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MINIREVIEWS

Binocular disturbance after glaucoma drainage device implantation

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Abstract

Binocular vision disturbance is a well-described complication of glaucoma drainage device (GDD) implantation. The pathophysiology is not well-understood, but may involve bulk effects from the implant and surrounding bleb, as well as modulation of muscle function due to surgical trauma and post-operative inflammation, resulting in a combined resection/posterior fixation effect. Retrospective studies have found the risks of motility disorder and diplopia vary widely, estimated to be 56%-86% and 57%-75%, respectively. More recently, cross-sectional studies and prospective trials estimate post-GDD incidence to be approximately 1%-44%, with the incidence in newer generation of implants designed to limit bleb size likely lower at 1%-5%. Suggested methods of management strategies include prismatic spectacles, monocular occlusion, extreme monovision, and strabismus surgery.

Key words: Glaucoma; Drainage; Implant; Device; Diplopia; Motility; Binocular; Disturbance; Strabismus

Core tip: The reported incidence of binocular disturbance after glaucoma drainage device (GDD) implantation is variable due to inconsistent study designs, disturbance definition and lack of pre-operative baseline evaluations. The incidence of motility disorder is likely higher than persistent diplopia, as some glaucoma patients requiring GDD are functionally monocular. The mechanism or disturbance is not well-understood, but the bulk of implant/bleb, changes in muscle length, tension and strength may result in a combined resection/posterior-fixation effect. Post-GDD diplopia may resolve spontaneously in some instances, while the intractable cases are usually managed with prismatic spectacles.

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INTRODUCTION

New onset, persistent binocular disturbance is a well-described complication of glaucoma drainage device (GDD) implantation[1]. Studies estimate the risk of post-GDD binocular disturbance to be between 2%-18%[2-10]. Broad categories such as "strabismus," "diplopia," "motility disorder," and "motility disturbance" are used to capture all cases of post-operative binocular disturbances without articulating their natures. The incidences of post-GDD binocular disturbance are often reported as complications in prospective trials or retrospective case series designed to assess the implants' efficacy in controlling intraocular pressure. Additionally, most of these studies lack rigorous pre-operative motility and binocular function evaluation, and the reports of post-operative binocular disturbances are often descriptive with no quantitative measurements. Assuming that the motility evaluations in these studies were triggered by the patients' complaint of diplopia, it

may be reasonable to assume that the true incidence of diplopia (especially if intermittent) and asymptomatic motility disturbances to be underestimated. Furthermore, intentional or unintentional post-operative anisometropia may result in decompensation of long-standing phoria and diplopia, which should not be attributed to the glaucoma drainage device implantation.

PATHOPHYSIOLOGY

The pathophysiology of binocular disturbance after GDD implantation is not well-understood, and the proposed mechanisms would include bulk effect, paresis, posterior fixation effect, and mechanical restriction. Earlier case reports suggest the implant or a large filtering bleb around the implant exerts a bulk effect on the globe, causing duction limitations in the direction of the implant[11-16]. Approximately 36% of patients in a retrospective series of double-plated Molteno implants had paretic strabismus in the muscle or muscles concordant to the quadrants of the implant. This suggests that muscle manipulation may result in injuries and paresis, and secondarily motility disturbance[17]. In other reports, a heterotropic, post-GDD eye deviates toward the implant 46%-100% of the time[1,17-21], implying a restrictive mechanism. During strabismus surgery to correct post-GDD strabismus, Roizen *et al*[22] noted uniformly restricted forced duction tests and presence of thick, fibrous capsule surrounding the implant and adjacent muscles, regardless of pattern of motility disorder. It is plausible that after the GDD implantation, the post-surgical inflammatory changes, development of the bleb and presence of muscle injury result in altered muscle length-tension relationship as well as a posterior fixation effect. Glaucomatous visual field loss may increase the risk of binocular disturbance due to brittle fusional ability from damaged peri-foveal visual fields and reduced binocular stimulation. This may suggest that patients with long-standing strabismus (presence of suppression) and/or greater visual field loss may be at higher risk of post-operative binocular disturbance. However, the tube versus trabeculectomy (TVT) study found that the mean deviation on automated visual field and prevalence of preoperative motility disturbance did not differ between those who had new-onset diplopia after GDD compared to those who did not, possibly due to insufficient power[20].

INCIDENCE OF POST-GDD BINOCULAR DISTURBANCES IN ADULTS

Binocular disturbance includes motility disorder, heterotropia and binocular diplopia, which describe a spectrum of dysfunctions ranging from limited ductional deficits to disrupted binocular cooperation. These entities can exist alone or, more frequently, in combination. General ophthalmic surgical approaches, especially when involving a peri- or retrobulbar infiltrate of local anesthetic agents, may result in binocular disturbances even when the extraocular muscles are not manipulated, although the risk is likely small. This provides a context of background incidence in which the incidence attributed to GDD can be elucidated. In a retrospective review of 20453 cataract cases performed under retrobulbar block with ropivacaine diluted with hyaluronidase, persistent diplopia was noted in 19 (0.093%) patients[23]. A similar survey of 2024 patients who had undergone cataract surgery with peri- or retrobulbar block yields an overall incidence of 0.25%[24]. Neither study includes a pre-operative assessment of motility and binocular function, but the reported incidences are adequate estimates of diplopia after procedures involving peri- or retrobulbar block anesthesia.

Retrospective studies on post-GDD binocular disturbance are often case series of consecutive glaucoma patients receiving implants or cross-sectional studies of diplopic patients referred to strabismus clinic who have previously received GDD implantation. In both scenarios, the patients originate from the glaucoma service and had undergone strabismus evaluation only after the onset of binocular disturbance, making baseline motility and binocular function tests rarely available. Frank *et al*[18] reviewed 7 patients who had undergone Krupin valve implantation, and found four patients (57%) with intermittent or constant diplopia, with the other three patients being functionally monocular. Six of the seven patients (86%) had significant deviation in primary position post-operatively[18]. Smith *et al*[21] described 37 eyes of 36 patients that had received Baerveldt glaucoma implant, with 5 of the 36 patient having documented motility disturbance and none with diplopia. Post-operatively, 23 of 30 eyes (77%) with adequate motility follow-up demonstrated motility restriction, and 11 of 17 (65%) binocular patients experienced diplopia[21]. It is not clear whether any or all of the 5 patients with pre-existing binocular disturbance were included in the follow-up. Wilson-Holt et al reported 16 eyes of 16 patients who had inferior surgical implantation of double-plate Molteno tubes and found 9 of the 16 patients (56%) developed a significant hypertropia, which averaged 8.9 prism diopters (range 2-15 prism diopters). The time of onset of diplopia and hypertropia after tube surgery ranged from 1 to 4 mo. All patients showed restriction on depression of the globe[25].

Taken together, one can infer from these three studies that the risk of motility disorder after GDD implantation in a glaucoma cohort ranges between 56%-86% and risk of diplopia between 57%-75%. Some patients develop heterotropia but not diplopia from being functionally monocular. It should be noted that some of these case series involve older generations of glaucoma drainage devices without modifications to modulate bleb size, thus the risks of motility disturbance and diplopia may be lower today with the newer generation devices.

Looking specifically at a group of patients carrying the diagnosis of "diplopia" or who had procedural codes for strabismus surgery, Abdelaziz *et al*[1] used financial claims information to identify patients who had undergone GDD surgery between 1991 and 2005 at a large ter-

tiary referral center[1]. After review of medical records to exclude diplopia or strabismus surgeries unrelated to the GDD, 1.4% of these patients had persistent, new-onset diplopia attributed to GDD implantation at one year. Despite the meticulous search methodology, the retrospective design and use of financial claims information is likely to underestimate the true incidence of new-onset, persistent diplopia after GDD implantation, especially if the diplopia diagnosis was not submitted for financial claims, or if the patients were lost to follow up.

Few prospective studies evaluated the effect of GDD implantation on motility. In a prospective, consecutive observational series, Dobler-Dixon *et al*[17] performed pre- and post-GDD (double-plated Molteno implant) sensory-motor testing on 24 patients undergoing GDD implantation. The majority had between 1 to 3 prior ocular surgeries. Eight patients (33%) had pre-existing motility disturbance, and 15 patients (63%) were binocular (defined as Snellen visual acuity of 20/70 or better in both eyes). New-onset, persistent motility disorder was noted in 11 of 24 patients (46%) after GDD implantation, 91% of which occurred in binocular patients. Seven of the 16 patients (44%) with normal pre-operative motility developed new-onset, persistent diplopia after GDD implantation, which were confirmed with red glass test. The authors further delineated the mechanism of strabismus to be paretic in 4 of the 11 patients, with high concordance of the paretic muscle being in the same quadrant as the implant, suggesting paresis associated with hardware implantation and intraoperative manipulation of the extraocular muscles during GDD implantation. However, the determination of paretic versus restrictive mechanisms and relative saccade velocities were not reported. The high likelihood of post-GDD motility disturbance in this series compared to the other studies may be attributed to meticulous post-GDD motility evaluations, which makes under-reporting less likely. The implant's double-plated design also requires access to multiple quadrants and larger peritomies, and the surgical technique requires elevation of at least one muscle in order to pass the distal plate underneath to the other quadrant.

The TVT Study included a formal motility evaluation on all patients at pre-operative baseline and at the 1-year follow-up visit[20]. A total of 101 patients were randomized to the tube group, 71% of whom were binocular (defined as Snellen visual acuity of better than 20/200 in both eyes). Pre-operatively, 26% of GDD patients were heterotropic (most commonly exodeviation at near), while only 2% had diplopia. Post-operatively, new-onset persistent diplopia developed in 5% of patients. However, saccade velocity and sensory confirmation of diplopia were not part of the pre- or post-operative evaluation, and the definition of binocularity was broad. The baseline pre-operative prevalence of heterotropia (26%) was much higher than that estimated by a random sampling of Medicare beneficiaries (< 1%) in a comparable age group[26]. This implies that glaucoma diagnosis and history of prior ocular surgeries (cataract extraction, glaucoma filtering procedures) may confer a higher risk of strabismus at baseline compared to the general population. Overall, the study's prospective design, large number of subjects and pre- and post-operative motility assessment makes it one of the more convincing reports on binocular disturbance after GDD implantation.

MANAGEMENT STRATEGIES

Anecdotally, many instances of motility disturbance will resolve without intervention within six months. However, if unresolved, the complex nature of post-operative binocular disturbance may require employment of a number of different strategies. Treatment is complicated by the variability of alignment during the healing process, incomitant nature of the deviations, torsion, and abnormal saccadic velocities.

Prismatic spectacle correction can be used as either a temporizing or a permanent solution, successfully alleviating symptoms in 65% of treated patients[1]. Prism correction can be used to facilitate fusion by aligning the images or by moving the second image further so that it can be suppressed. Its utility as a temporizing measure preceding strabismus surgery to test fusion or as a permanent measure to alleviate diplopia makes it extremely helpful in these complicated cases. The options include press-on Fresnel prisms for variable deviations or ground-in prisms for smaller, stable deviations.

Strabismus surgery may be indicated if the deviation is fairly comitant in a patient with adequate motor fusion; however, the patient must understand the goal of surgery is alleviation of diplopia in primary and reading positions and may not correct misalignment in other directions of gaze. The patient should be aware of the increased risk of compromised intraocular pressure control when operating next to filtering blebs and drainage devices. A multidisciplinary approach involving both strabismus and glaucoma services may increase the likelihood of success and minimize complications.

Alternatively, other surgical strategies include implanting a second implant in the opposite quadrant of the same eye (without or without removal of offending implant) or, when indicated, implanting a GDD in fellow eye under the yoke muscles. This strategy capitalizes on the observation that the implant may result in a combined resection/posterior-fixation effect, and thus decrease heterotropia. Aggressive lysis of adhesion with amniotic membrane grafts around the implant and affected muscles to reduce scarring have some anecdotal success.

Additionally, others have suggested extreme monovision in the form of glasses, contact lenses or intraocular lens implants as means to alleviate persistent diplopia by blurring the unwanted image and allowing suppression[27]. Lastly, while far from ideal, partial or complete occlusion of the involved eye with a patch, tape or foil may be the only option should resolution of the diplopia with alternate methods prove unsuccessful.

REFERENCES

1 **Abdelaziz A**, Capó H, Banitt MR, Schiffman J, Feuer WJ, McKeown CA, Spencer NE, Parrish RK. Diplopia after glaucoma drainage device implantation. *J AAPOS* 2013; **17**: 192-196 [PMID: 23622451 DOI: 10.1016/j.jaapos.2012.11.017]

2 **Ayyala RS**, Zurakowski D, Smith JA, Monshizadeh R, Netland PA, Richards DW, Layden WE. A clinical study of the Ahmed glaucoma valve implant in advanced glaucoma. *Ophthalmology* 1998; **105**: 1968-1976 [PMID: 9787371]

3 **Britt MT**, LaBree LD, Lloyd MA, Minckler DS, Heuer DK, Baerveldt G, Varma R. Randomized clinical trial of the 350-mm2 versus the 500-mm2 Baerveldt implant: longer term results: is bigger better? *Ophthalmology* 1999; **106**: 2312-2318 [PMID: 10599663]

4 **Christakis PG**, Tsai JC, Kalenak JW, Zurakowski D, Cantor LB, Kammer JA, Ahmed II. The Ahmed versus Baerveldt study: three-year treatment outcomes. *Ophthalmology* 2013; **120**: 2232-2240 [PMID: 23796764 DOI: 10.1016/j.ophtha.2013.04.018]

5 **Harbick KH**, Sidoti PA, Budenz DL, Venkatraman A, Bruther M, Grayson DK, Ko A, Yi GN. Outcomes of inferonasal Baerveldt glaucoma drainage implant surgery. *J Glaucoma* 2006; **15**: 7-12 [PMID: 16378010]

6 **Huang MC**, Netland PA, Coleman AL, Siegner SW, Moster MR, Hill RA. Intermediate-term clinical experience with the Ahmed Glaucoma Valve implant. *Am J Ophthalmol* 1999; **127**: 27-33 [PMID: 9932995]

7 **Krishna R**, Godfrey DG, Budenz DL, Escalona-Camaaño E, Gedde SJ, Greenfield DS, Feuer W, Scott IU. Intermediate-term outcomes of 350-mm(2) Baerveldt glaucoma implants. *Ophthalmology* 2001; **108**: 621-626 [PMID: 11237919]

8 **Lloyd MA**, Baerveldt G, Fellenbaum PS, Sidoti PA, Minckler DS, Martone JF, LaBree L, Heuer DK. Intermediate-term results of a randomized clinical trial of the 350- versus the 500-mm2 Baerveldt implant. *Ophthalmology* 1994; **101**: 1456-1463; discussion 1456-1463 [PMID: 8058290]

9 **Roy S**, Ravinet E, Mermoud A. Baerveldt implant in refractory glaucoma: long-term results and factors influencing outcome. *Int Ophthalmol* 2001; **24**: 93-100 [PMID: 12201350]

10 **Tsai JC**, Johnson CC, Kammer JA, Dietrich MS. The Ahmed shunt versus the Baerveldt shunt for refractory glaucoma II: longer-term outcomes from a single surgeon. *Ophthalmology* 2006; **113**: 913-917 [PMID: 16751034]

11 **Coats DK**, Paysse EA, Orenga-Nania S. Acquired Pseudo-Brown's syndrome immediately following Ahmed valve glaucoma implant. *Ophthalmic Surg Lasers* 1999; **30**: 396-397 [PMID: 10334029]

12 **Dobler AA**, Sondhi N, Cantor LB, Ku S. Acquired Brown's syndrome after a double-plate Molteno implant. *Am J Ophthalmol* 1993; **116**: 641-642 [PMID: 8238227]

13 **Danesh-Meyer HV**, Spaeth GL, Maus M. Cosmetically significant proptosis following a tube shunt procedure. *Arch Ophthalmol* 2002; **120**: 846-847 [PMID: 12049596]

14 **Prata JA**, Minckler DS, Green RL. Pseudo-Brown's syndrome as a complication of glaucoma drainage implant surgery. *Ophthalmic Surg* 1993; **24**: 608-611 [PMID: 8233335]

15 **Rhee DJ**, Casuso LA, Rosa RH, Budenz DL. Motility disturbance due to true Tenon cyst in a child with a Baerveldt glaucoma drainage implant. *Arch Ophthalmol* 2001; **119**: 440-442 [PMID: 11231780]

16 **Ventura MP**, Vianna RN, Souza Filho JP, Solari HP, Curi RL. Acquired Brown's syndrome secondary to Ahmed valve implant for neovascular glaucoma. *Eye* (Lond) 2005; **19**: 230-232 [PMID: 15184963]

17 **Dobler-Dixon AA**, Cantor LB, Sondhi N, Ku WS, Hoop J. Prospective evaluation of extraocular motility following double-plate molteno implantation. *Arch Ophthalmol* 1999; **117**: 1155-1160 [PMID: 10496387]

18 **Frank JW**, Perkins TW, Kushner BJ. Ocular motility defects in patients with the Krupin valve implant. *Ophthalmic Surg* 1995; **26**: 228-232 [PMID: 7651689]

19 **Muñoz M**, Parrish RK. Strabismus following implantation of Baerveldt drainage devices. *Arch Ophthalmol* 1993; **111**: 1096-1099 [PMID: 8352692]

20 **Rauscher FM**, Gedde SJ, Schiffman JC, Feuer WJ, Barton K, Lee RK; Tube Versus Trabeculectomy Study Group. Motility disturbances in the tube versus trabeculectomy study during the first year of follow-up. *Am J Ophthalmol* 2009; **147**: 458-466 [PMID: 19038375 DOI: 10.1016/j.ajo.2008.09.019]

21 **Smith SL**, Starita RJ, Fellman RL, Lynn JR. Early clinical experience with the Baerveldt 350-mm2 glaucoma implant and associated extraocular muscle imbalance. *Ophthalmology* 1993; **100**: 914-918 [PMID: 8510906]

22 **Roizen A**, Ela-Dalman N, Velez FG, Coleman AL, Rosenbaum AL. Surgical treatment of strabismus secondary to glaucoma drainage device. *Arch Ophthalmol* 2008; **126**: 480-486 [PMID: 18413516 DOI: 10.1001/archopht.126.4.480]

23 **Costa PG**, Debert I, Passos LB, Polati M. Persistent diplopia and strabismus after cataract surgery under local anesthesia. *Binocul Vis Strabismus Q* 2006; **21**: 155-158 [PMID: 16934027]

24 **Gómez-Arnau JI**, Yangüela J, González A, Andrés Y, García del Valle S, Gili P, Fernández-Guisasola J, Arias A. Anaesthesia-related diplopia after cataract surgery. *Br J Anaesth* 2003; **90**: 189-193 [PMID: 12538376]

25 **Wilson-Holt N**, Franks W, Nourredin B, Hitchings R. Hypertropia following insertion of inferiorly sited double-plate Molteno tubes. *Eye* (Lond) 1992; **6** (Pt 5): 515-520 [PMID: 1286718]

26 **Repka MX**, Yu F, Coleman A. Strabismus among aged fee-for-service Medicare beneficiaries. *J AAPOS* 2012; **16**: 495-500 [PMID: 23158551 DOI: 10.1016/j.jaapos.2012.07.010]

27 **Osher RH**, Golnik KC, Barrett G, Shimizu K. Intentional extreme anisometropic pseudophakic monovision: new approach to the cataract patient with longstanding diplopia. *J Cataract Refract Surg* 2012; **38**: 1346-1351 [PMID: 22727989 DOI: 10.1016/j.jcrs.2012.04.029]

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