INFERIOR OBLIQUE SURGERY FOR RESTRICTIVE STRABISMUS IN PATIENTS WITH THYROID ORBITOPATHY

BY **Steven A. Newman MD***

ABSTRACT

Introduction: Thyroid orbitopathy is the most common cause of restrictive strabismus. Patients often present with vertical or horizontal double vision, or both, due to restriction involving most commonly the inferior and medial rectus muscles. Traditional muscle surgery involves release of the tight muscles. Previous literature has described a frequent need for secondary operations and an overcorrection incidence of up to 50%. Recognizing that the tight muscles are also limited in their excursion, it was proposed that operating on the better-moving eye, particularly the inferior oblique, might produce an improvement in binocularity and decrease the incidence of overcorrection.

Methods: A total of 37 patients with restrictive strabismus due to thyroid orbitopathy treated at the University of Virginia over 12 years with inferior oblique surgery were retrospectively reviewed.

Results: Eight patients were treated with a combination of inferior oblique surgery and horizontal muscle surgery at the same time. One patient was treated with simultaneous inferior oblique and superior rectus surgery. Seven patients had vertical correction with inferior oblique surgery alone. Twenty-three patients required secondary procedures. Eight patients were overcorrected but only one following primary surgery. At the time of last follow-up, ranging from 6 months to 8 years, 33 patients had no diplopia, 2 had minimal diplopia, and 2 had persistent diplopia. All but two were completely functional.

Conclusion: Inferior oblique surgery by balancing the overall excursion of extraocular muscles in thyroid patients may produce binocularity in primary position and down reading gaze. The amount of vertical correction from inferior oblique surgery alone is limited, often requiring ipsilateral superior or contralateral inferior rectus surgery. Inferior oblique surgery likely increases the area of binocular single vision and decreases the incidence of overcorrection. The use of Hess screen and binocular single vision fields is helpful in assessment and planning of surgery in these patients.

Trans Am Ophthalmol Soc 2009;107:72-96

INTRODUCTION

While irritation, tearing, and pressure sensation associated with lid retraction, proptosis, and so on, are the most common symptoms and signs in patients with thyroid orbitopathy, no other symptom is as frequently disabling as double vision. Diplopia occurs in up to 40% of patients with thyroid orbitopathy,[1] making it more common than optic neuropathy. Procedures on the extraocular muscles are the most common ophthalmic surgical procedure in patients with Graves disease, although some groups reports higher rates of orbital or lid surgery.[2] In a population study,[3] men were more likely than women to require decompressive and strabismus surgery. The need for strabismus surgery also increases in frequency in patients who have undergone orbital decompressive surgery. Estimates suggest that upwards of 50% of patients will have a change in their motility following transantral decompression of the medial wall and floor of the orbit.[4]

The hallmark of thyroid orbitopathy is enlargement of the extraocular muscles.[5] Direct infiltration of the extraocular muscles has been demonstrated, but there may be an additional component related to venous outflow impairment.[6] Early speculation centered on muscle weakness, but most patients with thyroid orbitopathy have diplopia secondary to restrictive strabismus. This can be confirmed by forced ductions, force generation or saccadic velocity,[7] and elevation of intraocular pressure in eccentric gaze.[8-10] Mild intraocular pressure elevation may be a subclinical sign of thyroid orbitopathy,[11] which correlates with enlargement and fibrosis of the extraocular muscles.

During the acute phase characterized by muscle swelling, there is some evidence that botulinum toxin injection may be of some use[2,12-14] and may have the added benefit of reducing intraocular pressure.[14] In later stages with fibrosis, it is less clear that botulinum toxin is likely to be effective. High-dose intravenous corticosteroids have been noted to have an occasional beneficial effect,[15] but there is little evidence that corticosteroid therapy has a marked positive effect in most patients with restrictive strabismus.[16,17] Recent work suggests that more aggressive immune-modulating therapy, including cyclosporin,[18] may play a role in limiting diplopia in patients with thyroid orbitopathy, but strabismus surgery remains a mainstay of realignment in patients with persistent double vision problems. Although radiation therapy potentially plays a role in acute orbital inflammation,[19-21] its role in substantially improved motility remains unclear.[22-24] Prisms offer alignment but have limited applicability owing to the incomitance of the deviation. Occasionally patients may do well with Fresnel prisms.[25]

Early surgery on the restricted muscles often involved a fixed 5.5-mm recession with permanent sutures occasionally combined with a partial myotomy.[26] Because of the unpredictability of surgical approaches in patients with thyroid disease, attempts have been made to improve the results of strabismus surgery by modifying the technique. Modifications have included use of adjustable sutures,[27-31] intraoperative relaxed muscle positioning,[32] and surgery targeting the restrictive component, as opposed to simply alignment.[33,34] Prendiville and colleagues[35] suggested that the restriction in ductions (particularly of the contralateral vertical rectus

*Presenter.
Bold type indicates AOS member.
From the University of Virginia Health System, Charlottesville.

muscle) best predicted the amount of surgery necessary. Buckley[36] and others[32,37] have pointed out the change in the nomogram, where small recessions produce less than expected results, while large recessions are associated with larger than predicted changes in alignment.

In spite of these advances in technique, thyroid strabismus surgery has remained unpredictable, with a high frequency of overcorrection and the need for additional operative procedures, particularly when combining horizontal and vertical surgery.[38] It has been postulated that the overcorrection often is secondary to unrecognized restriction in the ipsilateral antagonist muscle. Other theories have included secondary scarring,[39] particularly around the Lockwood ligament, and slippage of the recessed muscle.[36] This is felt to be a particular problem with remodeling after the use of absorbable sutures.[40]

Because the most involved muscle is not only fibrotic but also relatively paretic, it was postulated that maximal binocularity could be obtained by early consideration of intervention on the contralateral, better-moving eye. Limitation of movement of the normal or more normal eye is a mainstay of surgery in persistent paretic conditions[41] and has been employed in patients with restrictive strabismus. Most reports that address contralateral surgery have suggested operating on the contralateral superior rectus in the setting of vertical incomitance due to inferior rectus restriction,[25,42] either with recession or even a Faden posterior fixation procedure.[36] Since the antagonist to the contralateral yoke muscle is the inferior oblique, and not the superior rectus, we postulated that the vertical incomitance across the horizontal could be collapsed by operating on the contralateral inferior oblique muscle.

METHODS AND MATERIALS

This is a retrospective review of 38 patients with restrictive strabismus due to thyroid orbitopathy seen at the University of Virginia over the last 12 years who underwent inferior oblique surgery. Consideration was given to any preceding muscle or orbital surgery. Patients were treated with isolated inferior oblique surgery as well as with combined additional extraocular muscle surgery. None of these procedures were combined with orbital or lid surgery. Twenty-five of the 38 patients were women, and 13 were men. Ages ranged between 34 and 78 years at the time of initial evaluation; average age was 59. Duration of known thyroid disease varied from unknown at the time of first referral in 7 patients to 8 years following first diagnosis of thyroid disease in one patient. Often the diagnosis of thyroid disease was obtained only after detailed questioning about past medical history, including previous treatment with radioactive iodine, previous hypothyroidism, and symptoms of both hypothyroidism and hyperthyroidism. Twelve of the 38 patients had orbital decompressive operations performed prior to strabismus surgery. Two patients required late orbital procedures. Several patients without classic findings had their diagnosis confirmed through thyroid function tests and computed tomographic (CT) orbital scanning.

All patients were evaluated with Hess screen tests when first seen and in follow-up. Deviation was assessed for comitance with a Maddox rod, and stereoacuity was measured by Titmus. Binocular single vision fields were done when binocularity was present. Patients were also measured in primary position and down reading gaze. Pneumotonometry was often performed in eccentric gaze to confirm restriction.

The surgical procedure was performed under regional block anesthesia supplemented with intravenous sedation. The inferior oblique muscle was approached through a fornix incision in the inferotemporal quadrant. The lateral rectus was placed on a muscle hook, and the inferior oblique insertion was exposed with a malleable retractor. The inferior oblique was dissected from the surrounding tenons and disinserted with sharp dissection. A clamp was placed on the muscle, and the muscle hook was moved to the inferior rectus. A cotton-tip applicator was placed between the inferior oblique and the globe. The inferior oblique was severed using bipolar cautery just lateral to the edge of the inferior rectus. This ensured that the neurovascular bundle was included. The proximal inferior oblique was noted to retract into its tenons tunnel. The conjunctiva was closed with interrupted sutures. The distal portion of the inferior oblique was sent for pathologic examination, which revealed a high incidence of low-grade inflammation and infiltration,[43] similar to other reports.[44]

RESULTS

One patient was subsequently diagnosed as having myasthenia gravis, but had previously been stable with good binocular vision. Several patients were studied with single-fiber EMG analysis to exclude myasthenia. One patient was diagnosed as having multiple sclerosis. One patient developed a third nerve palsy several years after muscle surgery, and one patient had a stroke, producing an incomplete homonymous hemianopsia and dysphasia.

One patient had bilateral inferior rectus surgery prior to the inferior oblique surgery. Another patient had bilateral lateral rectus and a contralateral inferior rectus procedure (producing a consecutive hypertropia).

Two patients were treated with simultaneous inferior oblique and ipsilateral superior rectus surgery, resulting in initial correction going from 35 diopters (D) to straight and from 28 D to 7 D. However, both patients subsequently developed consecutive contralateral hypertropia, which required contralateral inferior oblique and superior rectus surgery in one patient and contralateral inferior oblique and ipsilateral inferior rectus surgery in the other.

Only 8 patients of the 38 did not require additional vertical muscle surgery, and 3 of these had follow-up of 3 months or less (Table). One of these patients did require a second procedure after late orbital decompressive surgery.

For the second vertical muscle, 9 patients underwent ipsilateral superior rectus recessions and 14 patients had contralateral inferior rectus recessions. Overcorrection led to secondary ipsilateral inferior rectus recessions in two patients and contralateral superior rectus recession in one. Six patients eventually had inferior oblique surgery on the contralateral side. Fifteen patients required 3 or more

TABLE. RESULTS FOR THYROID ORBITOPATHY PATIENTS UNDERGOING INFERIOR OBLIQUE SURGERY

PATIENT	DATE OF SURGERY	AGE	GENDER	MONTH OF SYMPTOMS	PREVIOUS EOM SURGERY	PREVIOUS ORBITAL SURGERY	ADD-ITIONAL MUSCLES AT FIRST SURGERY	INITIAL VER-TICAL PRISM	SHORT-TERM VERTICAL FOLLOW-UP (<6 WK)	2ND OPER-ATION	3RD OPER-ATION	4TH OPER-ATION	MONTHS TO MOST RECENT FU	STEREOP-SIS SECONDS AT LAST VISIT	ADDITIONAL COMP-LICATIONS	PRISM IN GLASSES
1	7/15/1996	53	F	0			iSR	28	7	cSR	cIO		153	50		
2	5/19/1997	77	F	4	bIR			18	4				120	40		
3	6/18/1998	47	F	24				8	-4	cIO			92	40		
4	10/5/1998	51	F	12				17	5	cIR			96	40		
5	10/5/1998	60	F	24			iMR	6	0				3	40		
6	11/16/1998	73	F	3		cOrbit		43	40	cIR + cMR	cIO		24	Str		
7	12/28/1998	61	F	14				18	3				22	200		
8	1/25/1999	36	M	3		bOrbit		6	3				1	60		
9	2/1/1999	56	M	24				14	14	Orbit	cIR		7	140		
10	2/8/1999	73	M	48				27	18	cIR	iSR		33	No double		
11	7/19/1999	51	F	96		cOrbit		10	5				1	140		
12	10/4/1999	35	F	1		cOrbit		40	40	cIR + cMR	cSR	iIR	63	200		
13	12/13/1999	57	F							cIR	cSR			Double		
14	1/17/2000	76	M	13		cOrbit	cMR	6	9	Orbit					IIIrd nerve palsy	
15	2/28/2000	70	F	48				18	16	cIR + cMR			110	60		
16	4/10/2000	73	F	3				25	14	cIR			60	40		
17	6/12/2000	78	M	4		bOrbit	bMR	0	-3	iIR			10	140	MG	
18	9/25/2000	69	M	3				12	3				4	40		
19	2/26/2001	78	F	4				43	40	cIR			30	100		
20	3/19/2001	52	F	96				14	18	cIR	iSR	cIO	19	No double	MS	
21	5/21/2001	44	M	2				30	45	cIR	iIR+iMR		60	60		
22	1/20/2003	55	F	12		cOrbit		22	30	iSR	cMR		51	No double		6 D prism
23	8/4/2003	65	F	5				25	12	iSR	cSR	cIR	57	80		3 D prism
24	8/11/2003	54	M	3				45	15	iSR	cIO+cMR	iIR	34	200		
25	10/6/2003	63	F	84				18	18	iSR			20	40		
26	6/16/2004	67	F	48				10	2	iSR			28	40		
27	6/17/2004	46	F	9				35	40	iSR+iLR			27	40		
28	6/21/2004	56	M	10		cOrbit	iMR	50	28	cIR			9	40		
29	8/23/2004	70	M	84			iLR	22	11	cIR	cSR		31	80		
30	1/17/2005	74	F	74		bOrbit	iLR	3	0	iIR			39	40		
31	1/19/2005	34	M	6		bOrbit		40	8	bIR			10	40		
32	1/31/2005	61	F	11			iSR	35	0	cIO	iIR		44	60		
33	9/12/2005	62	F	12				25	16	iSR			23	40		
34	4/17/2006	35	F	13				16	28	iSR			16	40		
35	5/22/2006	65	M	13			iLR	18	10	cIR			29	140	Stroke	Prism
36	6/18/2007	57	F	84		bOrbit		8	7	cMR	cIR		15	50		
37	6/25/2007	50	F	66	cIR+bMR	bOrbit	iLR	15	10	cIR			15	60		
38	9/21/2007	48	M	18				20	14	iSR	iIR		15	Double		

EOM, extraocular muscle; MG, myasthenia gravis; MS, multiple sclerosis; Str, straight.

operations, although in one of these the third operation was only a medial rectus recession.

Progressive undercorrection occurred in 4 patients with short-term measurements worse than preoperative measurements, indicating that the thyroid disease was not stable. In 3 additional patients there was minimal vertical effect in primary position, although the Hess screen test often indicated a collapse of the secondary overaction.

In patients with severe hypotropia, 30 D or greater, marked undercorrection was common, occurring in 7 patients, 2 of whom were treated with subsequent ipsilateral superior rectus recessions and the remaining 5 with contralateral inferior rectus recessions.

Long-term follow-up ranged from 10 months to10 years. At the time of last follow-up, only 2 patients still had symptomatic diplopia in primary and reading gaze. Three patients had residual prism in their glasses to correct minimal residual vertical deviation. Many patients who denied having any double vision could be found to have mild residual abnormalities on their Hess screen test, but they were symptomatically without complaint.

CASE EXAMPLES

Case 1

A 77-year-old woman was referred in May 1996 with complaints of double vision following cataract surgery. Afferent system function was normal. Hertel readings were 16 OU without lid retraction. Motility examination revealed marked limitation in elevation, worse in the left eye than the right, with 16 D of right hyperdeviation increasing on up left gaze. Intraocular pressure measurements revealed 5 mm of elevation of intraocular pressure on attempted up gaze. A CT scan showed mild enlargement of the inferior rectus muscles greater than medial rectus muscles, worse on the left than the right. Because of the incomitant deviation (Figure 1, left), inferior oblique surgery was suggested. The patient asked for a second opinion, and it was suggested that inferior rectus surgery be performed. The patient underwent a left inferior rectus recession on adjustable stitch. Postoperatively, she had 4 D of right hypertropia in primary position and 9 D of left hypertropia on down gaze. Hess screen test confirmed the change in position of the patient's left eye, but still with significant restricted vertical gaze bilaterally (Figure 1, middle). Subsequently, in December 1996, she underwent a right inferior rectus recession. Postoperatively she was straight in down reading gaze, but retained 10 D of left hypotropia in primary position (Figure 1, right). In May 1997 she underwent a right inferior oblique extirpation and denervation, which collapsed the right hypertropia as seen on follow-up Hess screen and binocular single vision field tests (Figure 2). Initially she felt there was a major improvement in her double vision, but she returned 2 months later complaining of recurrent problems with double vision. She then underwent a left inferior oblique denervation and extirpation. Immediately postoperatively she noted double vision only in up gaze. Follow-up 10 years later revealed no double vision at all with 60 seconds of stereopsis on Titmus testing (Figure 3).

Comment. Although standard inferior rectus recessions released the restrictive strabismus here, this did not resolve the incomitant deviation with a persistent hypotropia on abduction. By extirpating the inferior oblique muscles, this apparent "secondary overaction" could be eliminated and the binocular single vision field expanded dramatically.

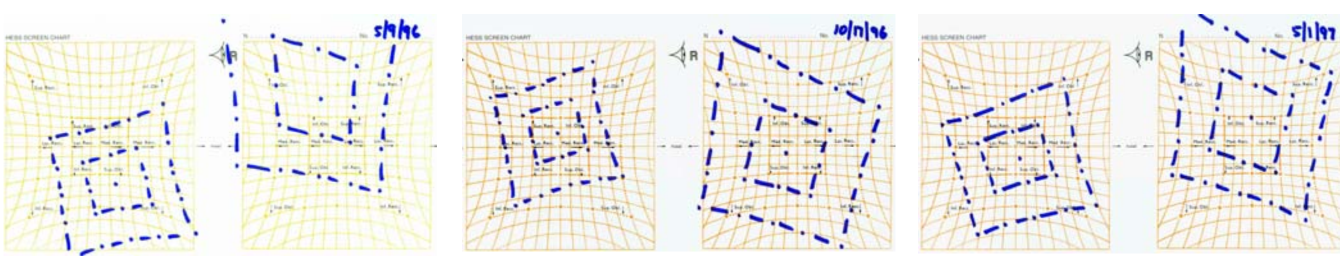


FIGURE 1

Case 1. Hess screens of 77-year-old woman with double vision (left), following left inferior rectus recession (middle), and following right inferior rectus recession (right).

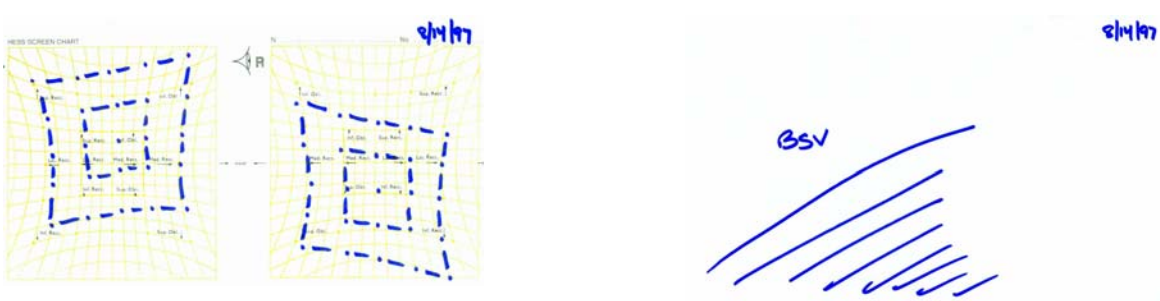


FIGURE 2

Case 1. Left, Hess screen following right inferior oblique extirpation. Right, Binocular single vision field following right inferior oblique extirpation demonstrating double vision on down right gaze, but single vision up left.

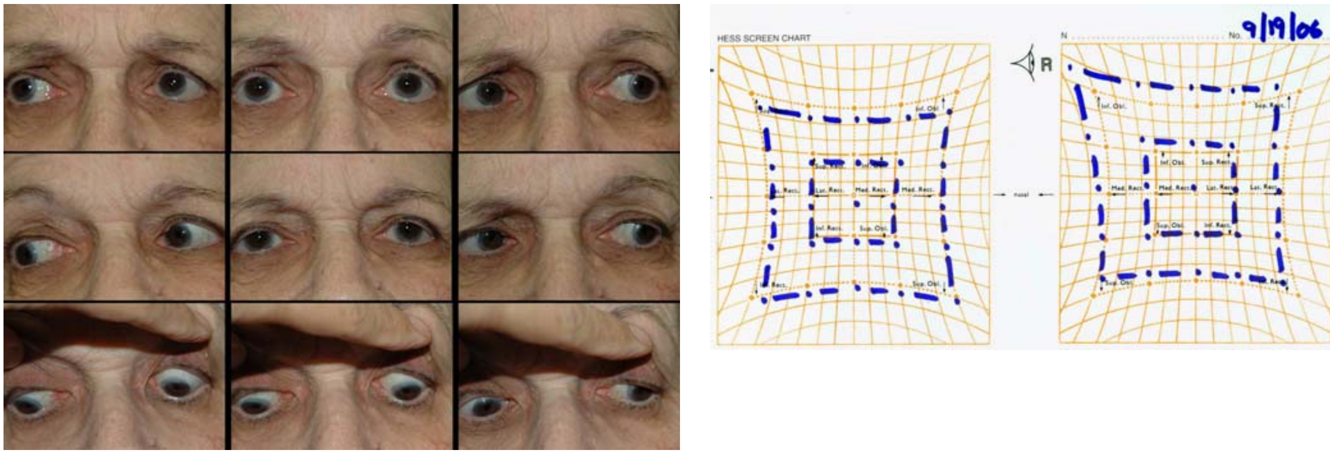


FIGURE 3

Case 1. Left, Nine cardinal positions following bilateral inferior oblique extirpation. Right, Hess screen demonstrating minimal residual right hypertropia. At that time the patient had no double vision.

Case 2

A 47-year-old woman was referred by a neurologist in January 1998 for complaints of double vision. Her previous medical history was remarkable for hyperthyroidism, diagnosed 2 years earlier and treated with radioactive iodine and thyroid hormone replacement. A CT scan confirmed bilateral thickening of the inferior rectus muscles. Visual acuity was correctible to 20/20 bilaterally with normal visual fields and Hertel measurements of 14.5 and 13.5 with mild lid retraction. She had limitation in elevation in abduction bilaterally (Figure 4, left) best seen on the Hess screen (Figure 4, middle). Although she had 40 seconds of stereopsis in primary position, binocular single vision fields revealed double vision 5° up and on right gaze (Figure 4, right). In June 1998 she underwent a left inferior oblique extirpation. Postoperatively she had a collapse of the overacting left inferior oblique with a reversal to a right hypertropia in up left gaze (Figure 5). By November 1998 she had a right hypertropia of 4 D in primary position, increasing on right gaze, and in November she underwent a right inferior oblique extirpation and denervation. She was left with 3 D of left hypertropia in primary position, which responded to prism correction in her glasses. At last follow-up, in June 2006, she had 40 seconds of stereopsis and noted double vision only when she was tired (Figure 6).

Comment. Gauging the amount of vertical obtained by inferior oblique surgery is difficult. With this patient, we were left with a small residual hyperdeviation, but by reducing the incomitance, we were able to correct this with residual prism. Although obviously retaining restriction in her inferior rectus, she was not bothered by head position abnormalities and had symptomatic relief of her diplopia.

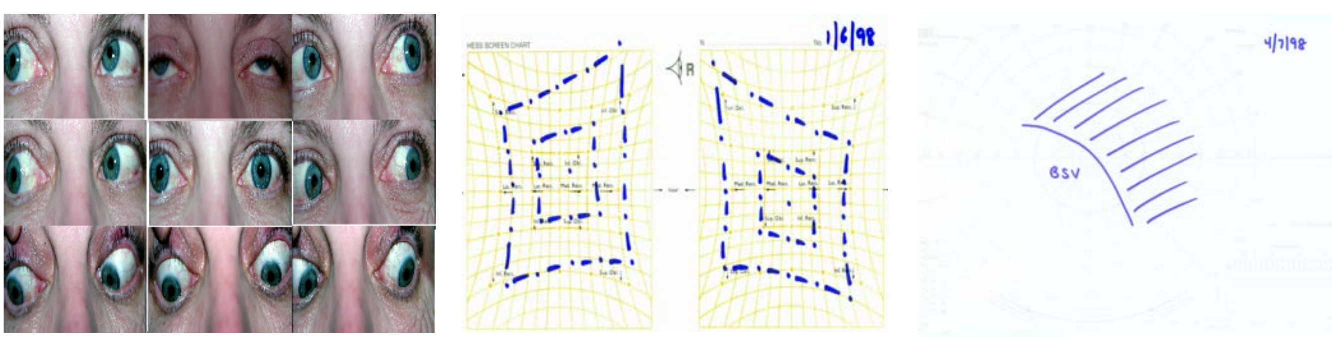


FIGURE 4

Case 2. Left, A 47-year-old patient with limitation in elevation bilaterally. Middle, Hess screen demonstrating secondary overaction of the inferior obliques due to restriction of the inferior rectus. Right, Binocular single vision fields done at the same time as the Hess screen illustrating single vision down and to the left and double vision up and to the right.

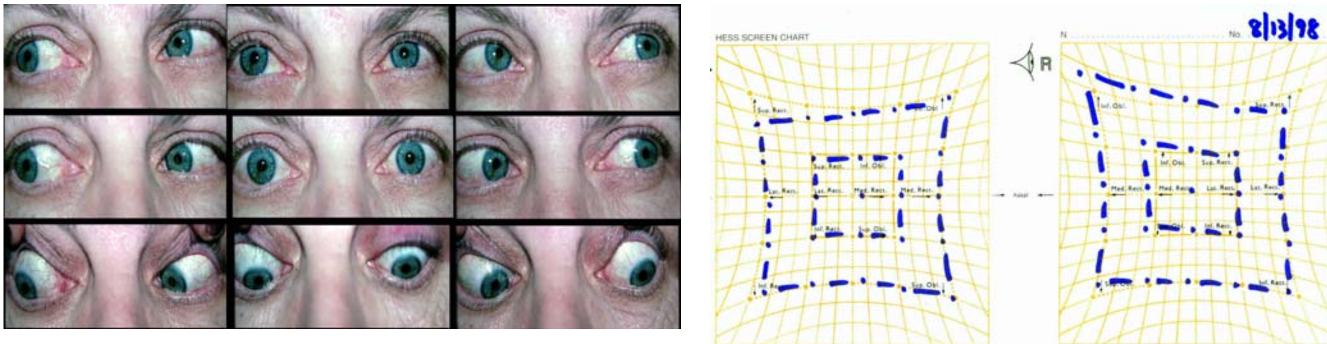


FIGURE 5

Case 2. Left, Nine cardinal positions following left inferior oblique extirpation. Right, Hess screen indicating residual right hypertropia on left gaze, but marked collapse of the previously noted left hypertropia on right gaze.

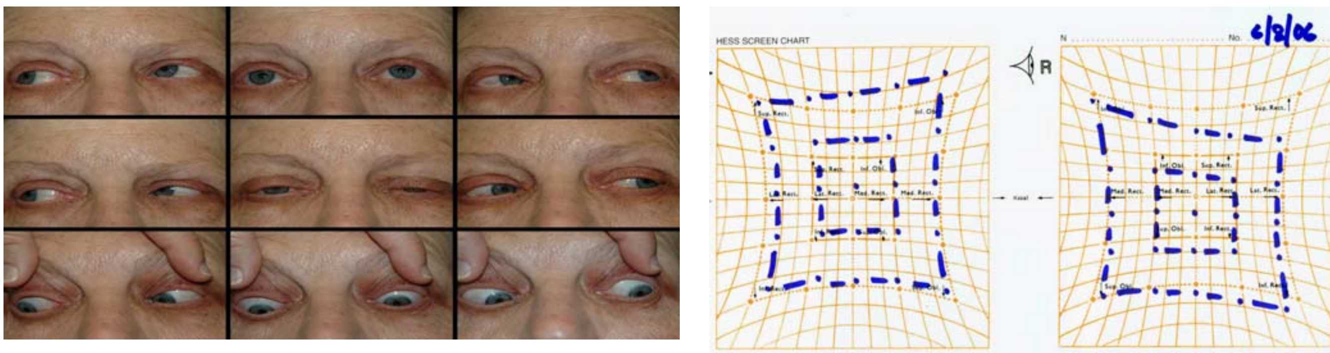


FIGURE 6

Case 2. Left, Nine cardinal positions following bilateral inferior oblique extirpation. At that time the patient was noting double vision only "when tired." Right, Hess screen illustrating persistent problems with elevation of the right eye in right gaze.

Case 3

A 69-year-old man was referred in August 2000 with a previous history of ocular hypertension and 4 months of progressive double vision. His afferent system was normal with 20/20 vision and normal visual fields. He had 12 D of left hypertropia in primary position with a left hypertropia increasing on up gaze (Figure 7, left) confirmed on Hess screen testing (Figure 7, middle). Binocular single vision fields revealed single vision on 35° down gaze (Figure 7, right). He did have 2 mm of proptosis on the right with mild

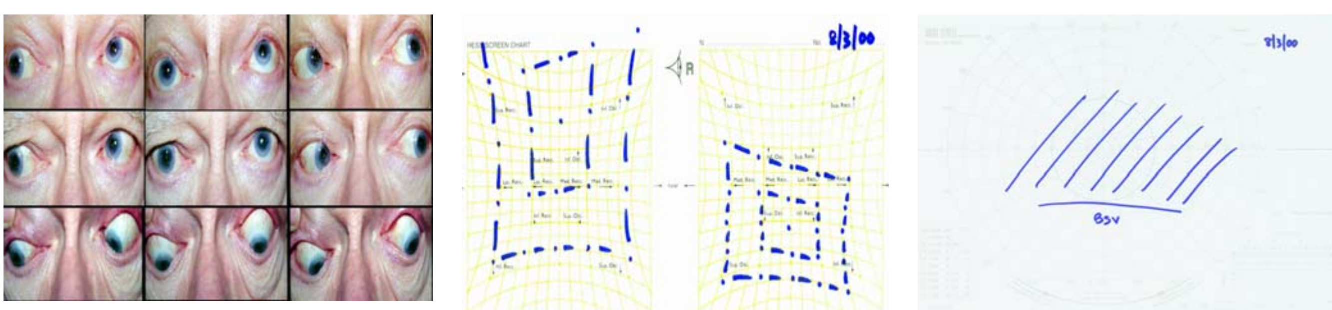


FIGURE 7

Case 3. Left, Nine cardinal positions in a 69-year-old man with limitation in elevation particularly of the right eye in right gaze. Middle, Hess screen illustrating marked limitation in elevation on the right side. Right, Binocular single vision fields done at the same time as the previous Hess screen demonstrate single vision but only 30° down.

resistance to retropulsion. Pneumotonometry tensions rose from 21 to 31 with attempted up gaze on the left and from 19 to 34 with attempted up gaze on the right. Subsequent thyroid function tests revealed a low TSH and elevated thyroid hormone levels. A CT scan confirmed enlargement of the inferior rectus muscles, right greater than left. On September 25, 2000, the patient underwent a left inferior oblique extirpation. Four-month follow-up revealed undercorrection, particularly in up right gaze (Figure 8, left and middle), but a marked improvement in binocular single vision fields, with double vision now only 12° above fixation (Figure 8, right). Because the patient was asymptomatic, no additional surgery was contemplated.

Comment. Although clearly this patient was undercorrected for his restrictive strabismus, by moving the area of binocularity and maximizing it through contralateral surgery, the patient was symptomatically improved. As had been pointed out by other investigators, the goal of surgery particularly in patients with significant paretic or restrictive strabismus is to maximize the area of binocularity, centering it on central fixation and down reading gaze.

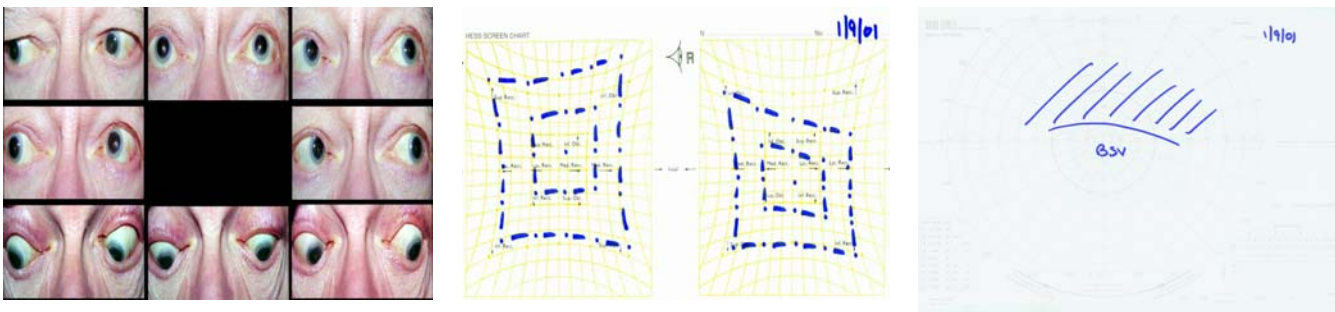


FIGURE 8

Case 3. Left, Nine cardinal positions following left inferior oblique extirpation showing similar limitation in elevation of the left eye in up right gaze. Middle, Hess screen illustrating persistent problems with elevation on the right, but decreased secondary overaction of the left inferior oblique following surgery. Right, Binocular single vision now reveals double vision only 10° above primary position.

Case 4

In June 1997 a 51-year-old woman was referred with a 3-year history of palpations and heat intolerance. She had been diagnosed with hyperthyroidism in August 1994 and treated with radioactive iodine and subsequent thyroid replacement. Nine months before her referral she began to notice double vision. On examination her afferent system was normal. Her motility examination, however, revealed restriction in elevation in abduction, worse on the left than the right, with 5 D of right hypertropia in primary position decreasing on down gaze (Figure 9). She was able to read with a chin-up position with normal stereopsis. Because of her minimal

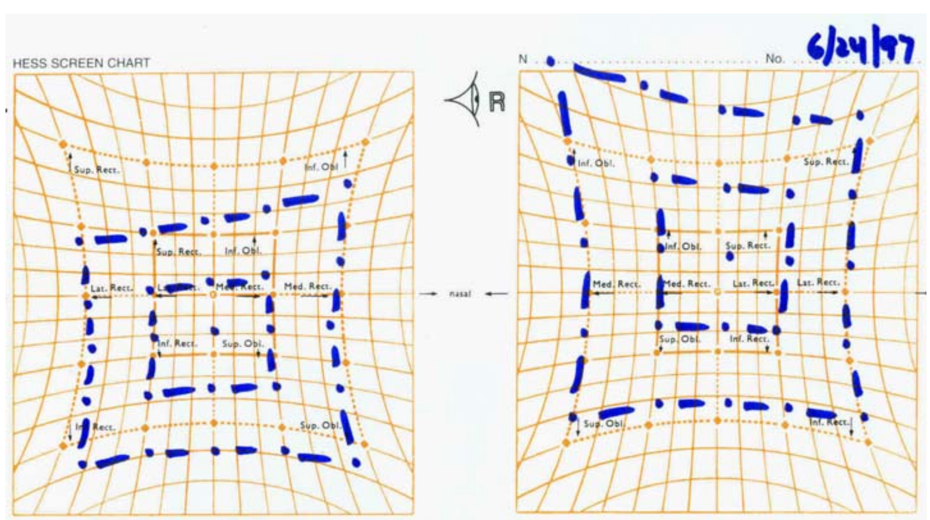


FIGURE 9

Case 4. Hess screen illustrating limitation in elevation of the left eye with secondary overaction of the right inferior oblique.

symptoms, no initial therapy was instituted, but by September 1998 her double vision had substantially worsened and she had 17 D of left hypotropia in primary position. It was felt that it would likely be necessary to do an inferior rectus recession on the left, but by collapsing the vertical with right inferior oblique weakening, it was hoped that the amount of recession would be less. On October 5, 1998, she underwent a right inferior oblique extirpation. This collapsed her vertical as seen in follow-up in April 1999, with about 12 D of residual right hypertropia (Figure 10, left and middle). Her binocular single vision fields revealed diplopia in down reading gaze (Figure 10, right). On July 12, 1999, she underwent a left inferior rectus recession on an adjustable stitch. This completely collapsed her vertical. She subsequently developed lid retraction and underwent a hard palate graft to the left lower lid in April 2000. At last follow-up in October 2006, she had no complaints of double vision and retained 40 seconds of stereopsis (Figure 11).

Comment. By limiting the elevation in the right eye, the amount of left inferior rectus recession could be reduced. This would seem to make subsequent overcorrection less likely, although certainly not impossible. Hopefully it will also limit the amount of lower lid retraction, although in this case lower lid retraction following inferior rectus recession did require a hard palate graft for corneal protection.

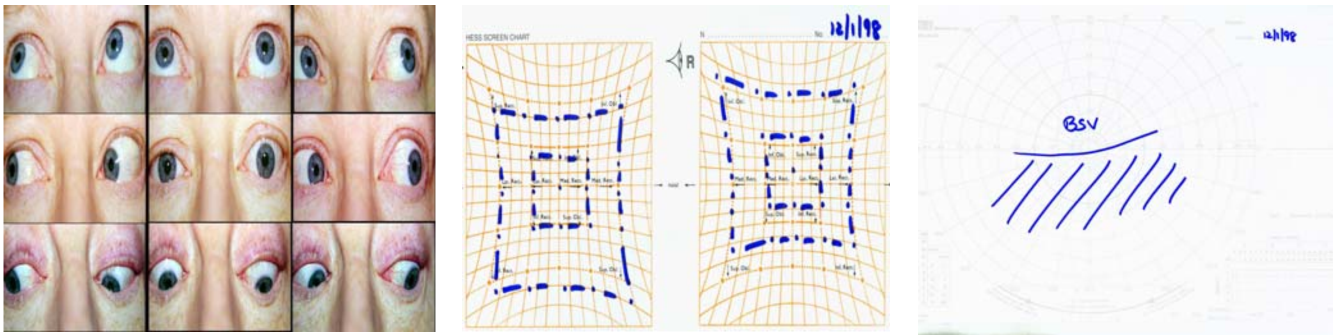


FIGURE 10

Case 4. Left, Nine cardinal positions following right inferior oblique extirpation resulting in similar limitation in elevation of the right eye to the left eye in attempt at up left gaze. Middle, Hess screen following right inferior oblique surgery reveals residual limitation in down gaze in the right eye. Right, Binocular single vision fields following right inferior oblique reveals single vision above fixation, but double vision below primary gaze.

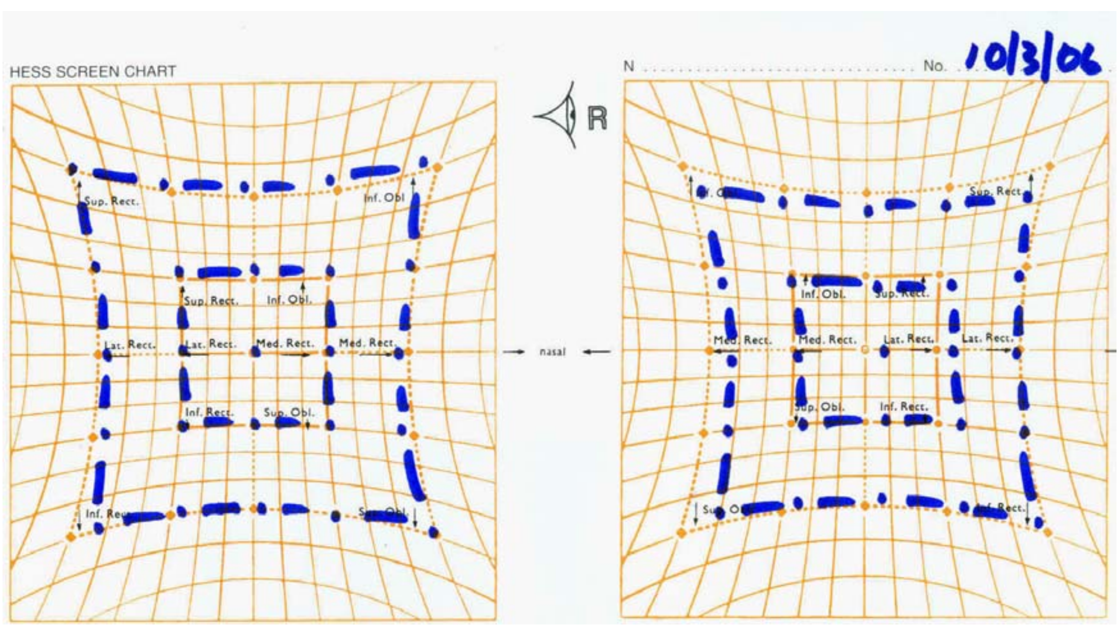


FIGURE 11

Case 4. Hess screen illustrating limitation in elevation of the left eye with secondary overaction of the right inferior oblique.

Case 5

A 63-year-old woman was referred for Graves disease in August 2003. She had a 7-year history of thyroid problems and a 4-month history of progressive proptosis and diplopia. Her afferent system was unremarkable. She did have moderate resistance to retropulsion, but no asymmetric proptosis, with Hertel measurements of 18 OU. She had mild limitation in abduction bilaterally but marked limitation in elevation, worse on the right than the left, and 18 D of right hypotropia with 8 D of exotropia in primary position (Figure 12). Pneumotonometry tensions demonstrated a 10-mm rise in intraocular pressure with attempted up gaze OD. It was felt that she would likely require inferior rectus surgery OD at some point, but she initially underwent a left inferior oblique extirpation, on October 6, 2003. This minimally collapsed her vertical deviation, and she had a persistent left hypertropia (Figure 13). Because the deviation was greater on down gaze, single-fiber EMG was done, and results were normal. A left superior rectus recession was performed with an adjustable stitch. Postoperatively the patient denied having any further double vision. At last follow-up in June 2005, she had minimal limitation in elevation, slightly worse on the left than the right, but normal stereopsis and no complaints of double vision (Figure 14).

Comment. One size does not fit all. The hyperdeviation worse on down gaze suggested more restriction in the superior rectus. Thus an ipsilateral superior rectus recession was done instead of a contralateral inferior rectus recession. This therapeutic decision was made because patients are far more symptomatic with overcorrection following inferior rectus surgery than superior.

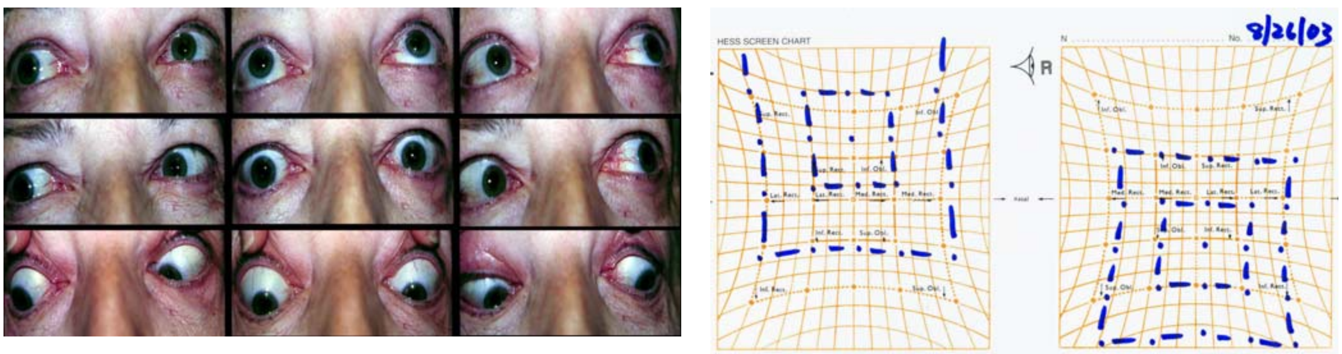


FIGURE 12

Case 5. Left, Nine cardinal positions of 63-year-old patient illustrating significant limitation in elevation particularly of the right eye in right gaze. Right, Hess screen done at the same time as the nine cardinal positions illustrates left hypertropia increasing on up gaze.

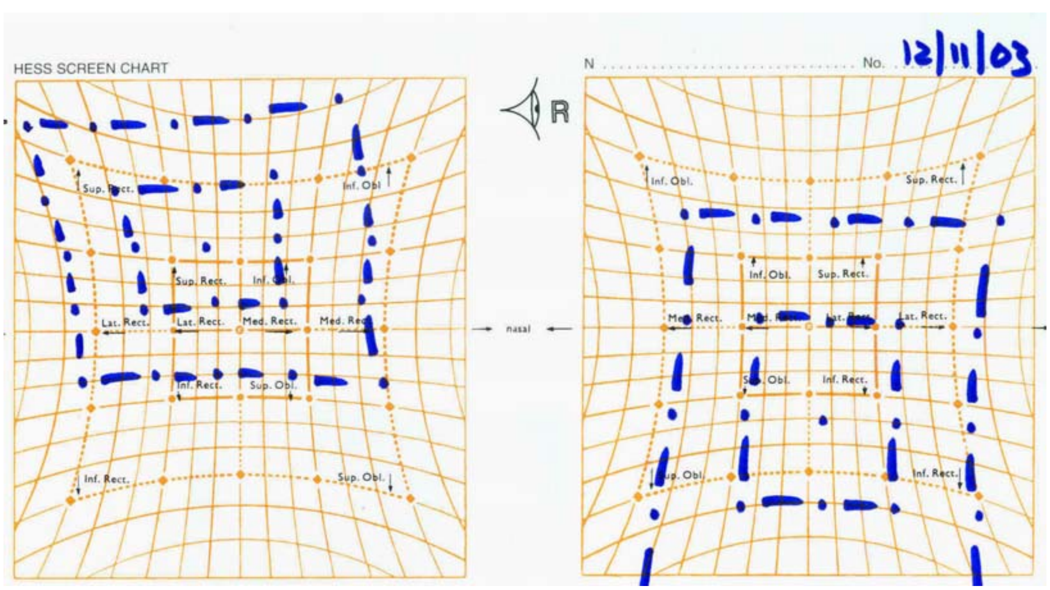


FIGURE 13

Case 5. Hess screen following left inferior oblique extirpation shows some limitation in overelevation of the left eye, but marked residual left hypertropia increasing on down gaze.

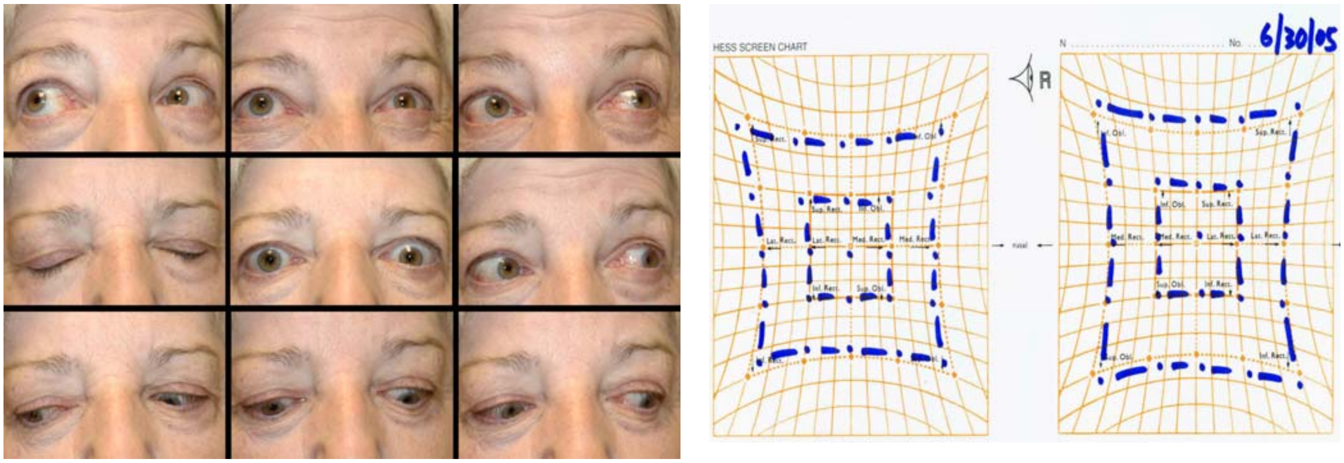


FIGURE 14

Case 5. Left, Nine cardinal positions following left superior rectus recession resulting in complete resolution of double vision. Right, Hess screen following left superior rectus recession shows minimal residual vertical limitation on the left.

Case 6

A 65-year-old woman was referred for a 5-month history of double vision and a 1-month history of diagnosed thyroid disease. Her afferent system was unremarkable with 20/20 and 20/25 vision without an afferent pupillary defect. She had limitation in elevation, OD worse than OS, with a left hypertropia increasing on up right gaze. She had 10 D of right hypotropia in primary position, but there was no deviation in down gaze (Figure 15). Thyrotropin inhibitor binding assay was abnormal at 87% (normal <10%). By 1 month follow-up, her hypodeviation had increased to 20 D with worsening problems with elevation. In August 2003 she underwent a left inferior oblique extirpation. This collapsed the hypertropia in up gaze, but left her with a significant left hypertropia deviation in down right gaze (Figure 16). On November 3, 2003, the patient underwent a left superior rectus recession with some residual double vision in up gaze (Figure 17). She was bothered by persistent double vision when looking up and to the right side, and on April 24, 2006, she underwent a right superior rectus recession on adjustable stitch. Initially, she was only slightly overcorrected, but by 3 weeks later her consecutive left hyperdeviation had increased to 20 D (Figure 18). When this failed to clear, she underwent a right inferior rectus recession on adjustable stitch in July 2006. Immediately postoperatively the muscle slipped (Figure 19), requiring reoperation 2 days later. This resolved her diplopia. With 2-year follow-up she has had no recurrent double vision (Figure 20).

Comment. This case demonstrates that the inferior oblique surgery will have only a limited effect on the amount of vertical. It also illustrates the potential for overcorrection, particularly with adjustable stitches, and the potential for slippage of a recessed muscle. Persistence here, in collapsing the better-moving eye, eventually resulted in complete resolution of the patient's diplopia.

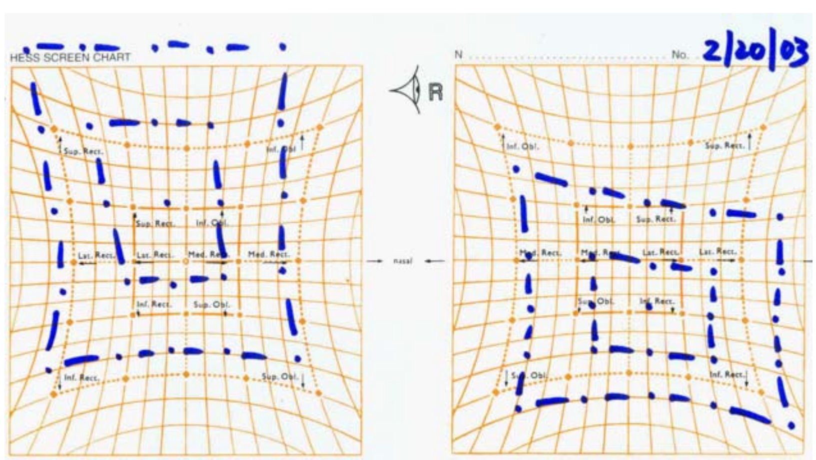


FIGURE 15

Case 6. Hess screen of a 65-year-old woman with a 5-month history of double vision demonstrating a left hypertropia increasing on up right gaze secondary to restriction of the right inferior rectus.

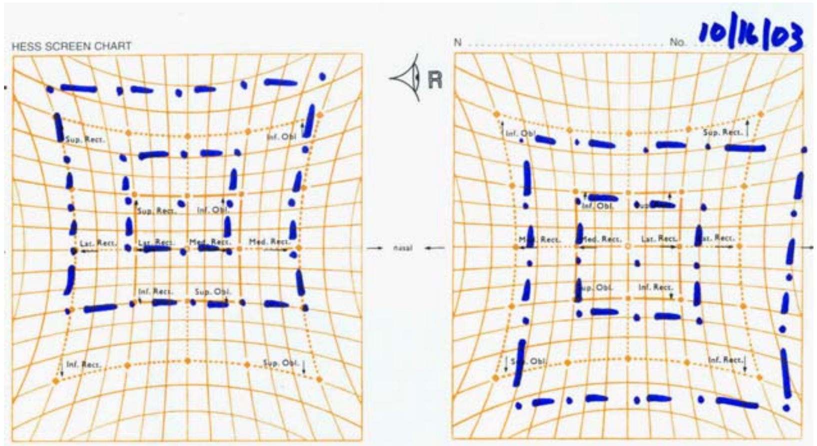


FIGURE 16

Case 6. Hess screen following left inferior oblique extirpation shows reduction of the overacting inferior oblique on the left side with persistent left hyperdeviation.

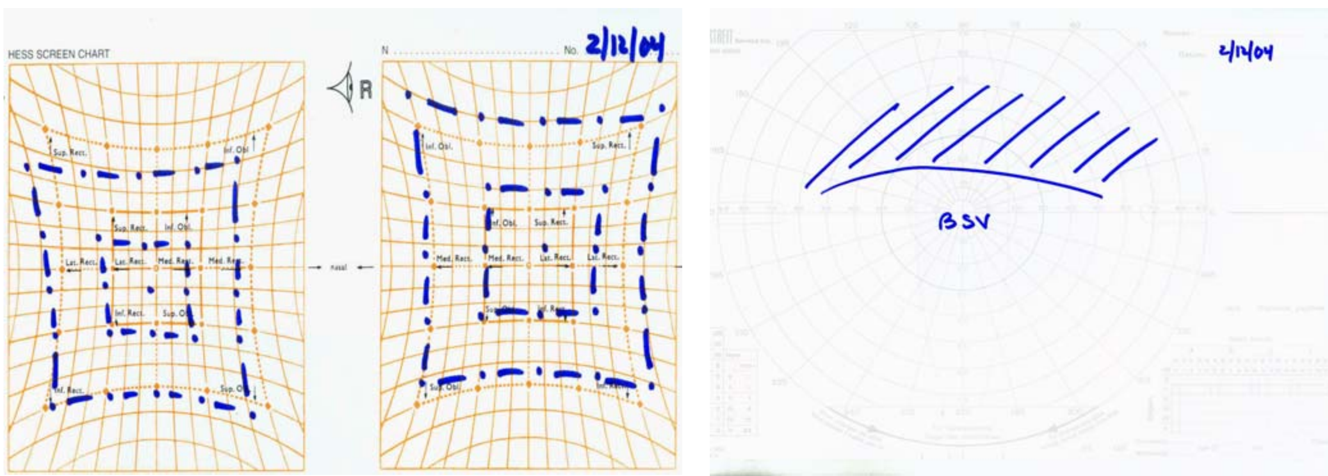


FIGURE 17

Case 6. Left, Hess screen following left superior rectus recession shows slight overcorrection now with a right hypertropia on up gaze. Right, Binocular single vision fields done at the same time show binocular single vision in primary position and down reading gaze but double vision 18° above primary gaze.

Case 7

A 34-year-old man was referred for “severe Graves disease” in June 2004. He had a 4-year history of rapid heart rate and weight loss, which led to the diagnosis of hyperthyroidism. He was treated with radioactive iodine twice in 2001 and subsequently started thyroid replacement therapy. He had a 1-month history of double vision. On examination, the afferent system was entirely normal with 20/20 acuity at distance and 3-point vision at near. Proptosis was 28 mm and 31 mm, with marked resistance to retropulsion and severe lid retraction with inferior scleral show (Figure 21, left). He also had mild lagophthalmos bilaterally. He had severe limitation in elevation bilaterally with an exodeviation decreasing on gaze to the right and with a left hypotropia greater on up gaze, measuring 20 D by Maddox rod (Figure 21, right). Because of the severe proptosis and mild exposure problems, he underwent bilateral orbital decompressive surgery through a transantral approach on October 11, 2004. Postoperatively he had 40 D of left hypotropia with 8 D of esotropia (Figure 22). To limit the amount of inferior rectus recession necessary, he first underwent a right inferior oblique extirpation and denervation on January 19, 2005. This minimally collapsed his vertical deviation but left severe residual limitation in elevation measuring 8 D of left hypertropia in primary position (Figure 23). On June 13, 2005, he underwent bilateral inferior rectus recessions on adjustable stitches. Postoperatively he had a slight consecutive right hyperdeviation. By 4-month follow-up he did note residual double vision when looking to the left and right as well as up (Figure 24, left and middle). He had 7 mm of reduction in proptosis following orbital surgery but still demonstrated marked limitation in abduction with severe limitation in elevation bilaterally. He was; however, able to fuse in primary position and in down reading gaze with 40 seconds of stereopsis (Figure 24, right).

Comment. With severe restriction exacerbated by orbital decompressive surgery, it is not surprising that we were unable to completely eliminate the vertical incomitance across the horizontal. With a combination of inferior oblique and inferior rectus surgery, however, we were able to maintain binocularity in primary position and down reading gaze.

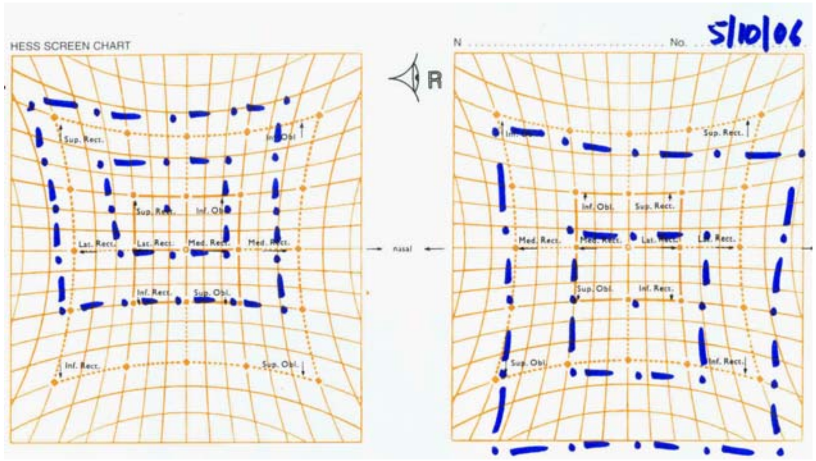


FIGURE 18

Case 6. Recurrent left hyperdeviation following presumed slippage of right superior rectus suture resulting in increasing double vision on down gaze.

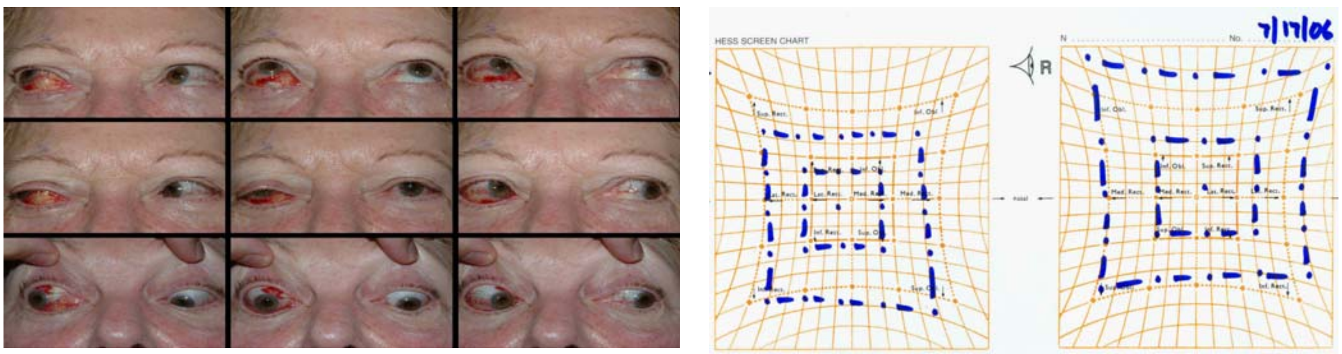


FIGURE 19

Case 6. Left, Nine cardinal positions immediately following right inferior rectus recession with slippage of the muscle. Right, Hess screen demonstrates a slight right hypertropia increasing on down gaze.

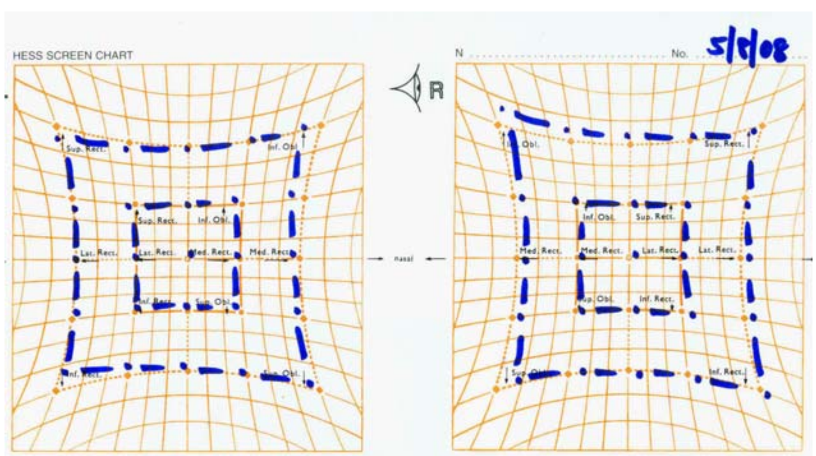


FIGURE 20

Case 6. Hess screen following reoperation with advancement of the slipped right inferior rectus. At that time the patient had no recurrent or residual problems with double vision.

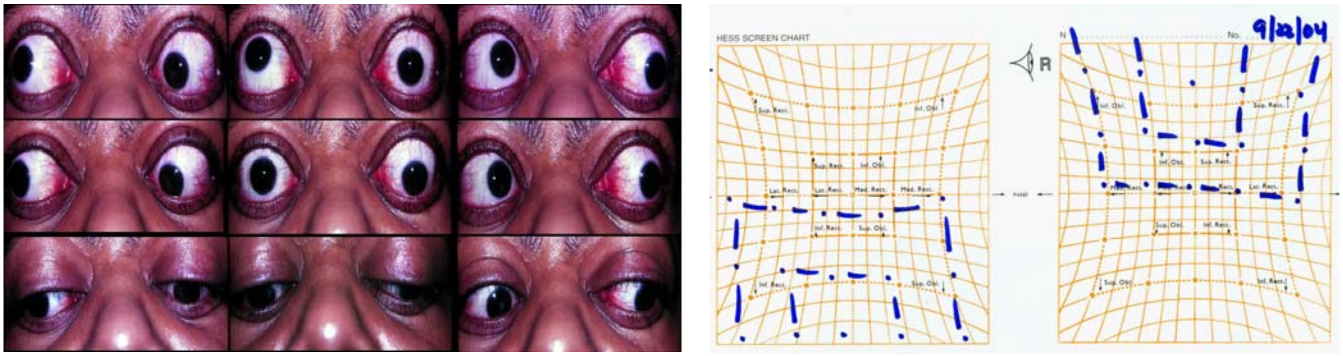


FIGURE 21

Case 7. Left, Nine cardinal positions of a 34-year-old patient with severe proptosis and limitation in elevation on the left. Right, Hess screen done at the time of nine cardinal positions demonstrates a marked left hypotropia.

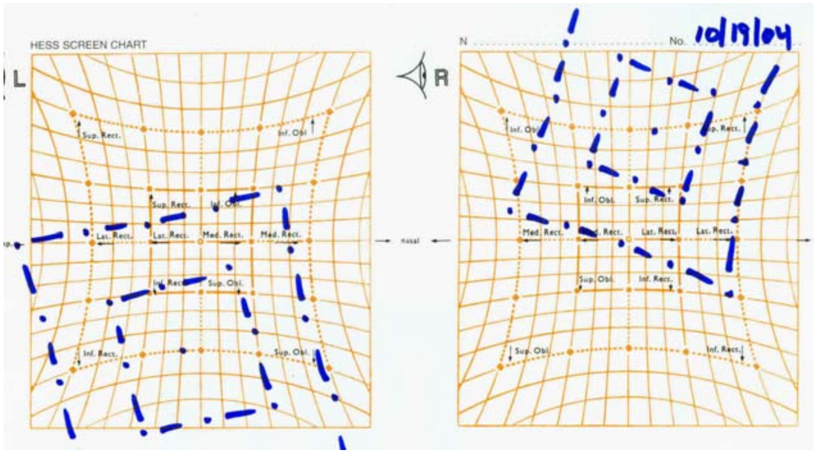


FIGURE 22

Case 7. Hess screen following bilateral orbital decompressive surgery with an increase in the relative left hypotropia.

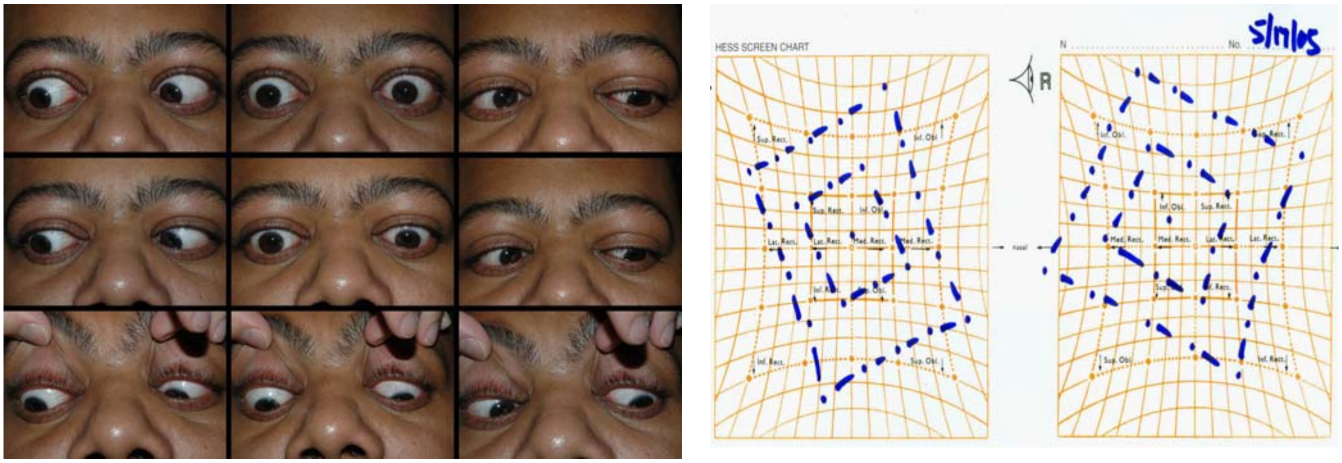


FIGURE 23

Case 7. Left, Nine cardinal positions following right inferior oblique extirpation. Right, Hess screen following right inferior oblique extirpation demonstrates persistent limitation in elevation and abduction.

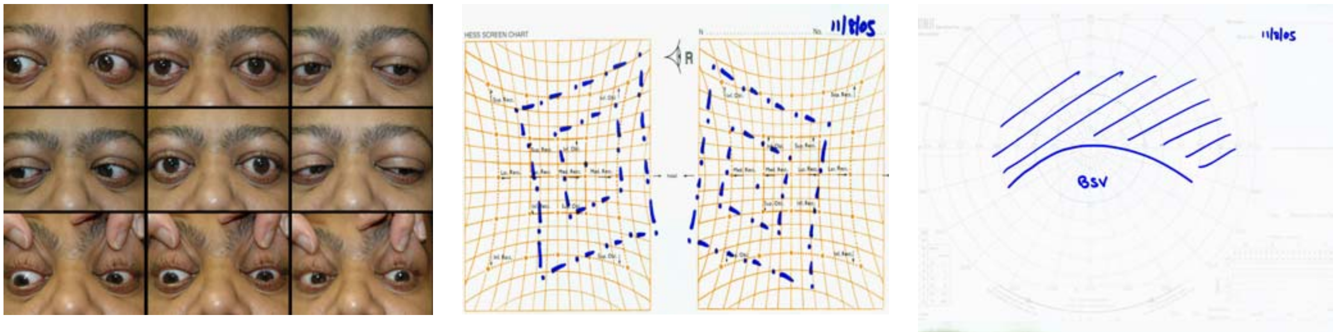


FIGURE 24

Case 7. Left, Nine cardinal positions following bilateral inferior rectus recessions with some minimal residual double vision when looking up and to either side. Middle, Hess screen done following bilateral inferior rectus recessions still shows effective overaction of the inferior obliques in spite of previous right inferior oblique extirpation. Right, Binocular single vision fields demonstrate single vision in primary position and in down reading gaze with persistent double vision up and to either side.

DISCUSSION

Muscle surgery in patients with Graves disease has been recognized as challenging for years.[45] One difficulty has been quantifying the motility abnormalities. Even the newer classification of Graves (VISA classification[46]) grades diplopia into categories of none, diplopia on horizontal or vertical gaze, intermittent diplopia in straight gaze, and constant diplopia in straight gaze. Strabismus surgeons have argued for maximizing the field of binocular single vision centered on primary and down reading gaze while eliminating abnormal head position. This may be best studied with the use of binocular single vision fields.[47-49] This has been specifically applied to patients with thyroid orbitopathy.[50] Failure to examine the patients in all fields often leads to better than expected results.[51] Ductions of each eye may be quantitatively assessed with an arc perimeter,[52] Goldmann bowl, or cervical range-of-motion apparatus.[53] Muscle surgery that aims to equalize individual ductions of the two eyes is likely to minimize residual diplopia.

Preceding orbital surgery has been recognized to exacerbate problems with diplopia.[4,54] Although the orbit may be decompressed by removing any of the four walls, surgery on the medial wall and floor seems to be most effective for relieving proptosis, ameliorating the compressive optic neuropathy, and reducing exposure keratopathy. As described by Walsh and Ogura,[55,56] and popularized at the Mayo Clinic, a transantral approach is quite effective. It also, however, is associated with the highest incidence of altering the muscle balance and producing or exacerbating diplopia.[4] This may be of particular concern when decompressive surgery is being done for cosmesis, as in the 1994 study by Fatourcehi,[57] in which 73% developed diplopia. Some investigators suggest that this induced diplopia may spontaneously improve (137 of 206 patients[58]), but it is unclear just how much detail was involved in this series follow-up. Other forms of orbital decompression, including a translid and coronal approach, have been reported to have a lower incidence of induced diplopia.[59] Other alterations in technique have been reported to decrease induced diplopia, including balanced lateral or keyhole decompression,[60-62] deep lateral wall decompression,[63] endoscopic decompression,[64] and transconjunctival fat decompression,[65] but none of these has been successful in eliminating it. Other investigators have argued that previous treatment, including decompressive surgery, does not influence the outcome of strabismus surgery.[66-68] Twelve of our 38 patients had preceding transantral decompressive procedures, and two others had orbital surgery following their initial muscle procedure.

Consecutive hypertropia following inferior rectus muscle surgery is common. Previous studies have suggested an incidence of between 17% and 45%.[26,33,35,42,45,66,69-74] More recent studies have suggested better results,[75] but detailed analysis removing the patients with only horizontal deviations suggests that the true incidence (4 in 15 [27%] if only the patients treated with inferior rectus or inferior and superior rectus surgery are included) is similar to previous reports. Inferior rectus surgery for other forms of restrictive strabismus also has a significant incidence of overcorrection.[76] Patients with larger amounts of proptosis and increased superior rectus muscle volume were found to have a higher incidence of late overcorrection.[77] Whereas arguments for this high frequency have emphasized the potential for muscle slippage and unmasking contralateral restriction, an additional component is that the involved muscles are not only restricted but also paretic (with reduced range of motion). To avoid overcorrection, some investigators have advocated intentionally undercorrecting the vertical strabismus.[73,78] Others advocate simultaneous bilateral inferior rectus recessions.[79]

Contralateral surgery has been previously recommended for restrictive strabismus. This usually involves the opposite superior rectus.[36] Small hypertropias may be addressed with a graded tenotomy.[80] Most investigators opt for recessions with or without adjustable sutures. Further limitation may be achieved with a posterior fixation suture. While surgery on the contralateral superior rectus is likely to have a greater effect than the contralateral inferior oblique, the maximal effect of the superior rectus recession will be when looking to the contralateral side instead of the ipsilateral, thus potentially exacerbating an incomitant deviation. There have been very few reports of prior inferior oblique surgery, most in the setting of multiple other muscle procedures.[81]

Two of our patients required subsequent orbital decompression for optic neuropathy. Ideally, orbital surgery will precede strabismus surgery, but uncommon late worsening of orbital apical compression may make it necessary. Our incidence of 5%

compares favorably with other series, where up to 28% required secondary orbital procedures.[82]

It is not surprising that the possibility of torsion in patients with thyroid orbitopathy has been explored.[81,83,84] In the series from the Mayo Clinic,[83] 21 patients were noted to have incyclotorsion and 5 excyclotorsion. All patients had previously undergone inferior rectus recessions. We did not routinely measure torsion in our patients, but none had torsional complaints. Inferior oblique extirpation may theoretically produce a component of incyclotorsion but likely less than recessing a tight inferior rectus.

Our ultimate results are very good, similar to those of previous series.[1,34,85,86] While success rates range between 43% and 100%,[87] the definition of success remains elusive. A recent debate suggests that operating for the restriction produces better results than operating for the deviation.[32,33] This has been challenged.[34] Ultimately, both need to be addressed while bearing in mind that restriction is unlikely to be isolated, and release of restriction will not restore normal excursion. The addition of inferior oblique surgery does not prevent overcorrection, but it is extremely unlikely. Undercorrection is common. When done alone as a staged procedure, this automatically increases the total number of operations.

The combination of inferior oblique surgery monitored with Hess screen and binocular single vision field testing appears to increase in binocularity. The major disadvantage in this series is the predetermined need for more than one operation in the majority of cases (79% of patients required at least a second procedure). The rate of second operations in strabismus surgery in thyroid patients has been between 17% and 45%.[82] The use of a traction suture with large recessions has been reported to be successful in 74% of cases in spite of a large preoperative deviation.[75] This “one stop” shopping certainly will be appealing. Only more detailed analysis and prospective studies will indicate whether the same degree of binocularity is ultimately achieved.

Our one patient subsequently diagnosed as having myasthenia raises the important coexistence of these two autoimmune diseases.[88] This may be present in 10% or more of patients and may produce an additional reason for malalignment. Diagnostic suspicion must remain high, especially with variable deviation.

Because of the unlikelihood of overcorrection with inferior oblique surgery alone, we feel that oblique surgery may be done earlier without waiting the traditional 4 to 6 months for stability. Interestingly, early surgery was first suggested to prevent late fibrosis by decreasing tension on the muscle.[89] Unfortunately, there is no evidence to support this hypothesis. While others have suggested that earlier surgery is possible,[82,90] intervention during the acute inflammatory phase may have a higher incidence of complications,[91] and progressive alteration in alignment is not unusual. In the series of Yang and associates,[82] 7 patients (28%) required subsequent orbital surgery. Progressive active disease may also be responsible for late undercorrection in patients initially well aligned following inferior rectus surgery.[92]

In addition, attention has been directed to possible excess inflammatory reaction in operating during the active phase of thyroid orbitopathy.[93] Attempts have been made to assess the activity of inflammation using B-scan[94] or MRI.[95-100] It has also been postulated that the muscle size will correlate with the results of strabismus surgery.[101] At this point it is unlikely that any test will guarantee stability and predict surgical outcome. Muscle surgery should be tailored to decrease the chance that late shifts will be in primary position and down reading gaze.

Rectus surgery has additional secondary effects. Some are beneficial, such as lowering intraocular pressure, and others less ideal, such as producing an increase in proptosis[102] and late lid retraction, which can exacerbate exposure problems.[86,103] Various modifications of surgical techniques have been introduced to minimize lower lid retraction,[104-106] but it is unlikely it can be completely prevented. While some investigators have suggested simultaneous lid and muscle surgery, this should not be entertained while operating on the vertical muscles.[107]

CONCLUSION

When dealing with thyroid patients with diplopia, it is critical to manage expectations. In 1971 John Dyer, wrote, “Since the muscles are no longer normal, the patient cannot expect to have a full range of binocular vision once this eye is operated on, and it is extremely important to impress on these patients that what we hope to gain is single binocular vision in a reasonable area in the primary position and in the reading field. I suggest that the Lancaster red-green test and the Hess screen are extremely valuable tools to use preoperatively as well as postoperatively.”[108] Unfortunately, even with control of the active inflammatory component (including cessation of smoking, steroid therapy, control of thyroid function, and potentially other immune modulators), residual fibrosis persistently limits ductions manifesting as restrictive strabismus. While the primary muscle procedure should be aimed at releasing restriction, it is imperative to keep in mind that release of restriction does not improve motility. It is likely that the worse-moving eye will continue to be more limited even if alignment is achieved in primary gaze, and thus early attention should be directed to the contralateral antagonist. Since the effect of inferior oblique surgery is hard to predict, an argument can be made for considering this as a first stage. Inferior oblique surgery tends to collapse the vertical deviation (especially across the horizontal). This permits less surgery on the vertical rectus muscle, which can be graded with adjustable sutures. By matching the excursion of the two eyes, the area of binocularity may be maximized. This has clearly been recognized in patients with long-standing paretic deviations. This study supports the notion that this is also an important concept in dealing with restrictive strabismus.

ACKNOWLEDGMENTS

Funding/Support: None
Financial Disclosures: None
Conformity With Author Information: This study was approved by the institutional review board of the University of Virginia.

REFERENCES

1. Bok C, Hidalgo C, Morax S. Surgical management of diplopia in dysthyroid orbitopathy. *J Fr Ophtamol* 2007;30:390-396.
2. Chu YK, Kim SJ, Lee SY. Surgical treatment modalities of thyroid ophthalmopathy. *Korean J Ophthalmol* 2001;15:128-132.
3. Looi ALG, Luu CD, Wong TY, Seah LL, Rootman J. Factors associated with decompression and strabismus surgery in thyroid eye disease. *Ann Acad Med Singapore* 2005;34:154-157.
4. Garrity JA, Fatourechi V, Bergstralh V, et al. Results of transantral orbital decompression in 428 patients with severe Graves' orbitopathy. *Am J Ophthalmol* 1993;116:533-547.
5. Werner SC, Coleman DJ, Franzen LA. Ultrasonographic evidence of a consistent orbital involvement in Graves' disease. *N Engl J Med* 1974;290:1447-1450.
6. Saber E, McDonnell J, Zimmerman, KM, Yugar JE, Feldon SE. Extraocular muscle changes in experimental orbital venous stasis: some similarities to Graves' orbitopathy. *Graefes Arch Clin Exp Ophthalmol* 1996;234:331-336.
7. Metz HS. Saccadic velocity studies in patients with endocrine ocular disease. *Am J Ophthalmol* 1977;84:695-699.
8. Helveston EM, Bick SE, Ellis FD. Differential intraocular pressure as an indirect measure of generated muscle force. *Ophthalmic Surg*1980;11;386-391.
9. Allen C, Stetz D, Roman SH, et al. Prevalance of increased intraocular pressure changes in Graves' disease. *J Clin Endocrinol Metab* 1985;61:183-187.
10. Spierer A, Eisenstein Z. The role of increased intraocular pressure on up gaze in the assessment of Graves ophthalmopathy. *Ophthalmology* 1991;98:1491-1494.
11. Gamblin GT, Harper DG, Galentine P, et al. Prevalence of increased intraocular pressure in Graves'disease: evidence of frequent subclinical ophthalmology. *N Engl J Med* 1993;308:420-424.
12. Scott AB. Injection treatment of endocrine orbital myopathy. *Doc Ophthalmol* 1984;58:141-145.
13. Dunn WJ, Arnold AC, O'Conner PS. Botulinum toxin for the treatment of dysthyroid ocular myopathy. *Ophthalmology* 1986;93:470-475.
14. Kikkawa DO, Cruz RC Jr, Christian WK, et al. Botulinum A toxin injection for restrictive myopathy of thyroid-related orbitopathy: effects on intraocular pressure. *Am J Ophthalmol* 2003;135:427-431.
15. Nagayama Y, Izumi M, Kiriyama T, et al. Treatment of Graves' ophthalmopathy with high dose intravenous methylprednisolone pulse therapy. *Acta Endocrinol* 1987;116:513-518.
16. Ohtsuka K, Sato A, Kawaguchi S, Hashimoto M, Suzuki Y. Effect of high dose intravenous steroid pulse therapy followed by 3 month oral therapy for Graves' ophthalmopathy. *Jpn J Ophthalmol* 2002;46:563-567.
17. Kaippinen-Makelin R, Karma A, Leinonen E, et al. High dose intravenous methylprednisolone pulse therapy versus oral prednisone for thyroid-associated ophthalmopathy. *Acta Ophthalmol Scand* 2002;80:316-321.
18. Meyer PAR. Avoiding surgery for thyroid eye disease. *Eye* 2006;20:1171-1177.
19. Wilson WB, Prochoda M. Radiotherapy for thyroid orbitopathy. Effects on extraocular muscle balance. *Arch Ophthalmol* 1995;113:1420-1425.
20. Mourits MP, van Kampen-Harteveld ML, Garcia ML, Koppescharr HP, Tick L, Terwee CB. Radiotheraphy for Graves' orbitopathy: randomised placebo-controlled study. *Lancet* 2000;355:1505-1509.
21. Kazim M. Radiotherapy for Graves orbitopathy: the Columbia University experience. *Ophthal Plast Reconstr Surg* 2002;18:173-174.
22. Gorman CA, Garrity JA, Fatourechi V, et al. A prospective randomized, double-blinded, placebo-controlled study of orbital radiotherapy for Graves' ophthalmopathy. *Ophthalmology* 2001;108:1523-1534.
23. Bartley GB, Gorman CA. The Mayo orbital radiotherapy for Graves' ophthalmopathy (ORGO) study: lessons learned. *Ophthal Plast Reconstr Surg* 2002;18:170-172.
24. Ferris JD, Dawson EL, Plowman N, Adams GG, Fells P, Lee JP. Radiotherapy in thyroid eye disease: the effect on the field of binocular single vision. *J AAPOS* 2002;62:71-76.
25. Schotthoefer EO, Wallace DK. Strabismus associated with thyroid eye disease. *Curr Opin Ophthalmol* 2007;18:361-365.
26. Evans D, Kennerdell JS. Extraocular muscle surgery for dysthyroid myopathy. *Am J Ophthalmol* 1983;95:767-771.
27. Scott WE, Martin-Casals A, Jackson OB. Adjustable sutures in strabismus surgery. *J Pediatr Ophthalmol* 1977;14:71-75.
28. Jampolsky A. Current techniques of adjustable strabismus surgery. *Am J Ophthalmol* 1979;88:406-418.
29. Lueder GT, Scott WE, Kutschke PJ, Keech RV. Long-term results of adjustable suture surgery for strabismus secondary to thyroid ophthalmopathy. *Ophthalmology* 1992;99:993-997.
30. Kraus DJ, Bullock JD. Treatment of thyroid ocular myopathy with adjustable and nonadjustable suture strabismus surgery. *Trans Am Ophthalmol Soc* 1993;91:67-79.
31. Kose S, Uretmen O, Emre S, Pamukcu K. Recession of the inferior rectus muscle under topical anesthesia in thyroid ophthalmopathy. *J Pediatr Ophthalmol Strabismus* 2000;39:331-335.
32. Dal Canto AJ, Crowe S, Perry JD, Traboulsi EI. Intraoperative relaxed muscle positioning technique for strabismus repair in thyroid eye disease. *Ophthalmology* 2006;113:2324-2330.
33. Nguyen VT, Park DJ, Levin L, Feldon SE. Correction of restricted extraocular muscle motility in surgical management of strabismus in Graves' ophthalmopathy. *Ophthalmology* 2002;109:384-388.
34. Thomas SM, Cruz OA. Comparison of two different surgical techniques for the treatment of strabismus in dysthyroid ophthalmopathy. *J AAPOS* 2007;11:258-261.

35. Prendiville P, Chopra M, Gauderman WJ, Feldon SE. The role of restricted motility in determining outcomes for vertical strabismus surgery in Graves' ophthalmopathy. *Ophthalmology* 2000;107:545-549.
36. Buckley EG. Restrictive strabismus. In: Plager DA, ed. *Strabismus Surgery–Basic and Advanced Strategies.* Hong Kong: Oxford University Press; 2004:117-153.
37. Schittkowski M, Fichter N, Guthoff R. Strabismus surgery in Graves' disease–dose-effect relationships and functional results. *Klin Montasbl Augenheilkd* 2004;221:941-947.
38. Eckstein A, Schulz S, Esser J. Is combined surgical correction of horizontal and vertical squint of value in Graves' ophthalmopathy? *Klin Monatsbl Augenheilkd* 2004;221:769-775.
39. Wright KW. Late overcorrection after inferior rectus recession. *Ophthalmology* 1996;103:1503-1507.
40. Ludwig I, Chow A. Scar remodeling after strabismus surgery. *J Pediatr Ophthalmol Strabismus* 2000;4:326-333.
41. Buckley EG. Paralytic strabismus. In: Plager DA, ed. *Strabismus Surgery–Basic and Advanced Strategies.* Hong Kong: Oxford University Press; 2004:83-117.
42. Dyer JA. Ocular muscle surgery in Graves' disease. *Trans Am Ophthalmol Soc* 1978;76:125-139.
43. Newman S, Gonzalez-Fernandez F. Thyroid orbitopathy—oblique muscle involvement. *Invest Ophthalmol Vis Sci* 2002;43: E-Abstract 1462.
44. Kakizaki H, Zako M, Iwaki M. Thyroid-associated inferior oblique myopathy. *Ophthalmology* 2007;114:2106.
45. Sugar HS. Management of eye movement restriction (particularly vertical) in dysthyroid myopathy. *Ann Ophthalmol* 1979;11:1305-1318.
46. Dolman PJ, Rootman J. VISA classification for Graves orbitopathy. *Ophthal Plast Reconstr Surg* 2006;22:319-324.
47. Woodruff G, O'Reilly C, Kraft SP. Functional scoring of the field of binocular single vision in patients with diplopia. *Ophthalmology* 1987;94:1554-1561.
48. Fitzsimmons R, Fraco JW. Functional scoring of the field of binocular single vision. *Ophthalmology* 1990;97:33-35.
49. Sullivan TJ, Kraft SP, Burack C, O'Reilly C. A functional scoring method for the field of binocular single vision. *Ophthalmology* 1992;99:575-581.
50. Haggerty H, Richardson S, Mitchell K, Dickinson AJ. A modified method for measuring uniocular fields of fixation. *Arch Ophthalmol* 2005;123:356-362.
51. Cruz O, Roper-Hall G, Feldon S. Relaxed muscle positioning technique. *Ophthalmology* 2007;114:2104-2105.
52. Mourits MP, Prummel MF, Wiersinga WM, Koornneef L. Measuring eye movements in Graves ophthalmopathy. *Ophthalmology* 1994;101:1341-1345.
53. Kupersmith MJ, Fazzone HE. Comparing ocular muscle limitation tests for clinical trial use. *Arch Opthalmol* 2004;122:347-348.
54. Shorr N, Neuhas RW, Baylis HI. Ocular motility problems after orbital decompression for dysthyroid ophthalmopathy. *Ophthalmology* 1982;89:323-328.
55. Walsh TE, Ogura JH. Transantral orbital decompression for malignant exophthalmos. *Laryngoscope* 1957;67:544-549.
56. Ogura JH. Surgical results of orbital decompression for malignant exophthalmos. *J Laryngol Otol* 1978;92:181-195.
57. Fatourcehi V, Garrity JA, Bartley GB, et al. Graves ophthalmopathy. Results of transantral orbital decompression performed primarily for cosmetic indications. *Ophthalmology* 1994;101:938-942.
58. Warren JD, Spector JG, Burde R. Long-term follow-up and recent observations on 305 cases of orbital decompression for dysthyroid orbitopathy. *Laryngoscope* 1989;99:35-40.
59. Paridaens D, Hans K, van Buitenen S, Mourits MP. The incidence of diplopia following coronal and translid orbital decompression in Graves' orbitopathy. *Eye* 1998;12:800-805.
60. Goldberg RA, Perry JD, Hortaleza V, et al. Strabismus after balanced medial plus lateral wall versus lateral wall only orbital decompression for dysthyroid orbitopathy. *Ophthal Plast Reconstr Surg* 2000;16:271-277.
61. Graham SM, Brown CL, Carter KD, Song A, Nerad J. Medial and lateral orbital wall surgery for balanced decompression in thyroid eye disease. *Laryngoscope* 2003;113:1206-1209.
62. Russo V, Querques G, Primavera V, Delle Noci N. Incidence and treatment of diplopia after three-wall orbital decompression in Graves ophthalmopathy. *J Pediatr Ophthalmol Strabismus* 2004;41:219-225.
63. Ben Simon GJ, Syed HM, Lee S, et al. Strabismus after deep lateral wall orbital decompression in thyroid-related orbitopathy patients using automated Hess screen. *Ophthalmology* 2006;113:1050-1055.
64. Roberts CJ, Murphy MF, Adams GG, Lund VJ. Strabismus following endoscopic orbital decompression for thyroid eye disease. *Strabismus* 2003;11:163-171.
65. Adenis JP, Camezind P, Robert PY. Is incidence of diplopia after fat removal orbital decompression a predictive factor of choice surgical technique for Graves' ophthalmopathy? *Bull Acad Natl Med* 2003;187:1649-1658.
66. Mourits MP, Koorneed L, van Mourik-Noordenbos AM, van der Meulen-Schot HM, Prummel MF, Wiersinga W. Extraocular muscle surgery for Graves' opthalmopathy: Does prior treatment influence surgical outcome? *Br J Ophthalmol* 1990;74:481-483.
67. Oguz V, Yolar M, Pazarli H. Extraocular muscle surgery in dysthyroid orbitopathy: influence of previous conditions on surgical results. *J Pediatr Ophthalmol Strabimus* 2002;39:77-80.
68. Gilbert J, Dailey RA, Christensen LE. Characteristics and outcomes of strabismus surgery after orbital decompression for thyroid eye disease. *J AAPOS* 2005;9:26-30.
69. Dyer JA. The oculorotary muscles in Graves' disease. *Trans Am Ophthmol Soc* 1976;74:425-456.

70. Ellis FD. Strabismus surgery for endocrine ophthalmopathy. *Ophthalmology* 1979;86:2059-2063.
71. Scott WE, Thalacker JA. Diagnosis and treatment of thyroid myopathy. *Ophthalmology* 1981;88:493-498.
72. Meyer DR. Surgical management of Graves orbitopathy. *Curr Opin Ophthalmol* 1999;10:343-351.
73. Lioumi D, Gregson R. Late overcorrections in inferior rectus muscle surgery for vertical squint correction. *Ann Ophthalmol* 2000;32:116-119.
74. Sharma P, Reinecke RD. Single-stage adjustable strabismus surgery for restrictive strabismus. *J AAPOS* 2003;7:358-362.
75. Yan J, Zhang H. The surgical management of strabismus with large angle in patients with Graves' ophthalmopathy. *Int Ophthalmol* 2008;28:75-82.
76. Bandyopadhyay R, Shetty S, Vijayalakshmi P. Surgical outcome in monocular elevation deficit: a retrospective interventional study. *Indian J Ophthalmol* 2008;56:127-133.
77. Hudson HL, Feldon SE. Late overcorrection of hypotropia in Graves ophthalmopathy. *Ophthalmology* 1992;99:356-360.
78. Nardi M, Barca L. Hypercorrection of hypotropia in Graves ophthalmopathy. *Ophthalmology* 1993;100:1-2.
79. Cruz OA, Davitt BV. Bilateral inferior rectus muscle recession for correction of hypotropia in dysthyroid orbitopathy. *J AAPOS* 1999;3:157-159.
80. Yim HB, Biglan AW, Cronin TH. Graded partial tenotomy of the vertical rectus muscles for treatment of hypertropia. *Trans Am Ophthamol Soc* 2004;102:169-176.
81. Vandelanotte S, De Clippeleir L, Dieltiens M, Van Lammeren M. Torsional diplopia in Graves' orbitopathy. Three case reports. *Bull Soc Belge Opthalmol* 2003;288:33-39.
82. Yang ML, Hsu HN, Ma L, Kao LY. Surgical management of strabismus for dysthyroid ophthalmopathy. *Chang Gung Med J* 2004;27:787-793.
83. Garrity JA, Saggau DD, Forman CA, et al. Torsional diplopia after transantral orbital decompression and extraocular muscle surgery associated with Graves' orbitopathy. *Am J Ophthalmol* 1992;113:363-373.
84. Chatzisefanou K, Kusher BJ, Moschos MN. An unusually large incyclotropia associated with dysthyroid ophthalmopathy. *Br J Ophthalmol* 2008;92:291-292.
85. Maillette de Buy Wenniger-Prick LJ, Van Mourik-Noordenbos AM, Koorneef L. Squint surgery in patients with Graves ophthalmopathy. *Doc Ophthalmol* 1986;61:219-221.
86. Flanders M, Hastings M. Diagnosis and surgical management of strabismus associated with thyroid-related orbitopathy. *J Pediatr Ophthalmol Strabismus* 1997;34:333-340.
87. Kuriyan AE, Phipps RP, Feldon SE. The eye and thyroid disease. *Curr Opin Ophthalmol* 2008;19:499-506.
88. Chen CS, Lee AW, Miller NR, Lee AG. Double vision in a patient with thyroid disease: What's the big deal? *Surv Ophthalmol* 2007;52:434-439.
89. Langham EJ. The surgical approach to endocrine ophthalmopathy. *Int Ophthalmol Clin* 1970;10:85-95.
90. Coats DK, Paysee EA, Plager DA, Wallace DK. Early strabismus surgery for thyroid ophthalmopathy. *Ophthalmology* 1999;106:324-329.
91. Inoue Y, Inoue R, Kouzaki A, Koumoto N. Ophthalmic surgery on Graves' ophthalmopathy. *Nippon Rinsho* 2006;61:2291-2296.
92. Scotcher SM, O'Flynn EA, Morris RJ. Inferior rectus recession–An effective procedure? *Br J Ophthalmol* 1997;81:1031-1036.
93. Metz HS. Complications following surgery for thyroid ophthalmopathy. *J Pediatr Ophthalmol Strabismus* 1984;21:220-222.
94. Hodes B, Shoch DE. Thyroid ocular myopathy. *Trans Am Ophthalmol Soc* 1979;77:80-103.
95. Ohnishi T, Noguchi S, Murakami N, et al. Extraocular muscles in Graves ophthalmopathy: usefulness of T2 relaxation time measurements. *Radiology* 1994;190:857-862.
96. Mayer E, Herdman G, Burnett C, Kabala J, Goddard P, Potts MJ. Serial STIR magnetic resonance imaging correlates with clinical score of activity in thyroid disease. *Eye* 2001;15:313-318.
97. Cakirer S, Cakirer D, Basak M, et al. Evaluation of extraocular muscles in the edematous phase of Graves ophthalmology on contrast-enhanced fat-suppressed magnetic resonance imaging. *J Comput Assist Tomogr* 2004;28:80-86.
98. Taoka T, Sakamoto M, Nakagawa H, et al. Evaluation of extraocular muscles using dynamic contrast enhanced MRI in patients with chronic thyroid orbitopahty. *J Comput Assist Tomogr* 2005;29:115-120.
99. Mayer EJ, Fox DL, Herdman G, et al. Signal intensity, clinical activity and cross sectional areas on MRI scans in thyroid eye disease. *Eur J Radiol* 2005;56:20-24.
100. Majos A, Pajak M, Grzelak P, Stefanczyk L. Magnetic resonance evaluation of disease activity in Graves' ophthalmopathy: T2-time and signal intensity of extraocular muscles. *Med Sci Monit* 2007;13(Suppl 1):44-48.
101. Pitz S, Esch A, Muller-Forell WS, Kahaly G, Mann W, Pfeiffer N. Is there a relationship between the degree of preoperative motility impairment or the muscle thickness and the outcome of strabismus surgery in patients with Graves' orbitopathy after decompression surgery? *Orbit* 2005;24:173-176.
102. Gomi CF, Yang S-W, Granet DB, et al. Change in proptosis following extraocular muscle surgery: effects of muscle recession in thyroid-associated orbitopathy. *J AAPOS* 2007;11:377-380.
103. Coats DK, Raab E. Lid position changes following inferior rectus recession. *J Pediatr Ophthalmol Strabismus* 2008;43:134-136.
104. Kushner B. A surgical procedure to minimize lower-eyelid retraction with inferior rectus recession. *Arch Ophthalmol* 1992;110:1011-1014.
105. Meyer DR, Simon JW, Kansora M. Primary infratarsal lower eyelid retractor lysis to prevent eyelid retraction after inferior rectus muscle recession. *Am J Ophthalmol* 1996;122:331-339.

106. Liao SL, Shih MJ, Lin LL-K. A procedure to minimize lower lid retraction during large inferior rectus recession in Graves ophthalmopathy. *Am J Ophthalmol* 2006;143:340-345.
107. McCracken MS, del Prado JD, Granet DB, Levi L, Kikkawa DO. Combined eyelid and strabismus surgery: examining conventional surgical wisdom. *J Pediatr Ophthalmol Strabismus* 2008;45:220-224.
108. Dyer JA. Lesions of the orbit.affecting motility. *Int Ophthalmol Clin* 1971;11:202-207.

PEER DISCUSSION

DR. DAVID K. WALLACE: Thank you for the opportunity to discuss this paper. I have no financial conflict of interest. Thyroid eye disease is an important cause of morbidity, and it certainly can be challenging to correct surgically. Our goal as surgeon is usually to relieve double vision in primary gaze in the reading position, although we also want to maximize the field of single binocular vision as much as possible. I want my patients to understand that, even with a good result, they are still likely to have double vision in extreme positions of gaze.

Dr. Newman presents a retrospective, descriptive case series of 38 patients who had inferior oblique weakening as part of their surgical treatment for strabismus associated with thyroid eye disease. Seven of these patients are presented in detail. His primary study question was: "Is vertical incomitance reduced by operating on the inferior oblique muscle?" Of the 38 patients included in the study, 30 (79%) had additional surgery beyond inferior oblique weakening, and 15 (39%) had 3 or more surgeries. Of the 7 patients presented in detail, all had significant improvement in, or resolution of, diplopia after one or more surgeries. Five of these 7 also had surgery on vertical rectus muscles.

Based on these results, what can we say about whether vertical incomitance is reduced by operating on the inferior oblique muscle? For the seven patients presented in detail, less incomitance was noted postoperatively by Hess screen. However, these results were not formally quantified using an alternate prism and cover and test. In addition, many patients had surgery on other muscles, making it difficult to "tease out" the effect of the inferior oblique surgery. There is insufficient detail presented on the other 31 patients to know if the inferior oblique surgery was effective or not. From these data I conclude that inferior oblique weakening may have a role in some patients. However, I consider this case series to be "hypothesis generating" rather than “hypothesis testing.”

How could the effectiveness of inferior oblique surgery be more directly addressed with a retrospective study design? If alternate prism and cover test measurements in right and left gaze were recorded, then vertical incomitance (or the difference between right and left gaze) could be determined for each patient preoperatively and postoperatively. In order to “tease out” the effect of inferior oblique muscle surgery, a comparison group is needed. To determine the effect of inferior oblique weakening, preoperative and postoperative vertical incomitance could be compared for those patients having inferior oblique surgery alone. To determine the effect of inferior oblique surgery versus rectus muscle surgery, the effect of each surgery on vertical incomitance could be compared. One might find equal or greater effect on vertical incomitance by recessing a tight inferior rectus muscle. To assess the additive effect of inferior oblique surgery, patients having rectus muscle and inferior oblique surgery simultaneously could be compared to those having rectus muscle surgery alone. Other important outcomes include alignment and diplopia in primary and reading positions, number of reoperations, and life quality (when possible).

Finally, I think it is critical to consider torsion as another cause of diplopia in thyroid patients. I routinely measure it preoperatively and incorporate it into the surgical plan. Usually it is due to tight rectus muscles, such as excyclotorsion seen with tight inferior rectus muscles. Fortunately, recessing these tight muscles typically improves the torsion in the proper direction.

In conclusion, Dr. Newman’s case series suggests that there may be advantages to incorporating inferior oblique surgery in the surgical management of strabismus associated with thyroid eye disease. These advantages include improvement of vertical incomitance, reduction in the amount of recession of the inferior rectus (if required) with less lower eyelid retraction, and improvement in excyclotorsion. Disadvantages to this approach include that inferior oblique surgery alone would be expected to undercorrect most thyroid patients (as found in this case series), rectus muscle restriction is not relieved, and more surgeries are probably required.

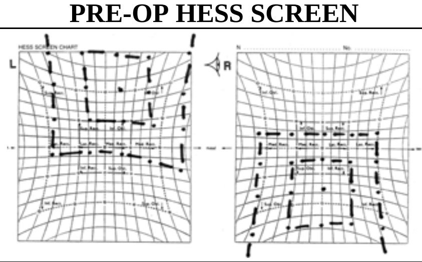
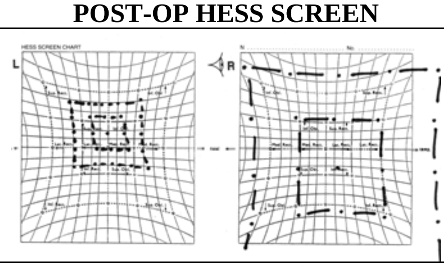
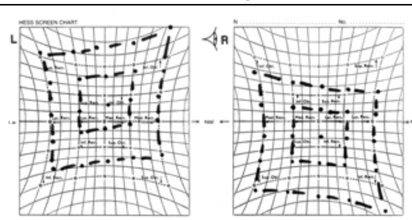
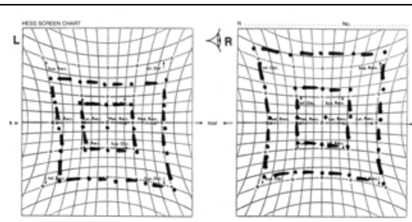
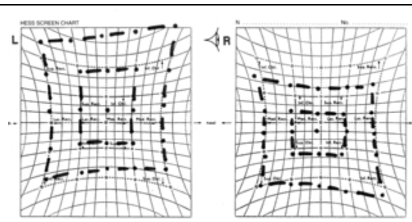
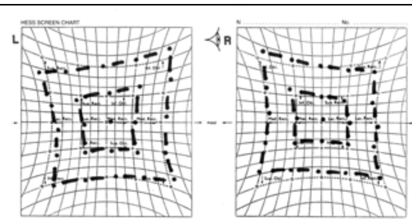
ACKNOWLEDGEMENTS

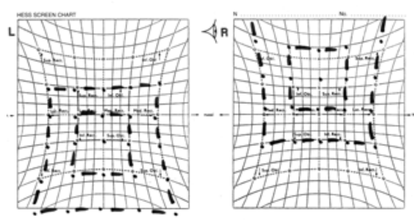
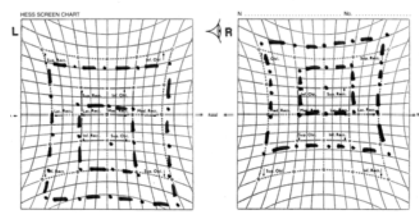
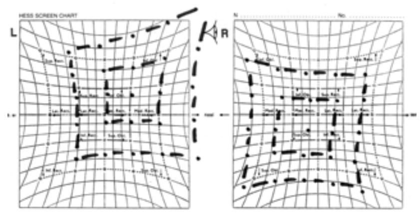
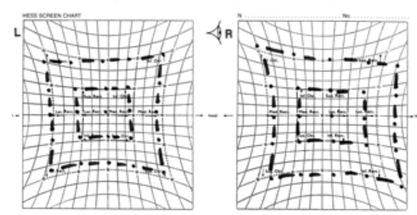
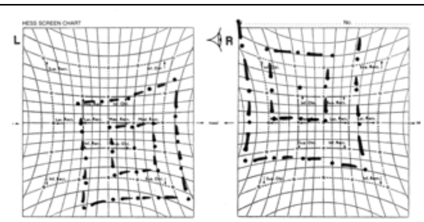
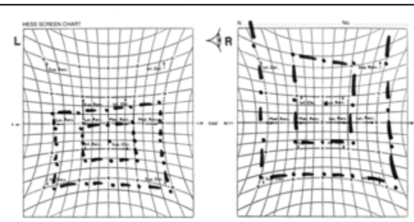
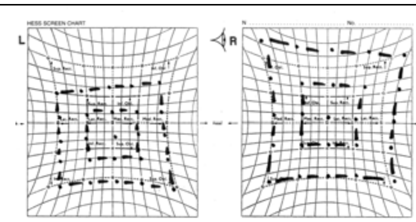
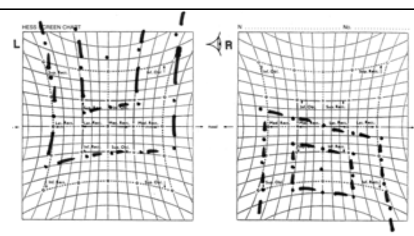
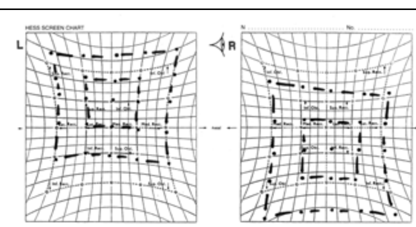
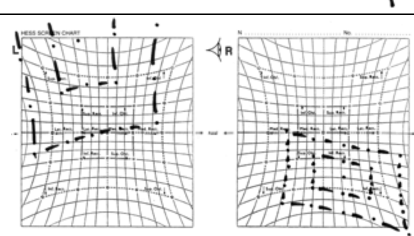
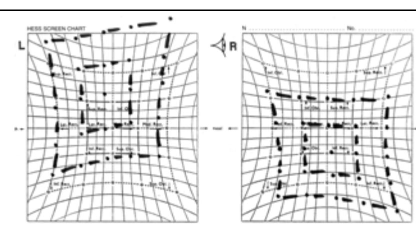
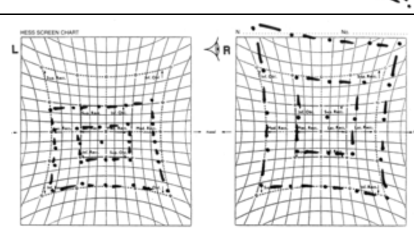
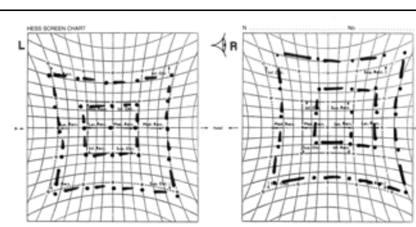
Funding/Support: None
Financial Disclosures: None

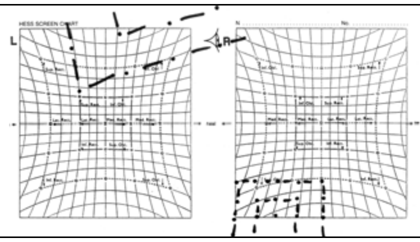
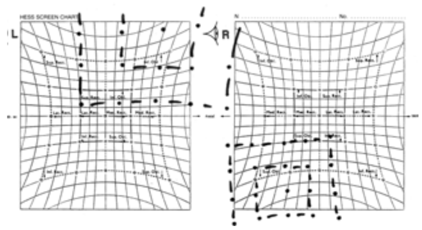
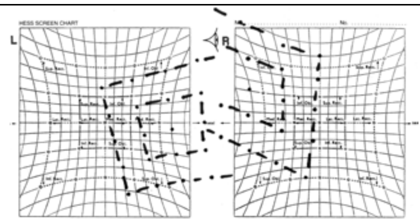
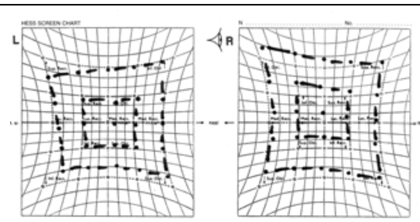
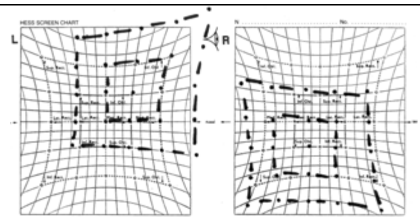
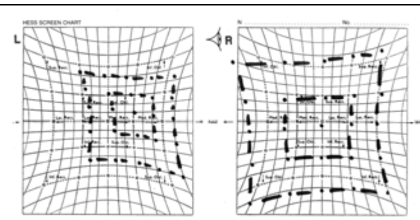
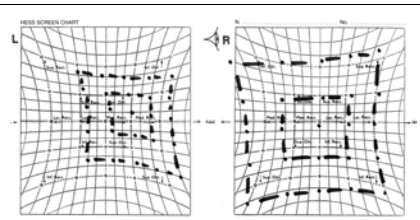
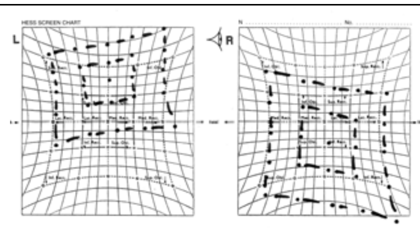
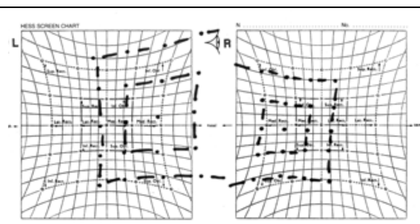
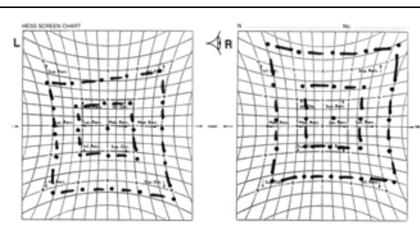
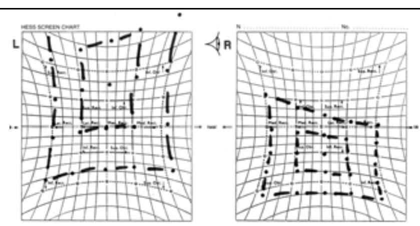
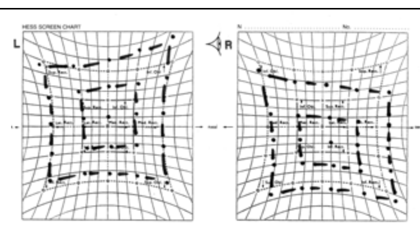
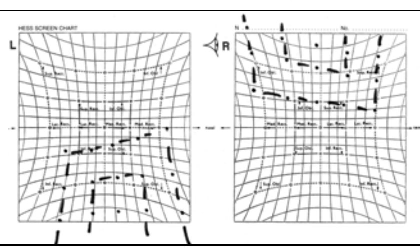
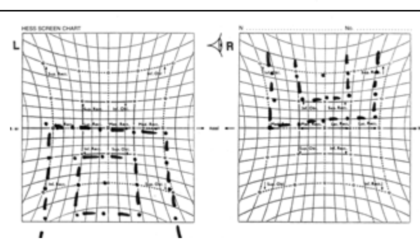
DR. IRENE H. LUDWIG: No conflicts. I believe that this is actually very important paper and a very important concept. I believe that this has been under-recognized in management of thyroid ophthalmopathy patients and in all patients with restrictive strabismus. These patients are typically told to just hope for alignment in the primary position and to put up with residual diplopia in other gaze positions. By careful tailoring of the operations that are in our armamentarium we can do much better than that. The inferior oblique recession, which is the operation I use for this, mainly results in a tether effect reducing up gaze and if you carefully titrate the surgery, can be very useful to balance the upgaze restriction in the other eye. I would consider graded recessions, rather than inferior extirpations, as you then have more flexibility to adjust the result in the case of over or undercorrection. I would also like to add that the very common late over correction after inferior rectus recessions are usually caused by stretching of the scar between the inferior rectus and the sclera. The inferior rectus is a very powerful muscle with an extreme force in thyroid ophthalmopathy and will gradually stretch away. This can usually be prevented if you use poly (ethylene terephthalate) (Mersilene polyester suture, Ethicon, Inc.) suture on the inferior rectus instead of polyglactin 910 (Vicryl, Ethicon, Inc.). An undercorrection of upgaze restriction after recession of the inferior oblique can also be due to stretching or migration of the inferior oblique. This can also be prevented with the use of non-absorbable suture. I usually will use a very small inferior rectus recession combined with an inferior oblique recession on the other eye. By balancing your surgical intervention, it is possible to sometimes obtain 100% functional motility in all gaze directions. I believe that it is a very important concept and am glad you have presented this information.

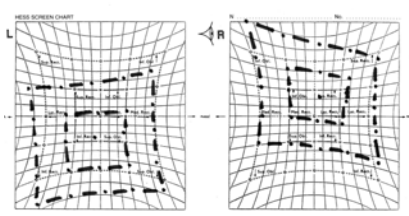
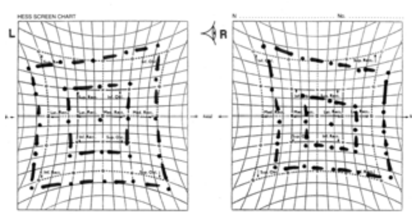
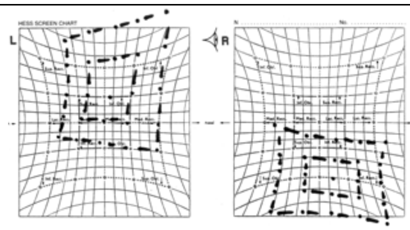
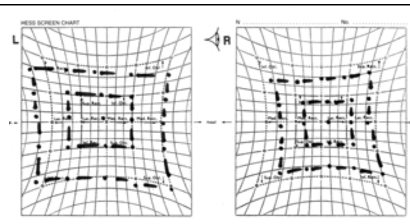
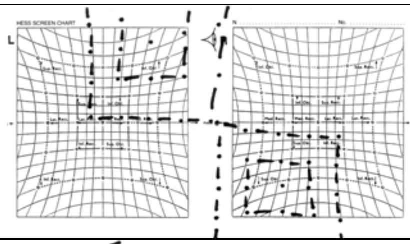
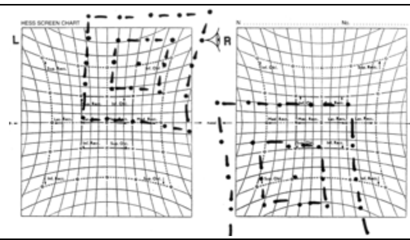
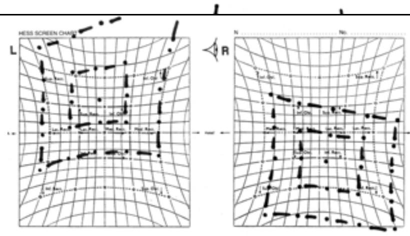
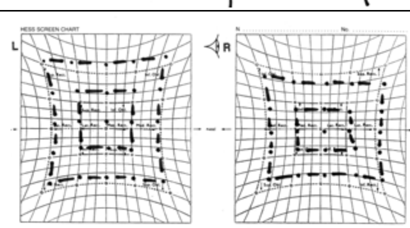
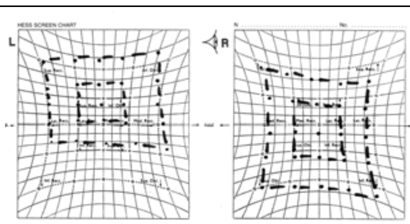
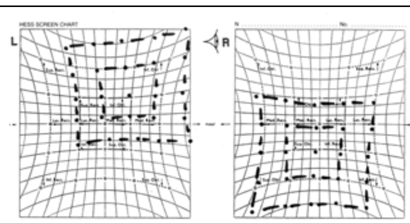
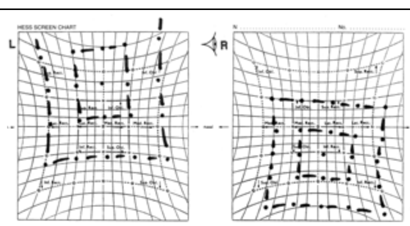
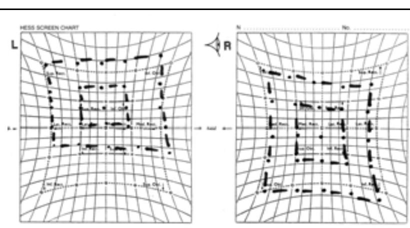
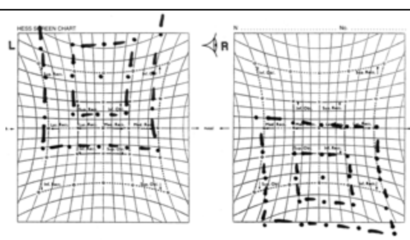
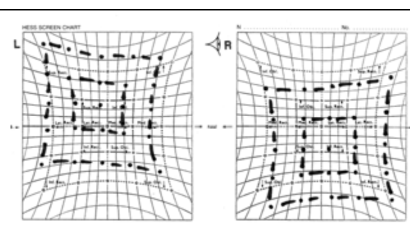
DR. M. EDWARD WILSON: No conflicts of interest. I think that what this paper represents is the concept of utilizing a normal muscle to match a duction limitation. With thyroid disease, like in some other conditions, we cannot always restore full ductions. I believe it is a good, yet under utilized, concept that if you cannot restore normal ductions you may utilize a normal muscle in the other eye and actually create a duction deficit to match that. We use this approach in strabismus associated with blow-out fracture by performing a posterior fixation suture on the opposite inferior rectus muscle. Using the same concept in thyroid associated restrictive strabismus is probably worth emphasizing and it will likely help. I remember a long time ago Bill Scott saying “If a horse limps you kick him in the other leg and he doesn’t limp anymore”. That is the concept that you are presenting.

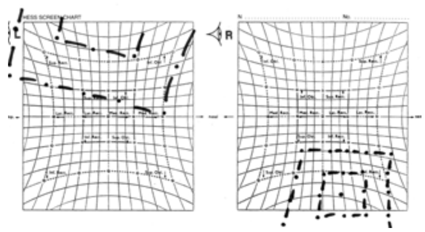
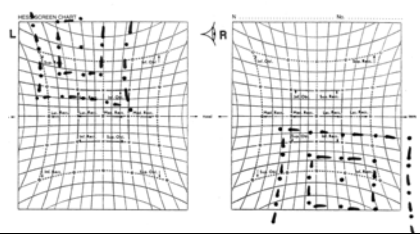
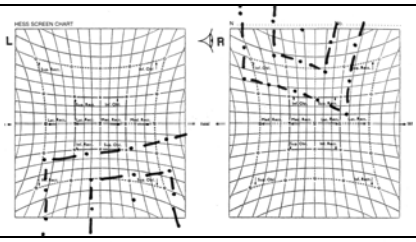
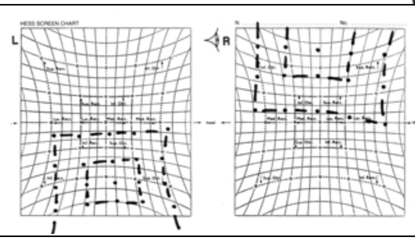
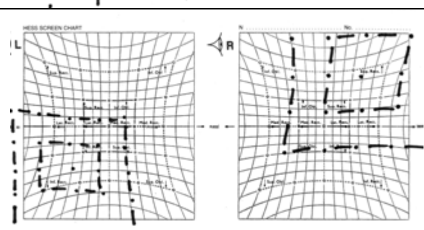
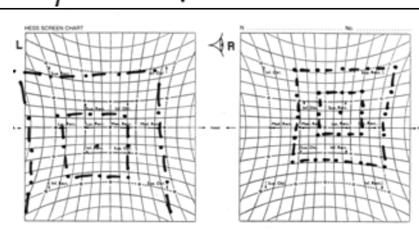
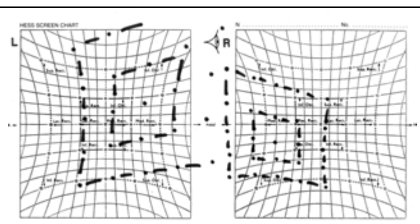
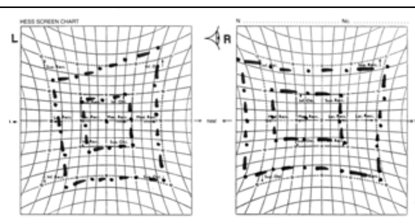
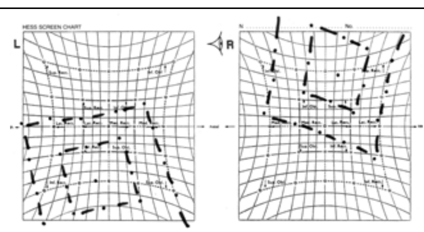
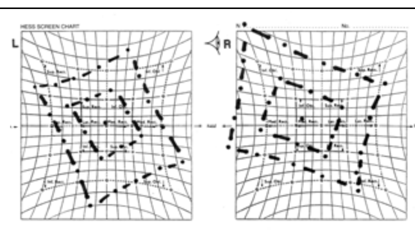
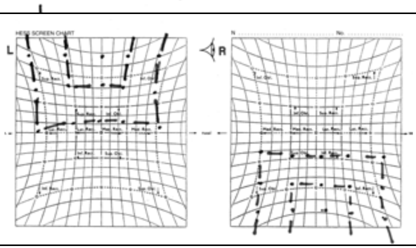
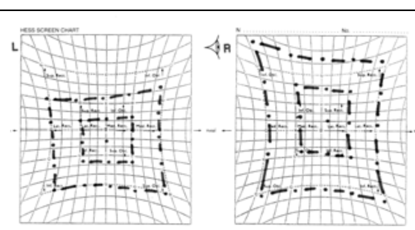
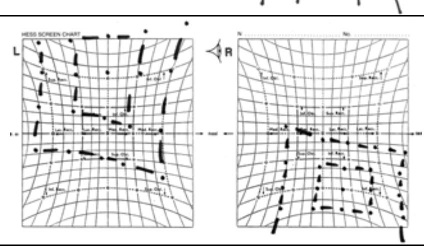
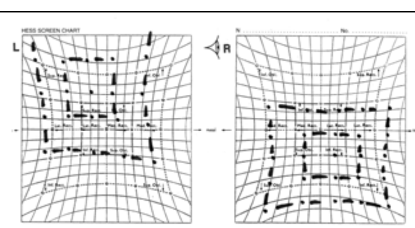
DR. STEVEN A. NEWMAN: I thank you for the comments. I like the horse limping analogy. What I tell my patients is “in patients with thyroid orbitopathy with restrictive strabismus I am never going to make the poorer moving eye move normally” because the restricted muscle is not normal. It is not just tight, but the excursion is limited. The Hess screen allows us to see the movement of the two eyes relative to each other. Thus, by weakening the better moving eye (inferior oblique surgery in this series) and utilizing the Hess screen to guide us, we can maximize binocularity. I am going to take this opportunity to read a quote from John Dyer who spoke here more than a few years ago in 1971. He wrote “since the muscles are no longer normal the patient cannot expect to have a full range of binocular vision once the eye is operated on and it is extremely important to impress on these patients that what we hope to gain is single binocular vision in a reasonable area in the primary position and in reading gaze. I suggest that the Lancaster red green test and the Hess screen are extremely valuable tools to use preoperatively as well as postoperatively.” As Bill Hoyt who was quoted yesterday once said, “When you think you have discovered anything new you have not been reading the old German literature” and obviously not paying attention to what has been presented at the AOS. I appreciate Dr. Wallace’s comments, he is absolutely correct that to make a change in our practice we want to analyze all data, not just selected cases. I am appending a table with both pre and postoperative Hess screens which will permit readers to better assess the effect of inferior oblique surgery. Thank you.

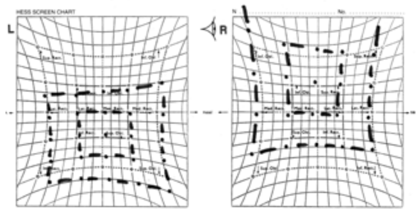
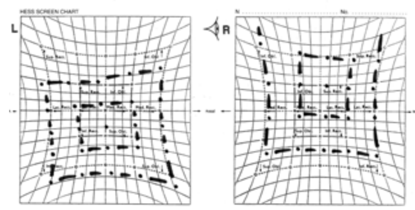
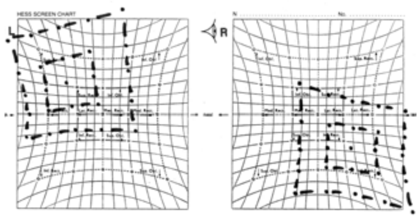
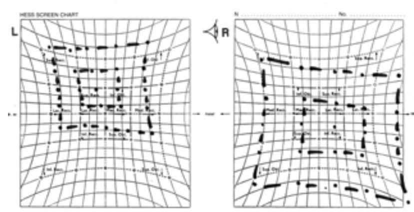
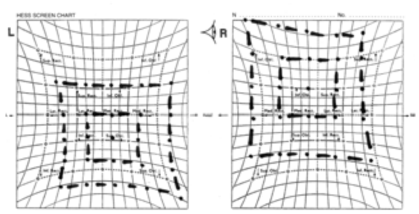
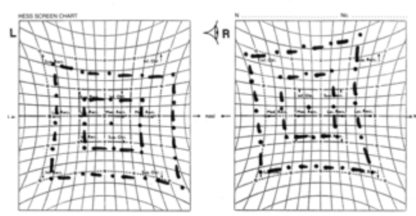
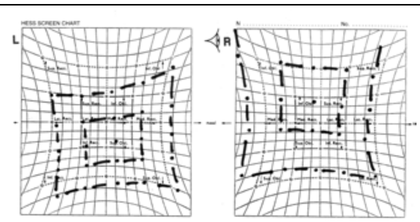
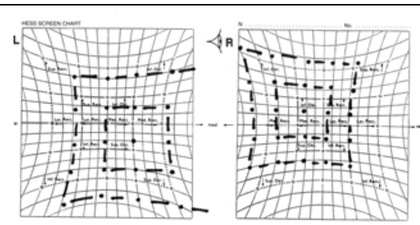
PATIENT	PRE-OP HESS SCREEN	POST-OP HESS SCREEN	COMMENTS
1			Inferior oblique plus ipsilateral superior rectus.
2			
3			

PATIENT	PRE-OP HESS SCREEN	POST-OP HESS SCREEN	COMMENTS
4			
5			Inferior oblique plus ipsilateral medial rectus.
6			
7			
8			
9			
10			
11			

PATIENT	PRE-OP HESS SCREEN	POST-OP HESS SCREEN	COMMENTS
12			
13			
14			Inferior oblique plus contralateral medial rectus.
15			
16			
17			Inferior oblique plus bilateral medial rectus.
18			
19			

PATIENT	PRE-OP HESS SCREEN	POST-OP HESS SCREEN	COMMENTS
20			
21			
22			
23			
24			
25			
26			

PATIENT	PRE-OP HESS SCREEN	POST-OP HESS SCREEN	COMMENTS
27			
28			Inferior oblique plus ipsilateral medial rectus.
29			Inferior oblique plus ipsilateral lateral rectus.
30			Inferior oblique plus ipsilateral lateral rectus.
31			
32			Inferior oblique plus ipsilateral superior rectus.
33			

PATIENT	PRE-OP HESS SCREEN	POST-OP HESS SCREEN	COMMENTS
34			
35			Inferior oblique plus ipsilateral lateral rectus.
36			
37			Inferior oblique plus ipsilateral lateral rectus.
38