	F	4+7	0		20		20	Aphakia, ambly opia/XT
34	KL	M	7+0	0	15			15	Arthritis, right aphakia
35	DM	M	9+3	70		15		15	X(T)
36	PM	M	5+6	3000	8	20	15	43	Down syndrome, ET
37	SM	F	8+4	?	30	25		55	XT, +7.50 OU

TABLE XVI (CONTINUED): NO APPARENT REASON FOR TORTICOLLIS

NO.	INIT	SEX	AGE	STEREO	TURN	TILT	CHIN	TOTAL (°)*	DIAGNOSIS
38	SN	F	13+3	0		5		5	ET, HT
39	MO	M	13+1	70	15	5		20	Astigmatism
40	KP	F	9+8	0		10		10	ET, HT
41	KP	F	5+6	100		10		10	Accommodative ET
42	AP	F	3+2	3000			15	15	X', lid lag
43	FP	F	11+6	30	5			5	Normal
44	MP	M	9+8	3000	10			10	XT
45	CR	F	6+6	20	10			10	Normal
46	JR	M	13+3	70		6		6	Accommodative ET
47	MR	M	6+9	0		8		8	HT
48	CR	M	1+8	?		15		15	Astigmatism
49	PS	M	5+3	30	15			15	E'
50	RS	F	7+4	100		10		10	Cataract
51	AT	F	9+3	3000		10		10	ET
52	AU	M	17+6	25		8		8	Myopia
53	JU	F	4+5	3000	8			8	ET
54	DW	M	2+9	200	25			25	Normal
55	JX	M	9+6	0	8			8	ET
56	AB	M	6+5	25		5		5	X' 15
57	SH	M	6+11	100	7	7		14	X(T)

*Average degrees 14.1.

careful observation, with the slit-lamp biomiocroscope or by indirect ophthalmoscopy.

Hertle and Zhu[14] studied 298 patients with abnormal head posturing, with use of infrared oculography (IROG) to assist in diagnosis. Of the group studied, 116 patients (39%) had head posturing with or without nystagmus. Eleven children, ranging in age from 11 to 39 months, with abnormal head and face posturing, lacking evidence of strabismus, orthopedic neck disorders, and clinical nystagmus, were found to have congenital nystagmus revealed by IROG. These 11 patients represented 4% of their total study group, and 10% of their patients with torticollis. Patient 47 and his family were able to travel to Dr Hertle's laboratory for IROG, and his results were normal.

The group of patients with unexplained ocular torticollis in this study also account for almost 10% of the total patients with torticollis. It is certainly possible that one or more of the patients have nystagmus detectable only by IROG. However, all patients were examined for nystagmus in the slit-lamp biomicroscope and by indirect ophthalmoscopy, and the likelihood of the majority of this group demonstrating nystagmus by IROG as the etiology for torticollis seems remote. Eleven patients of the 57 with

unexplained torticollis fell within the age range described by Hertle and Zhu, where nystagmus was found with IROG.

Bagolini striated glasses are useful in assessment of torticollis. Campos[12] described ARC, anomalous retinal correspondence with striated lenses, only in the position of torticollis, with diplopia or suppression in the other head positions. The anomalous type of binocularity or binocular vision provides the patient with a more advantageous visual state, with the patient assuming a pathological head position in order to achieve anomalous binocular vision. The results of testing with Bagolini striated glasses have been inconsistent, especially with children under 8 or 9 years of age, who have difficulty understanding the concept and verbalizing a mature response, even after various diagrams or charts are used.

von Noorden[121] recommended the Bagolini striate lenses because the lenses permit a nearly normal view of the visual environment, are not dissociating, and do not interfere with cyclofusion. The streaks of the lenses are placed on the horizontal axis in the trial frame, and a vertical prism is placed before one eye to separate the streaks.

The pathophysiology of idiopathic torticollis was investigated by Straube and Dieterich.[145] Possible disturbance of the vestibular system was investigated in 40 patients with idiopathic torticollis by using electroystagmography, fundus photography, measurement of the subjective visual vertical, and posturography. Results of the measurement of the visual vertical and the neuro-ophthalmologic testing were normal. There was slight pathologic monocular torsion in the fundus photography in 38% and a slight central vestibular preponderance in the electroystagmography in 53% of the patients. There were some indications of slightly decreased gain in visual contribution to the postural control process as measured posturographically. However, none of the results could be correlated with the extent of torticollis in each case. The investigators proposed that the results did not confirm the hypothesis of a primary vestibular lesion in the pathogenesis of the idiopathic torticollis. They suggested the possibility of a common basic lesion causing the torticollis as well as affecting the central vestibular system.

OCULAR TORTICOLLIS ASSOCIATED WITH MEDICAL AND NEUROLOGIC CONDITIONS

Table XVII and XVIII summarize this group, which comprises 25 patients, 14 male and 11 female, ranging in age from 6 months to 83 years (average age, 13 years 2 months). The average torticollis was 17.3°, primarily from face turns or head tilts, and occasionally from a chin posture. These medical and neurologic conditions are all present in the general population without ocular torticollis. This group of patients having ocular torticollis,

TABLE XVII: OCULAR TORTICOLLIS WITH ASSOCIATED MEDICAL/NEUROLOGIC CONDITIONS

NO.	INIT	SEX	AGE	STEREO	TURN	TILT	CHIN	TOTAL (°)*	DIAGNOSIS
1	SB	M	3+2	100	5	12		17	Traumatic brain injury
2	JB	M	7+2	100		10		10	Scoliosis
3	FC	M	83	30		12		12	Thyroid
4	NC	F	5+9	?	15			15	Brain tumor
5	TD	M	7+2	?	10			10	Hydrocephalus
6	KE	F	3+6	100	7	20		27	Vertebral fusion
7	NF	F	3+1	?	25			25	Traumatic brain injury
8	LF	F	4+7	200	10	10		20	Myelomeningocele
9	SG	F	10+7	0	10	5		15	Neurological/ plagiocephaly
10	AH	F	33	70			10	10	Traumatic brain injury/fusion
11	JJ	M	13+1	50		15		15	Osteogenesis imperfecta/ank spon
12	SL	F	0+7	?		8		8	Infant posture
13	LM	M	23+6	0	12			12	Tumor/hydro- cephalus/shunt
14	JM	M	62	3000	12	7		19	Ependymoma/ myasthenia gravis
15	JM	M	5+5	?	20			20	Hypotonia
16	MN	F	4+3	100	10			10	Cerebral palsy
17	MP	M	8+5	50	10	10	5	25	Spinal muscular atrophy
18	RR	F	9+5	0	7	5		12	Cerebral palsy
19	NR	M	3+0	?	15		10	25	Arthrogryposis syndrome
20	KS	M	2+10	100			10	10	Macrocephaly
21	JV	F	0+6	?			5	5	Myelomeningocele
22	DA	M	5+0	3000		20		20	cerebral hemor rhage/hemianopsia
23	SB	F	14+5	?	5	25		30	TBI/brain stem contusion
24	DB	M	11+0	?	12	15		27	Traumatic brain injury
25	JP	M	3+1	0	20	15		35	Ventricular hemorrhage/ cerebellar Encephalomalacia

*Average degrees 17.3.

included patient 6, whose torticollis was not orthopedic in nature, despite the vertebral fusion. The response to stereopsis testing was varied, as seen

TABLE XVIII: SUMMARY OF TORTICOLLIS GROUPS

TABLE	DIAGNOSIS	PATIENTS	MALE	FEMALE	AVE TORT (°)	AVERAGE AGE	AGE	RANGE
IV	Superior oblique palsy	59	31	28	13.9	14+6	2+0	76+0
V	Inferior oblique palsy	6	2	4	16.8	21+0	6+10	45+11
VI	Brown	25	10	15	9.4	11+3	2+0	49+0
VII	Double elevator palsy	9	3	6	19.3	19+1	0+4	73+6
VIII	Duane syndrome	46	22	24	14.3	9+1	0+7	34+8
IX	Acq hor incom asym surgery	48	22	26	13.3	21+3	3+2	82+7
X	Acq hor incom-A or V pattern	116	67	49	12.1	10+9	1+0	87+6
XI	6th nerve palsy	15	6	9	15.5	32+11	1+2	81+0
XII	Torsional incomitance	4	3	1	17.5	26+6	8+4	61+11
XIII	Nystagmus	120	75	45	21.1	9+2	0+9	65+6
XIV	Congenial esotropia/ ocular posture	69	39	30	14.1	7+4	0+8	32+1
XV	Permitting foveal	27	15	12	12.8	5+0	0+4	18+1
XVI	No apparent reason for torticollis	57	32	25	14.1	8+6	0+5	29+5
XVII	Assoc w/ med/neurol conditions	25	14	11	17.3	13+2	0+6	83+0
None	Superior rectus palsy	1	0	1	23	3+9	3+9	3+9
None	Third nerve palsy	1	1	0	17	14+0	14+0	14+0
None	Spasmus nutans	2	1	1	20	2+6	2+5	2+7
	Summary	630	343	287	15.7	13+3	0+4	87+6

in other groupings, ranging from 30 seconds to 3,000 seconds; 4 patients were without stereoscopic vision, and 8 who could not be tested. A common factor to explain the head postures in this group was not apparent. Because of the various medical and neurologic associations in this group, the etiology of the torticollis could not be objectively compared between patients, and further evaluation of this group was not considered for this

study.

SUPERIOR RECTUS PALSY, THIRD NERVE PALSY, SPASMUS NUTANS

One patient with a vague history of trauma presented with left hypertropia and underaction of the right superior rectus at age 2. She initially responded to a recession of the right inferior rectus, but in time developed right hypertropia, with a 15° left head tilt and 8° right face turn.

A 14-year-old boy had partial third nerve paralysis after falling out of a tree and was not a candidate for surgery. He lacked many of the findings usually seen in complete third nerve palsy.

One infant with spasmus nutans had surgery for esotropia at 11 months of age, and the other infant did not require surgery. Both patients have demonstrated reduction in nystagmus and in head bobbing.

DISCUSSION

During the past 20 years, this ophthalmology practice has seen a number of patients exhibiting ocular torticollis for no apparent or obvious reasons. The impetus for this study was to determine the cause of ocular torticollis within this group of patients and to compare results with others who have studied ocular torticollis, especially Kushner,[20] who found 8 basic mechanisms responsible for abnormal head postures. The intent of this paper, therefore, was to prospectively study, for 1 year, all patients who presented with torticollis of ocular origin and to identify and reevaluate the etiology of ocular torticollis among different groups, including those with unexplained ocular torticollis.

Unexplained ocular torticollis has been a historical reality. In a review of nonophthalmologic causes of torticollis, Boutros and Al-Mateen[146] state, "There remain a group of patients, where the cause will remain unclear." In reviewing causes for ocular torticollis, Kushner[20] states, "Care, however, must be taken to evaluate these patients properly, as the mechanism of the head posture is not always obvious." In their review of ocular torticollis, Rubin and Wagner[147] conclude, "Some patients may have an abnormal head position with no satisfactory explanation: This is a diagnosis of exclusion after all ocular and nonocular causes have been eliminated." Von Noorden[118] states, "The cause of an ocular head tilt in the absence of a cyclovertical muscle imbalance is not always clear," and in reviewing clinical and theoretical aspects of cyclotropia, von Noorden[121] states that "in a number of patients inconsistent (sensory) responses were obtained that defy explanation at this time and will not be considered here." In congenital esotropia with ocular posture, with a high incidence of DVD, Lang[133,134] notes that the head posture is not a mechanism for avoidance of

diplopia, since binocular vision is required for diplopia to be experienced. Bechtel and associates[135] reported a high incidence of head tilts in patients with DVD and congenital esotropia and also claim that DVD is a frequent cause of head tilts. Lang,[133,134] Bechtel and associates,[135] and others[136-140] have thus described head tilting in association with congenital esotropia and DVD but have not explained the mechanism of the head tilting. The ocular torticollis in these authors' series, as well as the torticollis in the group in this study, without apparent reason, maintains a head posture because of a presumed sensory adaptation or advantage, which is difficult or impossible for most patients to verbalize.

This author was successful in determining a number of the etiologies for ocular torticollis. In addition, after evaluation and reevaluation of patients with unexplained ocular torticollis, this author was able to reduce the number of cases of unexplained ocular torticollis. The results of the present study are best compared with that of Kushner,[20] who found 62.7% of patients with incomitance, compared with 52.4% found in this study. A similar incidence of nystagmus, 20.2%, paralleled this study at 19%. Congenital esotropia with ocular posture occurred in 6.3%, while this study found 10.9%. Permitting foveal fixation produced 5.3% in Kushner's study, as compared to 4.3% with this study. The remaining categories included a few patients with ocular posture for cosmetic reasons, ocular motor apraxia, spasmus nutans, and astigmatism. However, this study found 57 patients, or 9%, without obvious or apparent reasons for ocular torticollis. While many parameters were reviewed and possible reasons explored, no definite common factor or factors were found to explain the large number of patients in this group with varied visual and sensory responses demonstrating ocular torticollis, also without any evidence of musculoskeletal or neurologic etiology. All of the usual sensory tests available to the general ophthalmologist were used, but etiological questions remain.

Some patients were recategorized, reducing the number of cases of torticollis without any apparent reason. A few patients with several ophthalmic diagnoses could be placed in different categories. Each patient with torticollis above 5° was evaluated in the prospective study of torticollis in this practice and classified within a category outlined by Kushner.[20] Torticollis measurements were obtained by goniometer for optimal accuracy and reproducibility, and the 630 patients were classified within specific categories during the 1-year prospective study.

It should be noted that this author's effort to recategorize those patients with unexplained torticollis may not be without criticism. Any effort at reevaluating only one group of patients could potentially compromise the results of the study by affecting various other outcomes and

percentages within each category of patients. In addition, it should be noted that upon reevaluation, some patients with unexplained ocular torticollis demonstrated improvement or resolution of their head posture. It is therefore also quite possible that patients in other categories experienced similar outcomes.

With these concerns in mind, specific reexamination was undertaken of those 57 patients in the group with unexplained torticollis, in an effort to define the etiology of the torticollis. Fourteen of the 57 patients were lost to follow-up, despite extensive and careful searches. Of those patients who were able to return for reevaluation of the torticollis, 9 have shown a reduction or resolution of their torticollis. Of the 34 remaining patients without change or improvement in their torticollis, 7 could not comprehend sensory testing by way of double Maddox rod testing, Bagolini striated glass testing, Lancaster red-green testing, and objective torsion evaluations.

In Table XVI, the patients who were unable to be reevaluated sensorially included patient 11, who has developmental delay; patient 21, who is in special education and exhibits behavior problems; patient 29, who has chromosome 5 deletion and developmental delay and is nonverbal; patient 34, who is now blind from severe uveitis; patient 35, who has developmental delay; patient 36, who has Down syndrome with limited testing capability; and patient 54, who has developmental delay. While the initial findings of these patients did not merit classification under medical/neurologic conditions, some of the patients in this group could now be reclassified in Table XVII as patients exhibiting ocular torticollis with associated medical or neurologic conditions. Other reclassifications might include dissociated vertical deviation as a probable etiologic factor for the torticollis in patients 18 and 40, and incyclotorsion in patient 7, which may explain his right head tilt, to be described below.

Table XIX summarizes the sensory findings of 16 patients from the group who were reevaluated. The first column lists the patient number previously used in table XVI. The total degrees of torticollis are listed for comparison with the initial evaluation. Under the headings "Double Maddox" and "Bagolini," "Ex" represents excyclotorsion and "In" represents incyclotorsion. Under "Fundus torsion," the notations 1+, 2+, and 3+ are based on the grading system for estimating abnormal torsion established by Guyton.[119] The diameter of the disc is used as a guide, and abnormal torsion is graded according to each eighth of a disc diameter of abnormal displacement of the fovea from the normal range, which is within the upper third of the disc in the indirect ophthalmoscopic view. Of note is presence of torticollis in the absence of binocular vision in several patients.

Several reevaluated patients are worthy of discussion. Patient 7

TABLE XIX: NO APPARENT REASON FOR TORTICOLLIS: REEXAMINATIONS

NO.	INIT	AGE	BEST VISION		STRABISMUS		STER	W4D	TURN
			OD	OS	D	N			
2	SB	17+6	20/100	20/40	ET20	ET'25	0	ODSup	5R
6	LB	8+2	20/25	20/25	0	0	20	Norm	
7	RC	22+3	20/20	20/20	0	0	30	Alter	
13	SC	6+8	CSM	CSM	RHT5	ET'8	?	?	15R
26	JJ	16+4	20/20	20/20	0	X'4	20	Norm	
28	MJ	7+3	20/25	20/30	0	O	160	Norm	
31	EL	7+7	20/20	20/20	0	E'2	25	Norm	
32	SL	19+0	20/20	20/25	0	0	25	Norm	
33	JL	8+10	2/200	20/20	0	RET'30	0	ODSup	
36	PM	9+0	20/30	20/30	ET10	ET'14	3000	Norm	
46	JR	17+11	20/15	20/20	LET2	LET'4	140	OSSup	
47	MR	11+4	20/25	20/25	ET4	LHT12 XT'8	200	Alter	
48	CR	7+0	20/25	20/30	RET3	RET'5	400	ODSup	10L
49	PS	10+3	20/15	20/20	0	E'2	25	Norm	12L
53	JU	8+8	20/30	20/25	E 6	E'6	3000	ODSup	5R
55	JX	14+6	20/20	20/20	ET14	RET'18	40	ODSup	5L

preferred fixation with the right eye, with a persistent right head tilt. The double Maddox rod test, Bagolini, and Lancaster red-green tests revealed incyclotorsion of both eyes. Although cyclotorsion is a rare cause of torticollis, this patient stated that the horizontal lines of the eye chart, as well as distant objects, appeared parallel to the floor when he tilted his head to the right. However, when his head was held in primary position or in left tilt, he described the lines on the eye chart and distant objects at an "angle" to the floor. He preferred using his right eye, with right head tilt posture, presumably to reduce the right incyclotorsion, rather than upright posture or left head tilt with increasing right incyclotorsion. Patient 7 had not been able to describe this preference when last seen at age 17. Therefore, this patient could be reclassified as torticollis due to torsional incomitance.

Patient 53 has persistent torticollis, suppression of OD, and inconsistent sensory responses, including 10° right incyclotorsion and 10° left excycltorsion confirmed with double Maddox rod testing and Lancaster red-green tests (Fig 4). However, torsion was not detected with the

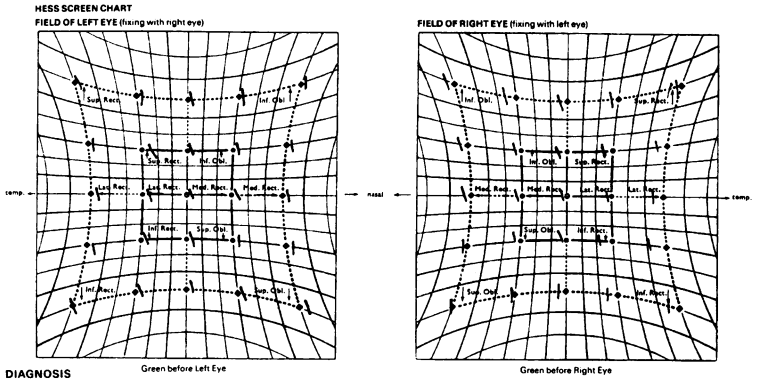

FIGURE 4
Lancaster red-green test demonstrating incyclotorsion OD and excyclotorsion OS.

Bagolini striated glasses, and her fundus examination revealed 2+ incyclotorsion, both eyes. Patient 55 still presents with torticollis yet has had inconsistent sensory responses and no fundus torsion. Of the 2 patients with autism, patient 26 was more able and cooperative than patient 13, yet both patients' tests did not reveal the etiology of the torticollis. Patient 32 has now shown resolution of her torticollis, and the etiology of her previous torticollis remains unknown. The remainder of the patients in Table XIX demonstrate varying degrees of torticollis and strabismus, and almost

all have normal double Maddox rod testing, Bagolini striated glass testing, and Lancaster red-green testing, yet several demonstrated objective fundus torsion.

The status of the torticollis of the 14 patients lost to follow-up is unknown, and until proven otherwise, these patients should remain classified as having torticollis without apparent reason. The torticollis of 9 patients has been resolved. Two additional patients could be reclassified with dissociated vertical deviation as the etiology of their torticollis, and one patient could be reclassified with ocular torsion as his torticollis etiology. This reduced the number of patients with unexplained torticollis from 57 to 45, reducing the percentage of unexplained torticollis to 45 of 630, or 7%.

CONCLUSION

This study has found patients with ocular torticollis not of orthopedic or neurologic etiology, which defies clinical explanation. Because a number of patients demonstrate contradictory sensory testing, unanticipated and unpredictable testing responses, and the frequent and consistent inability to verbalize the reasons for their preferred head tilt, further studies are indicated.

Other areas for future study include an evaluation of patients with Down syndrome, with a seemingly high number found with A and V pattern, as compared with all other groups. The limited response to sensory testing in the group with congenital esotropia has been well documented in other studies.[133-136,138-140,143] A future study is planned to review our large group of infants with congenital esotropia without torticollis for comparison of their sensory findings, as published in other studies.[134,141-143] The range of stereoscopic vision in the A and V pattern groups also suggests further investigation, as the head postures did not consistently correlate with the clinical eye muscle patterns.

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