THYROID
Volume 14, Number 5, 2004

Primary-Gaze Diplopia in Patients with Thyroid-Related Orbitopathy Undergoing Deep Lateral Orbital Decompression with Intraconal Fat Debulking: A Retrospective Analysis of Treatment Outcome

Guy J. Ben Simon, Lillian Wang, John D. McCann, and Robert A. Goldberg

Our goal was to investigate the incidence of postoperative primary gaze diplopia in patients with thyroid-related orbitopathy (TRO) undergoing deep lateral wall orbital decompression surgery with intraconal fat debulking in the Jules Stein Eye Institute over a period of $4^1/_4$ years. Overall 201 orbital decompression surgeries were performed in 116 patients (23 males, 93 females). All surgeries were performed by two of the authors (R.A.G. and J.D.M.) and in the noninflammatory phase of the disease. Exophthalmos decreased by an average of 3.4 ± 2.7 mm from 23.8 ± 3.2 mm (17–31) to 20.4 ± 2.5 mm (14–29), $p < 0.001$, 95% confidence interval (CI) (3.0:3.8). 31% of patients had preoperative primary gaze diplopia and 28.4% had postoperative primary gaze diplopia. Thirty (83%) of the 36 patients with preoperative diplopia had also postoperative diplopia; 6 (16.7%) of the 36 patients had improvement in diplopia following deep lateral wall decompression. Of the 80 (69%) of patients without preoperative double vision 3 developed postoperative double vision in primary gaze (2.6% of all patients). These 3 patients were older (56 versus 46 years, $p = 0.047$), had more limitation in ocular movements ($p = 0.017$) and achieved more decrease in proptosis with surgery (6 versus 3.1 mm, $p = 0.024$). No complications were associated with orbital decompression. In conclusion deep lateral wall orbital decompression surgery with intraconal fat debulking is associated with a low rate (2.6%) of new-onset primary gaze diplopia. Some patients (5.2%) with preoperative diplopia actually had improvement in diplopia postoperatively. This surgery is effective in reduction of congestion and exophthalmos, and is not associated with detrimental effects on visual acuity.

Introduction

Orbital decompression is effective in treating compressive optic neuropathy, exposure keratopathy and proptosis in thyroid related orbitopathy (TRO) (1–3). It is useful in treating congestive orbitopathy. Patients with thyroid orbitopathy often have a diffuse pressure pain and some diffuse limitation of eye movements, which is related to decreased venous outflow and orbital congestion (4,5). This symptom complex can be substantially disabling, and responds well to orbital decompression, which improves venous outflow and relieves or substantially improves the congestive symptoms. Many patients without severe proptosis or compressive optic neuropathy suffer from the symptoms of congestive orbitopathy. In the postinflammatory phase, medical treatment and radiation are typically ineffective in relieving these symptoms.

Diplopia is a common complaint in patients with TRO. Worsening of preexisting diplopia or development of new-onset diplopia remains a major concern in patients undergoing orbital decompression surgery with rates as high as 63% being reported (4,6–8). Primary gaze diplopia is most debilitating to patients, especially when it cannot be adequately corrected using prisms. Many patients prefer to cover one eye in an attempt to avoid double vision.

Several investigators advocate different surgical techniques with the goal of reducing postoperative diplopia. A common approach is balanced orbital decompression by medial and lateral wall surgery. This approach results in approximately 10% rate of postoperative diplopia (9,10). Other investigators report even lower incidences of post-operative diplopia (8,11–13).

The purpose of this study was to present a retrospective evaluation of the rates of new-onset double vision after deep

Jules-Stein Eye Institute and Department of Ophthalmology, David Geffen School of Medicine at UCLA, Los Angeles, California.

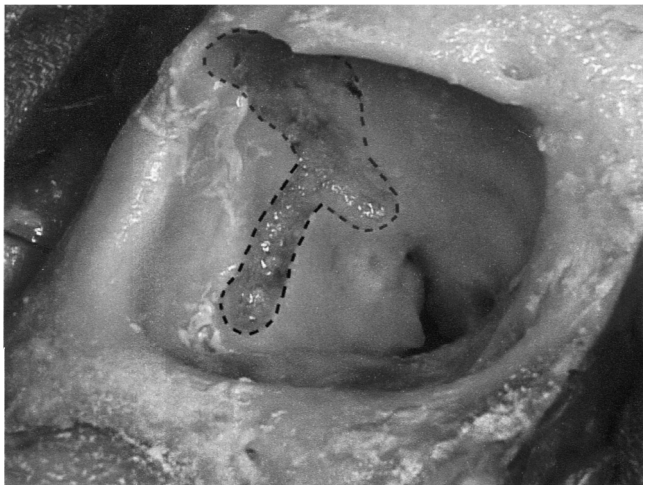

FIG. 1. Cadaver dissection showing the extent of orbital decompression (dashed line) in relation to anatomic superior and inferior orbital fissure.

lateral wall orbital decompression with intraconal fat removal.

Patients and Methods

This study is a retrospective, interventional cases series. Medical records of all patients who underwent orbital decompression for TRO at the Jules Stein Eye Institute between January 1, 1999 and March 1, 2003 were reviewed. The study complied with the policies of the local Institutional Review Board. Data regarding primary gaze strabismus, visual acuity, intraocular pressure (IOP), exophthalmometry, clinical assessment of duction restriction and single binocular vision, and complications of surgery were recorded and analyzed.

Patients were categorized according to one of the following groups: those without preoperative primary gaze diplopia and no new-onset postoperative diplopia (group no, no); those without preoperative diplopia who later developed new-onset permanent diplopia (no, yes); those who had preoperative diplopia and remained that way after decompression surgery (yes, yes) and those who had preoperative diplopia that resolved after orbital decompression (yes, no).

Surgical technique

The orbital surface of the sphenoid bone was exposed through an eyelid crease incision. Using a high-speed neurosurgical drill, cortical bone was removed from the lacrimal gland fossa, the marrow space of the sphenoid between the superior and inferior orbital fissure, and the zygomatic marrow space on the anterior rim of the inferior orbital fissure (Fig. 1). The extent of bone removal was individualized: patients with substantial proptosis, for example more than 26 mm, underwent maximal bone removal from each of the three areas, but patients with lesser degrees of proptosis were treated with more conservative bone removal. In all patients, the maximal available intraconal fat located between the lateral and inferior rectus muscle was bluntly dissected out of the muscle cone and excised; the volume of fat removed ranged from 1.5 to 3 cc.

Statistical analysis

Statistical analysis was performed using paired samples *t* test to evaluate preoperative and postoperative data such as visual acuity (VA), IOP, exophthalmometry, and strabismus measurements. Pearson bivariate correlation was used to examine influence of age, visual acuity, IOP and extent of exophthalmos on treatment outcome. Nonparametric Wilcoxon Mann-Whitney two independent samples were used to compare different variables in patients without preoperative diplopia who developed consecutive diplopia to those who remained symptoms free postoperatively.

Results

Overall, 201 orbital decompression surgeries were performed for TRO on 116 patients (23 males, 93 females). Data regarding patient demographics are summarized in Table 1. Mean visual acuity and IOP remained unchanged postoperatively. After orbital decompression exophthalmometry measurements decreased by an average of 3.4 ± 2.7mm from 23.8 ± 3.2mm (17–31) to 20.4 ± 2.5mm (14–29), $p < 0.001$, 95% CI (3.0:3.8; Fig. 2).

Most of the patients who had preoperative primary-gaze diplopia showed evidence of postoperative diplopia. Thirty-one percent of patients had preoperative primary gaze diplopia and 28.4% had postoperative primary gaze diplopia, with horizontal strabismus being the most common pattern (Table 2).

Limitations in ocular ductions in all positions of gaze did not change significantly post-operatively (Table 2), limitations in upgaze were most common. Field of binocular single vision increased postoperatively in up-gaze and down-gaze.

TABLE 1. DEMOGRAPHICS OF STUDY POPULATIONS

201 cases (116 patients)	*Preoperative*	*Postoperative*	p
Gender	Male 23 (20%) Female 93 (80%)		
Age (years)	48 ± 12 (22–78)		
Follow-up (months)	11.8 ± 10.3 (6–57.4)		
Visual acuity	20/25 (20/15–20/1600)	20/25 (20/15–20/800)	ns
Proptosis (mm)	23.8 ± 3.2 (17.0–31)	20.4 ± 2.5 (14–29)	< 0.001
IOP	15.7 ± 3 (10–21)	18.9 ± 2.3 (16–22)	ns

IOP, intraocular pressure; ns, not significant.

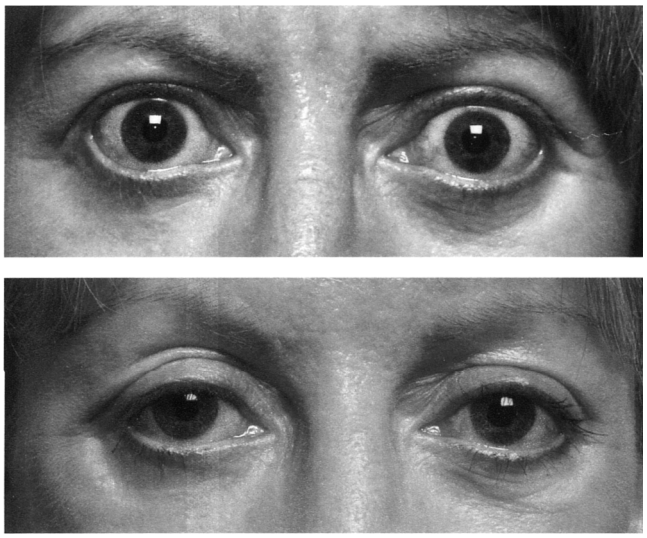

FIG. 2. Clinical photograph of a 41–year-old female preoperative (upper image) and 1 year postoperative (lower image) bilateral deep lateral wall orbital decompression. This patient also had bilateral eyelid lowering surgery as well as the orbital decompression.

Thirty-six patients (31%) had preoperative strabismus, defined as diplopia in the primary position of gaze. Of this subset, 30 (83%) had persistence of double vision after surgery. Six (16.7%) had improvement in double vision after orbital decompression surgery to the point that single vision was present in primary gaze. Of the remaining 80 (69%) patients without preoperative strabismus, 3 patients (2.6% of all patients) had double vision in primary position after surgery (Table 3 and Fig. 3).

The three patients (six cases) who did not have diplopia preoperatively but who developed primary gaze diplopia after surgery were older (56 ± 8.2 versus 46 ± 11 years; $p = 0.047$), Wilcoxon Mann-Whitney independent samples), had more limitation in ocular movements (limitation in up-gaze -1.3 ± 1.0 versus -0.4 ± 0.6, $p = 0.017$), had lower postoperative visual acuity (20/40 versus 20/20, $p = 0.01$) and achieved a greater decrease in proptosis with surgery (6 versus 3.1mm, $p = 0.024$). They also had more proptosis preoperatively (26.2 ± 2.9 mm versus 23.6 ± 3.2 mm) but this was not statistically significant ($p = 0.07$).

No severe complications of orbital decompression such as stroke, death, or vision loss occurred. In this series of patients there were no dural tears or cerebrospinal fluid (CSF) leaks. Numbness or paresthesia in the lacrimal and zygomaticofacial nerve distributions occurred frequently, representing the most common postoperative complaint reported by patients, but severe prolonged numbness, paresthesia, or neuralgia did not occur in this series of patients.

Discussion

Our results support deep lateral orbital decompression with intraconal fat removal as a safe and effective surgery in patients with TRO. This type of surgery is also associated

Table 2. Strabismus Patterns and Limitations in Ocular Ductions Preoperatively and Postoperatively

	Preoperative	*Postoperataive*	p
Diplopia[a]	31%	28.4%	
Esotropia	39.1%	62.5%	
Exotropia	26.1%	12.5%	
Hypertropia	8.7%	10%	ns
Hypotropia	26.1%	15%	
Ocular ductions limitations[b]			
Up-gaze	−1.1	−1.1	
Down-gaze	−0.2	−0.3	
Right	−0.8	−0.6	ns
Left	−0.8	−0.8	
Binocular single vision (degrees)[c]			
Up-gaze	10°	20°	< 0.001
Down-gaze	30°	40°	< 0.001
Right	30°	30°	ns
Left	40°	30°	< 0.001

[a]Angle of deviation was measured in prism diopters in 50% of the cases with primary gaze diplopia.
[b]Range (−4–0) 0, normal ductions; −4, severe restriction in gaze.
[c]*p* values for binocular single vision was calculated using Wilcoxon two-related samples (signed-ranks nonparametric test).

Table 3. Primary Gaze Diplopia According to Surgical Decompression Technique, Preoperatively and Postoperatively

Group	n *patients (%)*
Preoperative and postoperative no diplopia (no, no)	77 (66.4%)
Preoperative no diplopia, Postoperative diplopia (no, yes)	3 (2.6%)
Preoperative diplopia, Postoperative no diplopia (yes, no)	6 (5.2%)
Preoperative diplopia and postoperative diplopia (yes, yes)	30 (25.8%)

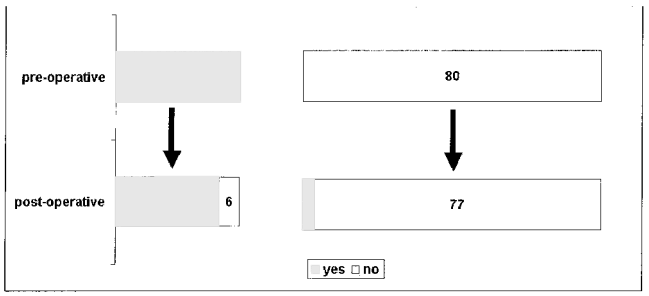

FIG. 3. Number of patients with preoperative and postoperative primary gaze diplopia. no, no primary-gaze diplopia; yes, primary-gaze diplopia (either preoperatively or postoperatively).

with a low rate of new-onset postoperative primary gaze diplopia.

Reducing the incidence of postoperative new-onset diplopia has been a goal of many reported variations in orbital decompression surgery and rates of new onset strabismus range from 0% to 62.5% (Table 4). These studies, similar to the series we present, suffer from the retrospective nature of determination of motility status, and by lack of controlled standardization of the study population. The available studies do not therefore answer the question whether one technique results in less new onset strabismus. However, overall the balanced or lateral decompression probably results in less new onset strabismus and our own experience and reported work supports that concept (8–11).

Our orbital decompression technique (19) involves the deep lateral wall and is similar to the neurosurgical approach. The surgery we perform is more extensive than the anterior lateral wall approach previously reported by other investigators.

In our study patients without primary gaze diplopia who developed new onset postoperative diplopia had a trend toward more proptosis preoperatively compared to those who did not develop consecutive strabismus; they also achieved more reduction in exophthalmos. We think that greater reduction in orbital volume with surgery may be the cause of extraocular muscle misalignment and inflammation or scarring of the orbital tissue. These patients had more limitation in ocular movements preoperatively which may imply an increased risk for developing new-onset postoperative diplopia. These findings are not in line with the work of Nagy et al. (20) who examined the mechanism of diplopia and found no correlation between connective tissue expansion, proptosis, and diplopia grade or muscle thickness and concluded that connective tissue expansion is not a major factor in diplopia.

Similar results were reported by other investigators (21,22). Postoperative primary gaze diplopia was found to be more common in patients with preoperative restrictive motility or diplopia compared to patients with normal versions and without diplopia preoperatively (21,22). Patients with TRO who have had orbital decompression have a lower success rate of surgery for strabismus, more frequently need correction for both horizontal and vertical deviations, and have more muscles operated on than patients who have not had orbital decompression (23).

We found no significant change of ductions in any direction at 2 months after surgery; these findings are similar to reports by other investigators (10).

Over the past 15 years, we have moved away from the unbalanced inferomedial approach, and utilize the deep lateral orbit, combined with intraconal fat debulking, as the first-line surgical treatment.

One of the goals of converting to a deep lateral approach for orbital decompression, including intraconal fat debulking, was the anticipation that the risk of consecutive strabismus would be reduced. Previous studies, including one from our institute (11), have suggested this. The results of this study suggest a decreased incidence of new onset diplopia compared to previous series with inferomedial orbital decompression, including report from our center.

The present study was an uncontrolled study, and the patient population of the current cohort may not be comparable to patients in other studies. However, we believe, as do other authors, that new onset strabismus is reduced with the deep lateral approach compared to the unbalanced inferomedial approach. This may be related to decreased medial shifting of the muscle cone as opposed to unbalanced decompression where the muscle cone is shifted immediately into the sinus cavities placing additional stretch on the already tight inferior and medial rectus muscle, exacerbating esotropia and hypotropia.

Our initial concern in removing intraconal fat was that dissection and scar tissue within the muscle cone might pre-

TABLE 4. INCIDENCE OF NEW-ONSET PRIMARY GAZE DIPLOPIA IN DIFFERENT TECHNIQUES OF ORBITAL DECOMPRESSION SURGERY

Author	*Type of orbital decompression*	n *(patients)* (n *cases)*	*New-onset diplopia*
Metson and Samaha (7)	Endoscopic decompression using orbital sling	13 (20)	0%
	Endoscopic decompression without a sling	24 (38)	30%
Seiff et al. (14)	Transantral with preservation of anterior periorbita	15 (30)	0%
Linnet et al. (15)	Two walls transcranial	30 (50)	3.3%
Kalmann et al. (12)	Three walls, coronal approach	125	3.2%
Eloy et al. (16)	Transnasal	16 (27)	62.5%
Shepard et al. (17)	Endoscopic medial and extended lateral	11 (18)	18%
Wright et al. (18)	Transnasal endoscopic medial and inferior	11 (21)	18%
	Transnasal endoscopic medial and inferior with preservation of the inferomedial bony strut	6	0%

dispose patients to double vision. However, the results of this study do not suggest that removing intraconal fat increases the risk of double vision, relative to the inferomedial orbital decompression. In fact, our clinical intuition is that removing intraconal fat, and breaking up some of the fibrous septa within the congestive orbit, may actually improve ocular motility.

Although no severe complications of orbital decompression occurred in our study, potential complication of such a surgery may include: intraorbital hemorrhage, vision loss, dural tears, and lateral canthal misalignment.

In summary, we have converted to a paradigm of removing the deep lateral wall and intraconal fat as the first approach for orbital decompression in TRO. Our experience demonstrated in this retrospective study suggests a lower rate of new-onset strabismus compared to the inferomedial approach. Orbital decompression surgery is effective in treating the symptoms of congestion and bulging that are disabling for patients with TRO, newer techniques substantially decrease the incidence of new-onset strabismus, and the risk of severe complications such as stroke, death, or decreased vision are low.

Acknowledgments

The work was supported in part by Dr. Bernard and the Jenny Nelson Trust.

References

1. Kazim M, Trokel SL, Acaroglu G, Elliott A 2000 Reversal of dysthyroid optic neuropathy following orbital fat decompression. Br J Ophthalmol **84:**600–605.
2. Hutchison BM, Kyle PM 1995 Long-term visual outcome following orbital decompression for dysthyroid eye disease. Eye **9:**578–581.
3. Trobe JD, Glaser JS, Laflamme P 1978 Dysthyroid optic neuropathy. Clinical profile and rationale for management. Arch Ophthalmol **96:**1199–1209.
4. Lyons CJ, Rootman J 1994 Orbital decompression for disfiguring exophthalmos in thyroid orbitopathy. Ophthalmology **101:**223–230.
5. Inoue Y, Tsuboi T, Kouzaki A, Maeda T, Inoue T 2002 Ophthalmic surgery in dysthyroid ophthalmopathy. Thyroid **12:**257–263.
6. Paridaens D, Hans K, van Buitenen S, Mourits MP 1998 The incidence of diplopia following coronal and translid orbital decompression in Graves' orbitopathy. Eye **12:**800–805.
7. Metson R, Samaha M 2002 Reduction of diplopia following endoscopic orbital decompression: The orbital sling technique. Laryngoscope **112:**1753–1757.
8. Unal M, Leri F, Konuk O, Hasanreisoglu B 2003 Balanced orbital decompression combined with fat removal in Graves ophthalmopathy: Do we really need to remove the third wall? Ophthal Plast Reconstr Surg **19:**112–118.
9. Graham SM, Brown CL, Carter KD, Song A, Nerad JA 2003 Medial and lateral orbital wall surgery for balanced decompression in thyroid eye disease. Laryngoscope **113:** 1206–1209.
10. Paridaens DA, Verhoeff K, Bouwens D, van Den Bosch WA 2000 Transconjunctival orbital decompression in Graves' ophthalmopathy: Lateral wall approach ab interno. Br J Ophthalmol **84:**775–781.
11. Goldberg RA, Perry JD, Hortaleza V, Tong JT 2000 Strabismus after balanced medial plus lateral wall versus lateral wall only orbital decompression for dysthyroid orbitopathy. Ophthal Plast Reconstr Surg **16:**271–277.
12. Kalmann R, Mourits MP, van der Pol JP, Koornneef L 1997 Coronal approach for rehabilitative orbital decompression in Graves' ophthalmopathy. Br J Ophthalmol **81:**41–45.
13. Vaseghi M, Tarin TT, Levin PS, Terris DJ 2003. Minimally invasive orbital decompression for Graves' ophthalmopathy. Ann Otol Rhinol Laryngol **112:**57–62.
14. Seiff SR, Tovilla JL, Carter SR, Choo PH 2000 Modified orbital decompression for dysthyroid orbitopathy. Ophthal Plast Reconstr Surg **16:**62–66.
15. Linnet J, Hegedus L, Bjerre P 2001 Results of a neurosurgical two-wall orbital decompression in the treatment of severe thyroid associated ophthalmopathy. Acta Ophthalmol Scand **79:**49–52.
16. Eloy P, Trussart C, Jouzdani E, Collet S, Rombaux P, Bertrand B 2000 Transnasal endoscopic orbital decompression and Graves' ophtalmopathy. Acta Otorhinolaryngol Belg **54:**165–174.
17. Shepard KG, Levin PS, Terris DJ 1998 Balanced orbital decompression for Graves' ophthalmopathy. Laryngoscope **108:**1648–1653.
18. Wright ED, Davidson J, Codere F, Desrosiers M 1999 Endoscopic orbital decompression with preservation of an inferomedial bony strut: Minimization of postoperative diplopia. J Otolaryngol **28:**252–256.
19. Goldberg RA, Kim AJ, Kerivan KM 1998 The lacrimal keyhole, orbital door jamb, and basin of the inferior orbital fissure. Three areas of deep bone in the lateral orbit. Arch Ophthalmol **116:**1618–1624.
20. Nagy EV, Toth J, Kaldi I, Damjanovich J, Mezosi E, Lenkey A, Toth L, Szabo J, Karanyi Z, Leovey A 2000 Graves' ophthalmopathy: Eye muscle involvement in patients with diplopia. Eur J Endocrinol **142:**591–597.
21. Shorr N, Neuhaus RW, Baylis HI 1982 Ocular motility problems after orbital decompression for dysthyroid ophthalmopathy. Ophthalmology **89:**323–328.
22. Nunery WR, Nunery CW, Martin RT, Truong TV, Osborn DR 1997 The risk of diplopia following orbital floor and medial wall decompression in subtypes of ophthalmic Graves' disease. Ophthal Plast Reconstr Surg **13:**153–160.
23. Ruttum MS 2000 Effect of prior orbital decompression on outcome of strabismus surgery in patients with thyroid ophthalmopathy. J AAPOS **4:**102–105.

Address reprint requests to:
Guy J. Ben Simon, M.D.
Jules-Stein Eye Institute
100 Stein Plaza
Box 957006
Los Angeles, CA 90095-7006

E-mail: simon@jsei.ucla.edu