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Article

Motility Changes and Diplopia After Baerveldt Glaucoma Drainage Device Implantation or After Trabeculectomy

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Purpose: The purpose of this study was to quantify any diplopia and motility changes after the implantation of a Baerveldt glaucoma implant (BGI) or after trabeculectomy (TE).

Methods: We analyzed 51 patients with a BGI and 52 patients with a TE from a prospective cohort study. To quantify any diplopia, we asked patients about the presence of diplopia at 1 year after surgery. To quantify any ocular motility changes, we measured ductions in eight gaze directions, the patients' ocular alignment and their fusion range before and 1 year after surgery.

Results: In the BGI group, 14 patients (28%) experienced diplopia compared with one patient (2%) in the TE group ($P < 0.001$). Duction changes were more commonly observed in the BGI group (35%) than in the TE group (19%). In the BGI group, ductions were mostly restricted in elevation (13%; $P < 0.001$), in abduction (13%), in elevation in 25° adduction (13%; $P = 0.044$), and in elevation in 25° abduction (25%; $P < 0.001$). In 32% of the patients, their near horizontal ocular alignment shifted, notably in exodirection ($P = 0.04$). The fusion range decreased significantly in the horizontal direction ($-12.6° \pm 10.3°$, mean $\pm$ standard deviation; $P = 0.01$).

Conclusions: BGI surgery was significantly associated with postoperative diplopia and impaired eye motility (reduced ductions), mostly present in abduction, elevation, elevation in 25° adduction, and elevation in 25° abduction. Even without impaired ductions, diplopia could come about.

Translational Relevance: By studying diplopia across glaucoma patients prospectively with diplopia questionnaires and extensive orthoptic measurements, we gain better insight into its occurrence.

Introduction

Diplopia is a known complication of implanted glaucoma drainage devices (GDDs), such as the Baerveldt glaucoma implant (BGI).[1–4] In daily life, our eyes move in unison to maintain binocular vision and to avoid diplopia. However, glaucoma patients with a GDD may show a motility disturbance, possibly resulting in diplopia. In general, the oculomotor system shows remarkable plasticity to changes, caused by growth, aging, trauma, and asymmetrical visual input caused by anisometropia, for example.[5] Following surgery in the orbit, such as placing a GDD, it is likely that the oculomotor system needs to adjust. When the demands for oculomotor plasticity are too high, motility disturbances and even diplopia may occur.

By contrast, trabeculectomy (TE), an alternative surgical procedure for lowering the intraocular pressure (IOP) in the management of glaucoma, shows a lower incidence of diplopia after surgery.[2,3]

Previous studies into the incidence of diplopia after GDD implantation have been quite variable in design and results. A recent study[3] assessed the incidence of diplopia after glaucoma surgery (either GDD or TE) by use of a diplopia questionnaire and revealed a 23% incidence after GDD placement and 3% after TE

surgery. Unfortunately, no ocular motility measurements were conducted in this study to quantify the motility disturbances. A retrospective study[6] showed that 1 year postoperatively, 1.4% of the patients had developed diplopia secondary to GDD implantation. The analysis was based on a chart review. Any cases of diplopia that had not been explicitly stated in the medical records were probably missed.

In the Tube versus Trabeculectomy (TVT) study,[2] 1 year after surgery, persistent diplopia was reported in 5% of the patients in the group with a BGI and no patients in the TE group. Postoperative motility disturbances developed or worsened in 9.9% of the BGI patients. Patients studied in the TVT study had had previous ocular surgery (TE or cataract surgery). As GDDs, instead of TE, are more often used in a primary pressure lowering surgical procedure,[7,8] we wanted to know the effect of primary BGI implantation (i.e., BGI implantation in patients without previous ocular surgery).

We therefore performed a prospective study into the presence of diplopia and any motility changes after glaucoma surgery (TE or BGI). None of the patients had had previous ocular surgery that could affect eye movements. The preoperative visit included an extensive orthoptic examination and was repeated 1 year after surgery.

Methods

Study Design

The study protocol has been described previously in detail.[9] Briefly, 119 glaucoma patients with no history of intra- or extraocular surgery were enrolled in a prospective cohort study at the Rotterdam Eye Hospital, The Netherlands. The study was approved by the medical ethics committee of the Erasmus Medical Center (Rotterdam, The Netherlands), and was registered in at www.trialregister.nl (identifier NTR1142). The study protocol adhered to the tenets of the Declaration of Helsinki. Exclusion criteria consisted of a history of strabismus, non-Caucasian ethnicity, best-corrected Snellen visual acuity less than 20/200 in one or both eyes, or previous ocular surgery (e.g., cataract surgery, trabeculectomy, or strabismus surgery). We assigned patients randomly to either TE or BGI. The primary outcomes consisted of IOP and failure rate[9]; secondary outcomes were diplopia and ocular motility. The latter are the subjects of this paper.

Surgical Procedure

Sixty patients underwent a TE with Mitomycin-C (MMC) superotemporally. The procedure has been described in detail elsewhere.[9] In short, a limbus-based conjunctival flap was created, and sponges soaked with MMC (0.2 mg/mL) were applied for 1 minute to the sclera. The tissue was then copiously rinsed with saline. A scleral flap was fashioned and a limbal block was removed (1.5 mm in diameter) from underneath the scleral flap with a Crozafon-De Laage punch (Moria, Paris, France). Following a peripheral iridotomy, the scleral flap was then sutured with three to four interrupted nylon 10.0 sutures. The conjunctiva and Tenon's capsule were closed with a running suture (nylon 10.0). Postoperative laser suture lysis was performed at the surgeon's discretion in case of inadequate filtration.

Fifty-nine patients received a BGI (BG-101-350 mm^2; Advanced Medical Optics Inc. Santa Ana, CA). The surgical procedure has been described in detail before.[9] Briefly, the BGI plate was placed in the superotemporal quadrant underneath the lateral and superior rectus muscles and was sutured to the sclera. The tube was occluded with a single vicryl 7.0 suture and sized to fit in the anterior chamber of the patients. The surgeon made an entry into the anterior chamber behind the limbus and positioned the tip of the tube at a maximal distance of the corneal endothelium, anterior to the iris. The tube was then covered with a graft of donor sclera, which was sutured to the recipient sclera with interrupted vicryl 7.0 sutures. The surgeon closed the conjunctiva and Tenon's capsule with a running suture (vicryl 7.0). At the end of both surgical procedures, a Celestone Chronodose (betamethasone phosphate/betamethasone acetate; Merck Sharp & Dohme bv, Haarlem, The Netherlands) injection was administered in the inferior subconjunctival space and Dexamytrex (Dexamethasone/gentamycin; Bausch & Lomb, Bridgewater, NJ) eye ointment was applied in the eye before closing with an eye bandage. The postoperative regimen also consisted of local steroids that were tapered over a period of 3 months. IOP-lowering medication was prescribed as required at the surgeon's discretion.

Diplopia

To quantify any diplopia, we asked patients about the presence of any diplopia 1 year after surgery. When diplopia was reported, we determined the severity of this complication as follows: the patient

experienced diplopia in one or more gaze directions (gaze-evoked diplopia); the patient had diplopia in the primary and/or reading position.[10] Patients with diplopia in the primary and/or reading position could experience this diplopia either intermittently or continuously.

Motility Changes

To quantify any ocular motility changes after surgery, we measured the ductions, ocular alignment, and fusion range of both eyes in each patient. The measurements were performed before and 1 year after surgery.

The ductions of both eyes were measured in eight gaze directions by means of a synoptophore; these were elevation, depression, adduction, abduction, elevation in 25° abduction, elevation in 25° adduction, depression in 25° abduction, and depression in 25° adduction. A duction change between preoperative and 1 year postoperative measurements was defined as 2° or more as measured with the synoptophore. This level was based on the repeatability of motility measurements.[2,11] The maximum range of the synoptophore is 40° (in abduction and adduction) and 30° (all other directions). We assumed that there were no duction limitations beyond these extremes. A patient could have a change in more than one direction.

We evaluated the patients' ocular alignment at near fixation with the alternate prism cover test with the patient fixating at 30-cm distance. A change in ocular alignment between preoperative and 1 year postoperative measurements was defined as 4 prism diopters (PD) or more.[2] Additionally, ocular alignment with the patient fixating at distance was measured with the synoptophore. The horizontal and vertical fusion range was also determined with the synoptophore.

Visual Acuity and Fields

We assessed the best-corrected visual acuity by using an Early Treatment Diabetic Retinopathy Study (ETDRS) chart. We also examined the visual fields by white-on-white standard automated perimetry with the Humphrey Field Analyzer 24-2 SITA standard programme (HFA; Carl Zeiss Meditec, Dublin, CA) for each eye. Hence, we knew if the patient had any visual field loss in the operated and/or fellow eye that could affect any diplopia.

Statistical Analysis

A univariate analysis was performed by using the unpaired *t*-test for independent samples and a paired *t*-test for dependent samples with a normal distribution or the Mann-Whitney *U* test for independent samples with nonnormal distribution, such as motility changes. Categoric variables, such as diplopia, were evaluated with Fisher's exact test. We analyzed the results between the BGI and the TE group (unpaired test) and between the visits before and after surgery within the groups (paired test). All statistical calculations were done in R (R Core Team (2013) R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. URL http://www.R-project.org/.)

Results

Baseline

Between July 2008 and September 2014, 119 patients were randomized in either the TE group or the BGI group. However, some patients received a BGI in the fellow eye during the first year of follow-up, which might confound the results. They were therefore excluded from further analysis. Another three TE patients were lost to follow-up after the inclusion and one TE patient and one BGI patient already experienced diplopia before surgery. After excluding these patients, 103 patients remained for analysis (Table 1).

We found no statistically significant differences in patient characteristics at baseline between the two groups (Table 2). We found duction restrictions in both groups before surgery. For our study, we focused on surgically induced changes.

At baseline, we found a horizontal ocular devia-

Table 1. Number of Patients in Both Treatment Groups, Before and After Exclusions

	TE Group	BGI Group
Before exclusions	60	59
Exclusions due to		
Diplopia before surgery	−1	−1
BGI in fellow eye within first year of follow-up	−4	−7
Inadequate follow-up	−3	0
Final group size after exclusions	52	51

Table 2. Preoperative Characteristics of the TE Group and BGI Group

	TE Patients $n = 52$	BGI Patients $n = 51$	P Value Between the Groups
Age (y), mean ± SD	60.7 ± 7.6	60.3 ± 10.7	0.79[a]
Sex (n) male:female	30:22	27:24	
Study eye OD:OS	25:27	28:23	
Previous ocular surgeries, mean ± SD	0	0	NA
Diplopia level			
No diplopia	52 (100%)	51 (100%)	NA
In gaze direction(s)	0	0	
In Primary position and/or reading position	0	0	
No duction restriction *n*	36 (69%)	45 (88%)	
Duction restriction, *n*: (a patient can have an restriction in >1 direction)	13 (25%)	5 (10%)	
Abduction	3	0	0.61[b]
Adduction	2	1	0.18[b]
Elevation	7	3	0.39[b]
Depression	0	0	0.99[b]
Elevation in 25° abduction	10	4	0.87[b]
Elevation in 25° adduction	9	4	0.37[b]
Depression in 25° abduction	0	0	0.10[b]
Depression in 25° adduction	0	0	0.35[b]
Unknown	3 (6%)	1 (2%)	
ETDRS VA study eye, mean ± SD	0.0 ± 0 0.2	0.1 ± 0.3	0.72[a]
Humphrey visual fields			
MD study eye, mean ± SD	−11.7 ± 8.8	−13.4 ± 8.9	0.31[a]
PSD study eye, mean ± SD	8.5 ± 4.6	9.0 ± 3.6	
MD fellow eye, mean ± SD	−6.1 ± 6.7	−6.7 ± 6.2	0.71[a]
PSD fellow eye, mean ± SD	6.4 ± 4.6	6.8 ± 4.2	
Ocular alignment deviation			
Horizontal, orthophoric			
Near (PD)	32 (61%)	31 (60%)	0.90[b]
Distance (°)	33 (63%)	36 (70%)	0.16[b]
Horizontal, esodeviation			
Near (PD)	1 (2%)	0	0.90[b]
Distance (°)	18 (35%)	8 (16%)	0.16[b]
Horizontal, exodeviation			
Near (PD)	16 (31%)	19 (37%)	0.90[b]
Distance (°)	1 (2%)	5 (10%)	0.16[b]
Vertical, orthophoric			
Near (PD)	50 (96%)	50 (98%)	0.44[b]
Distance (°)	50 (96%)	50 (98%)	0.68[b]
Vertical, deviation			
Near (PD)	1 (2%)	1 (2%)	0.44[b]
Distance (°)	1 (2%)	1 (2%)	0.68[b]
Unknown	1 (2%)	1 (2%)	

Table 2. Continued

	TE Patients $n = 52$	BGI Patients $n = 51$	P Value Between the Groups
Fusion range			
Horizontal (°)			
0–10	3 (6%)	5 (10%)	0.27[b]
11–20	26 (50%)	16 (31%)	
21–30	13 (25%)	12 (23%)	
>30	6 (12%)	15 (29%)	
Unknown	4 (7%)	4 (7%)	
Vertical (°)			
0–3	9 (17%)	10 (20%)	0.98[b]
4–6	33 (63%)	29 (57%)	
>6	4 (8%)	8 (16%)	
Unknown	6 (12%)	5 (10%)	

[a] Paired Student's *t*-test
[b] Mann-Whitney *U* test.

tion at near fixation of −2.3 ± 3.4 PD (mean ± standard deviation [SD]) in the TE group and of −2.6 ± 3.6 PD (mean ± SD) in the BGI group. The vertical ocular alignment at near fixation was −0.1 ± 0.4 PD (mean ± SD) in the TE group and −0.1 ± 0.5 PD (mean ± SD) in the BGI group. All deviations in ocular alignment were latent deviations (phorias).

Diplopia

One year after surgery, significantly more patients experienced diplopia in the BGI group than in the TE group (<0.001; Fisher's exact test; Table 3). Only one TE patient mentioned diplopia, which occurred in primary position and/or the reading position. In the BGI group, however, 14 (27%) of the patients experienced diplopia 1 year after surgery; six of those patients had gaze-evoked diplopia and eight patients had diplopia in primary position (3 patients) or in the reading position (5 patients). Only one of 14 patients experienced the diplopia continuously (in primary position).

Ductions

BGI Group Versus TE Group

Changes in ductions before and 1 year after surgery in the operated eye were more commonly observed in the BGI group (35%) than in the TE group (19%; Table 3). The duction changes in the BGI group, compared to the TE group, were statistically significant in abduction ($P = 0.02$), in elevation ($P = 0.002$), and in elevation in 25° abduction ($P = 0.005$; Table 3). As for the fellow eye, no significant duction changes were observed in the two groups.

BGI Group

The changes in ductions in the BGI patients, 1 year postoperatively, compared with before surgery, were mostly restricted in elevation (13%; $P < 0.001$), in abduction (13%; $P = 0.09$), in elevation in 25° adduction (13%; $P = 0.044$), and in elevation in 25° abduction (25%; $P < 0.001$) (Fig. 1; Table 3). Only few restrictions were found in adduction (8%; $P = 0.26$) and depression (2%; $P = 0.48$).

The observed duction limitations were independent of diplopia (Fig. 1). All patients with gaze-evoked diplopia (6 patients) had reduced ductions. Two of eight patients with diplopia in primary position or the reading position had one or more reduced ductions. In the nondiplopia group, 10 of 37 patients had reduced ductions.

Ocular Alignment and Fusion Range

BGI Group Versus TE Group

Both groups had a similar number of patients with a change in horizontal ocular alignment (Table 3). A shift of the ocular alignment occurred more commonly in the exodirection than in the esodirection. As for the fusion range, patients with a BGI showed a larger loss in horizontal fusion range than did the TE patients (Table 3).

Table 3. Changes in BGI Group Versus TE Group

	TE Patients $n = 52$	*P* Value Before and After Surgery
Diplopia level *n*		NA
No diplopia	51 (98%)	
In gaze direction(s)	0	
In primary position and/or reading position	1 (2%)	
No duction changes *n*	40 (77%)	
Duction changes *n*	10 (19%)	
Changes overall group: (a patient could have a change in >1 direction)		
Δ Abduction mean ± SD (°)	0.3 ± 1.3	0.19[c]
Δ Adduction mean ± SD (°)	0.5 ± 2.6	0.19[c]
Δ Elevation mean ± SD (°)	0.4 ± 2.6	0.48[c]
Δ Depression mean ± SD (°)	0.0 ± 0.2	0.30[c]
Δ Elevation in 25° abduction mean ± SD (°)	0.5 ± 2.9	0.44[c]
Δ Elevation in 25° adduction mean ± SD (°)	0.9 ± 3.4	0.32[c]
Δ Depression in 25° abduction mean ± SD (°)	0.3 ± 3.5	0.71[c]
Δ Depression in 25° adduction mean ± SD (°)	−0.1 ± 0.2	0.15[c]
Unknown *n*	2 (4%)	
Changes in ETDRS VA, Δ mean ± SD	0.04 ± 0.1	**0.034**[d]
Changes in Humphrey visual fields		
Δ MD study eye, mean ± SD	−0.7 ± 1.9	**0.008**[d]
Δ PSD study eye, mean ± SD	0.4 ± 2.2	0.46[d]
MD fellow eye, mean ± SD	−0.9 ± 2.7	**0.03**[d]
PSD fellow eye, mean ± SD	0.3 ± 1.5	0.18[d]
Ocular alignment deviation		
Δ Near, horizontal (PD)		0.41[c]
No change *n*	24 (46%)	
Change exodirection *n* (mean ± SD)	13 (25%) (−6.5 ± 3.5)	
Change esodirection *n* (mean ± SD)	1 (2%) (4.0 ± 0.0)	
Unknown *n*	14 (27%)	
Δ Near, vertical (PD)		0.10[c]
No change *n*	37 (71%)	
Change *n* (mean ± SD)	3 (6%) (3.67 ± 1.53)	
Unknown *n*	12 (23%)	
Δ Distance, horizontal (°)		0.30[c]
No change *n*	14 (27%)	
Change exodirection *n* (mean ± SD)	6 (11%) (−1.6 ± 0.8)	
Change esodirection *n* (mean ± SD)	18 (35%) (2.9 ± 2.0)	
Unknown *n*	14 (27%)	
Δ Distance, vertical (°)		1.00[c]
No change *n*	33 (63%)	
Change *n* (mean ± SD)	5 (10%) (1.6 ± 0.9)	
Unknown *n*	14 (27%)	

Table 3. Continued

	TE Patients $n = 52$	*P* Value Before and After Surgery
Fusion range		
Horizontal (°)		0.94[c]
Decrease *n* (mean ± SD)	19 (36%) (−4.8 ± 2.6)	
No change *n*	1 (2%)	
Increase *n* (mean ± SD)	17 (33%) (9.9 ± 13.1)	
Unknown *n*	15 (29%)	
Vertical (°)		0.15[c]
Decrease *n* (mean ± SD)	12 (23%) (−1.4 ± 0.6)	
No change *n*	7 (13%)	
Increase *n* (mean ± SD)	18 (35%) (2.1 ± 1.5)	
Unknown *n*	15 (29%)	

[a] Fisher's exact test.
[b] Wilcoxon rank-sum test.
[c] Wilcoxon signed-rank test.
[d] Paired *t*-test.
[e] Unpaired *t*-test. A *P*-value of 0.05 or smaller was considered significant (bold values).

Table 3. Extended

	BGI Patients $n = 51$	*P* Value Before and After Surgery	*P* Value in Change Between the Groups
Diplopia level *n*		NA	<**0.001**[a]
No diplopia	37 (72%)		
In gaze direction(s)	6 (12%)		
In primary position and/or reading position	8 (16%)		
No duction changes *n*	26 (51%)		
Duction changes *n*	18 (35%)		
Changes overall group: (a patient could have a change in >1 direction)			
Δ Abduction mean ± SD (°)	−0.5 ± 2.1	0.09[c]	**0.02**[b]
Δ Adduction mean ± SD (°)	0.3 ± 1.8	0.26[c]	0.50[b]
Δ Elevation mean ± SD (°)	−1.9 ± 4.2	<**0.001**[c]	**0.002**[b]
Δ Depression mean ± SD (°)	0.0 ± 0.0	1.00[c]	0.07[b]
Δ Elevation in 25° abduction mean ± SD (°)	−2.3 ± 4.0	<**0.001**[c]	**0.005**[b]
Δ Elevation in 25° adduction mean ± SD (°)	−1.4 ± 4.5	**0.044**[c]	0.05[b]
Δ Depression in 25° abduction mean ± SD (°)	0.0 ± 0.3	1.00[c]	0.19[b]
Δ Depression in 25° adduction mean ± SD (°)	0.1 ± 0.9	0.48[c]	0.19[b]
Unknown	7 (14%)		
Changes in ETDRS VA, Δ mean ± SD	0.02 ± 0.1	0.29[d]	0.52[e]
Changes in Humphrey visual fields			
Δ MD study eye, mean ± SD	−0.7 ± 2.7	0.08[d]	0.94[e]
Δ PSD study eye, mean ± SD	−0.2 ± 108	0.23[d]	0.16[e]
MD fellow eye, mean ± SD	−0.5 ± 1.9	**0.04**[d]	0.51[e]
PSD fellow eye, mean ± SD	0.0 ± 1.3	0.91[d]	0.37[e]
Ocular alignment deviation			
Δ Near, horizontal (PD)		**0.04**[c]	0.11[b]
No change *n*	35 (68%)		
Change exodirection *n* (mean ± SD)	7 (14%) (−9.5 ± 5.9)		
Change esodirection *n* (mean ± SD)	3 (6%) (4.7 ± 1.1)		
Unknown *n*	6 (12%)		
Δ Near, vertical (PD)		**0.01**[c]	0.08[b]
No change *n*	39 (76%)		
Change *n* (mean ± SD)	7 (12%) (4.14 ± 4.33)		
Unknown *n*	5 (10%)		
Δ Distance, horizontal (°)		0.30[c]	0.69[b]
No change *n*	39 (76%)		
Change exodirection *n* (mean ± SD)	2 (4%) (−9.5 ± 7.8)		
Change esodirection *n* (mean ± SD)	4 (8%) (4.6 ± 1.4)		
Unknown *n*	6 (12%)		
Δ Distance, vertical (°)		**0.004**[c]	0.39[b]
No change *n*	35 (68%)		
Change *n* (mean ± SD)	9 (16%) (2.05 ± 1.8)		
Unknown *n*	7 (14%)		

Table 3. Extended, Continued

	BGI Patients $n = 51$	*P* Value Before and After Surgery	*P* Value in Change Between the Groups
Fusion range			
Horizontal (°)		**0.01**[c]	0.06[b]
Decrease *n* (mean ± SD)	23 (45%) (−12.6 ± 10.3)		
No change *n*	3 (6%)		
Increase *n* (mean ± SD)	16 (31%) (7.2 ± 8.6)		
Unknown *n*	9 (18%)		
Vertical (°)		0.31[c]	0.22[b]
Decrease *n* (mean ± SD)	15 (29%) (−1.9 ± 1.1)		
No change *n*	12 (23%)		
Increase *n* (mean ± SD)	11 (21%) (1.6 ± 0.9)		
Unknown *n*	13 (25%)		

[a] Fisher's exact test.
[b] Wilcoxon rank-sum test.
[c] Wilcoxon signed-rank test.
[d] Paired *t*-test.
[e] Unpaired *t*-test. A Pue of 0.05 or smaller was considered significant (bold values).

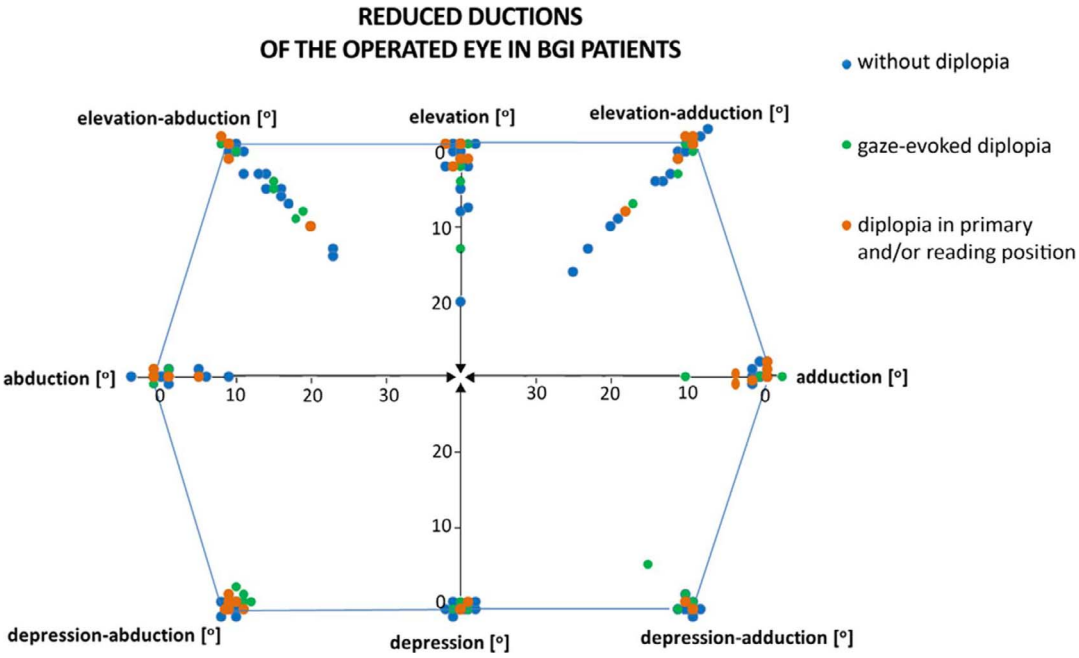

Figure 1. Changes in duction between baseline and 1 year after surgery (represented by *dots*) in the operated eye in the BGI group. Each patient is represented by eight dots, one dot for each gaze direction. A *dot* on the *hexagon* represents no restrictions in that direction. We assumed no reduced duction when the patient reached the same amount of degrees at baseline as at 1 year after surgery. Hence, all *dots* within the *hexagon* represent a reduced duction in that direction.

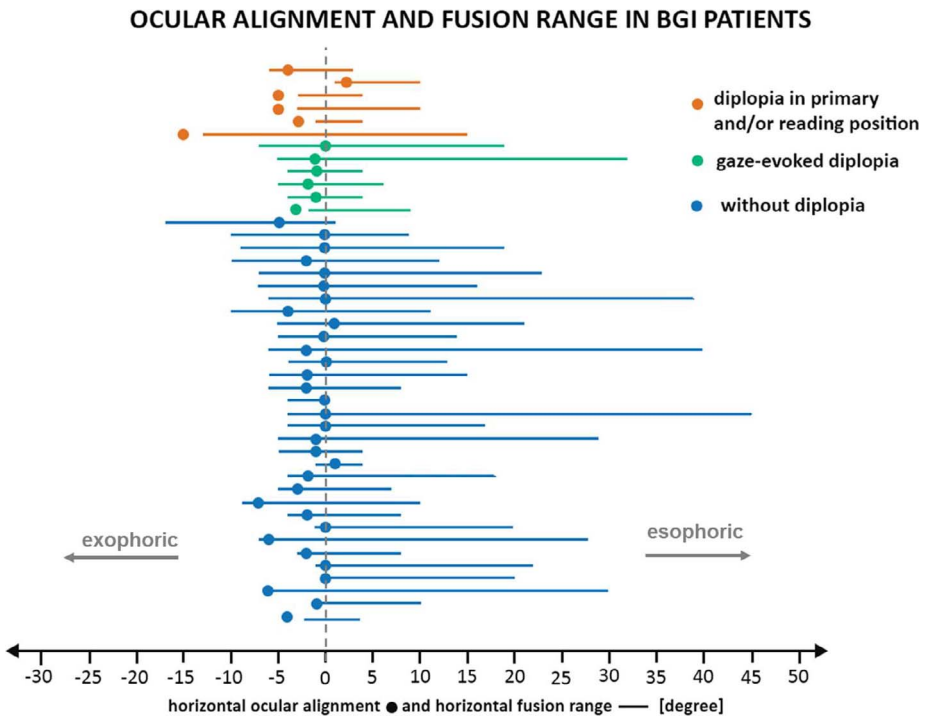

Figure 2. Ocular alignment in horizontal direction at 30 cm (marked as *dots* in the graph, with 2 PD = 1°) and horizontal fusion range (the range of the fusion is represented by a *line*) measured with the synoptophore at distance 1 year after surgery. Negative numbers reflect exophoria, positive numbers represent esophoria. When the ocular alignment is 0°, there is no phoria present.

BGI Group

At 1 year after surgery, 68% of the patients did not show any change in their near horizontal ocular alignment (Table 3). However, in the patients with a change in their near horizontal ocular alignment, the ocular alignment shifted notably in exodirection ($P = 0.04$). At distance, the ocular alignment showed an increase in vertical deviation, in which the operated eye could deviate in downward or upward position ($P = 0.02$; Table 3).

The fusion range decreased significantly in the horizontal direction (−12.6° ± 10.3°, mean ± SD; $P = 0.01$; Table 3). The fusion range enables one to cope with any deviation in ocular alignment. Figure 2 combines both parameters in the horizontal direction for the BGI group. When the ocular alignment (the dot) falls outside the range of the fusion (the line), diplopia would be likely. Of 37 patients without diplopia, only one (2.7%) had an ocular deviation (in primary position at 30 cm) exceeding his/her horizontal fusion range measured at distance. This patient had advanced visual field loss in the operated eye (mean deviation [MD] of −16.6 dB 1 year after surgery).

In the group with gaze-evoked diplopia, only one of six patients (16%) had an ocular deviation exceeding his horizontal fusion range.

Four of eight patients with diplopia in primary position and/or the reading position had an ocular deviation exceeding the capabilities of their fusion range in the horizontal direction (Fig. 2). Of two diplopia patients in primary position and/or the reading position whose horizontal ocular deviation fell within their horizontal fusion range, one had a large vertical ocular deviation (of 3°).

We were not able to measure the fusion range of four patients without diplopia and two patients with diplopia in primary position and/or reading position, and they have therefore not been represented in Figure 2.

Visual Field

BGI Group Versus TE Group

The MD of the visual fields of both groups worsened over the 1 year postoperative follow-up period (Table 3). No statistically significant difference in change was found between the two treatment groups.

BGI Group

Eighty-six percent of the patients that experienced diplopia (12/14 patients) had mild glaucomatous visual field loss[12] in one or both eye(s), while this was observed in only 57% of the patients without diplopia (Fig. 3). Figure 3 shows a tendency toward a higher incidence of

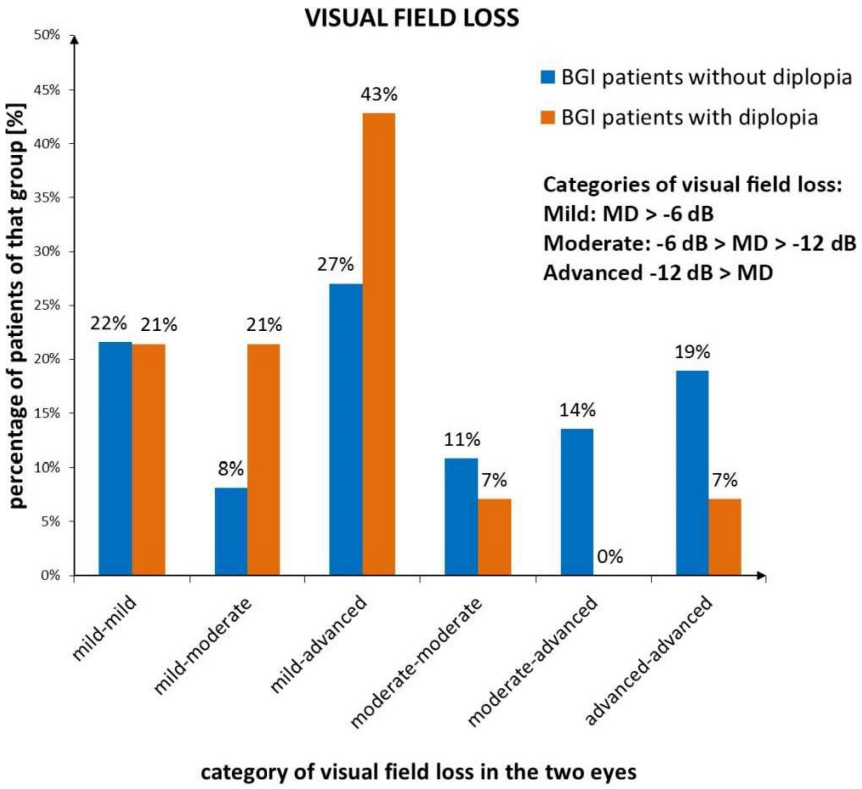

Figure 3. MD divided into three visual field loss categories (mild, moderate, and advanced, after Hodapp-Parrish-Anderson[12]). This results in six combinations of the visual field loss in the two eyes.

diplopia in patients with mild glaucomatous visual field loss in one or both eye(s) (12/14 patients with diplopia versus 21/37 patients without diplopia; $P = 0.056$). However, both percentages showed a wide confidence interval (for patients without diplopia 41%–71%; for patients with diplopia 60%–96%).

Discussion

The results of our study showed that the risk of developing diplopia and reduced ductions was much higher after BGI than after TE, which agrees with previous studies.[2–6] The reduced ductions were most prominent in the upward gaze directions, some in outward directions, and almost none in downward directions.

The TVT study[2] described the incidence of diplopia and/or motility changes in patients with previous ocular surgery during 1 year of follow-up. There were no diplopia cases in the TE group of the TVT study, which is consistent with our results. In the BGI group, we found a large difference in diplopia results between the TVT study (5%) and our study (28%). The difference in study group could clarify this dissimilarity. The TVT study included monocular patients (29% of their BGI patients) and patients with strabismus, while these were excluded from our study.[13,14]

Contrary to the BGI group in our study (22% motility changes), the TVT study found that only 9.9% of the BGI patients had motility changes 1 year after surgery. An explanation for their lower incidence may be differences in measurement technique. By measuring the change in ocular alignment in only four gaze directions, they were bound to miss any ocular alignment changes in the other four (tertiary) gaze directions. We based our ocular motility on the duction changes in eight gaze directions. We found that elevation and adduction showed duction restrictions, but also in elevation in 25° abduction and elevation in 25° adduction.

Because diplopia did not occur in our TE group, we will only discuss the occurrence of diplopia in our BGI group. Table 4 will be used as a guideline throughout this discussion in an attempt to reveal the possible explanation(s) for the presence/absence of diplopia.

All patients with gaze-evoked diplopia had one or more reduced ductions (Table 4). A reduced duction results in one eye staying behind in the movement in that direction. For binocular single vision, the eyes have to move in unison. A reduced duction disrupts

Table 4. Overview of Results and Possible Explanation(s) for the Presence/Absence of Diplopia

Diplopia	Patients	Details
Gaze-evoked	6	All 6 patients had reduced duction(s)
In primary position	3	All 3 patients had their ocular alignment exceeding their fusion range
In reading position	5	Possibly undetected reduced ductions in depression, due to measuring limitation of 30° by synoptophore or ocular alignments in depression exceeding their fusion range
No diplopia	37	22 patients had no reduced ductions or ocular alignment exceeding their fusion range 7 had advanced visual field loss in at least one eye 1 patient had central visual field loss 7 unknown

the binocular single vision. The image that the fovea of one eye receives will fall in an extra-foveal area of the retina of the fellow eye, resulting in diplopia.[15] Hence, one would expect diplopia to occur when gazing into the direction of the reduced duction.

The ductions were mostly restricted in the outward and upward directions. We think that placing the plate of the BGI underneath the ocular muscles, responsible for elevation and/or abduction, may limit their functioning. It is unclear whether scar tissue formation and/or the volume of the plate and its fluid-filled capsule mechanically hinder the free movements of the globe, or whether the contraction of the involved muscles itself is restricted directly, for instance by scarring.

No patients with diplopia in the primary position had reduced duction(s) (Table 4). This indicates that the diplopia was caused by something else. The tendency of eyes to deviate from bifoveal fixation (phoria) is controlled by the fusional vergence.[16] The larger the phoria the more difficult it is to obtain or maintain binocular, fused vision. When a heterotropia is present, the degree of misalignment exceeds the capabilities of fusional vergence. In general, people suffering from diplopia in primary position often have a smaller fusion range and a larger ocular deviation.[16] In our study, all patients with diplopia in primary position had a deviation in their ocular alignment (horizontal and/or vertical) exceeding their fusion range.

Costa Lanca and Rowe[16] have suggested that patients with a reduced fusion range have a higher risk of developing diplopia. Combined with our results, this would suggest that patients with a small fusion range and up to moderate visual field loss in either eye have an increased risk of developing diplopia after BGI surgery. As future work, we may suggest that it is worth exploring the preoperative fusion range as a possible predictor for diplopia. It may also be of interest to explore the extent and location of any visual field damage as predictors of diplopia.

Although almost no reduced ductions were measured in depression, five patients did experience diplopia in the reading position, which typically requires depression of both eyes. A possible explanation could be that we did not find impaired ductions in these directions due to the limitations of the synoptophore, which cannot measure depressions beyond 30°, although the downward ocular movement may reach 50°.[11,17] The Goldmann perimeter can measure up to 70° in all gaze directions, which might have been a better choice to detect reduced depressions.

The overwhelming majority of the BGI patients did not experience diplopia. However, some of them did have reduced duction(s). We propose two possible explanations for not having diplopia, despite the reduced ductions.

First, the patient may have a reduced duction but also a visual field loss in either eye, which suppresses the diplopic image (Table 4). Previous studies[18,19] suggested that patients with advanced visual field loss have a higher risk of developing diplopia. The visual field defect could result in a deterioration of the stimulus for binocular single vision. In turn, this could lead to decompensation as it is no longer possible to maintain the binocular single vision. However, our study did not confirm this theory, but showed an opposite tendency (i.e., that patients with moderate to advanced visual field loss in either eye rarely experienced diplopia).

Secondly, the patient did not have reduced duction(s) but due to the patients' visual field loss in that direction, the small picture in the synoptophore was difficult to follow and resulted in a false reduced duction measurement.

Finally, we found that elevation was one of the most restricted ductions. Elevation is probably less commonly used in daily life. Patients may therefore simply not experience diplopia, which can explain the absence of reported diplopia in some patients.

In conclusion, our data confirm that people experience diplopia significantly more commonly following a BDI than following a TE. We also observed markedly impaired ductions in many of the eyes that had undergone BDI, mostly in abduction, elevation, elevation in 25° abduction, and elevation in 25° adduction, and few in adduction and depression. Even without impaired ductions, diplopia could come about; however, and this happened notably when the ocular alignment was outside the fusion range.

Patients need to be aware of the risk of diplopia associated with BGI surgery. Diplopia interferes with the patients' daily activities, like driving or working, therefore it is important to lower the risk of developing diplopia. More research is necessary to minimize this postoperative complication.

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