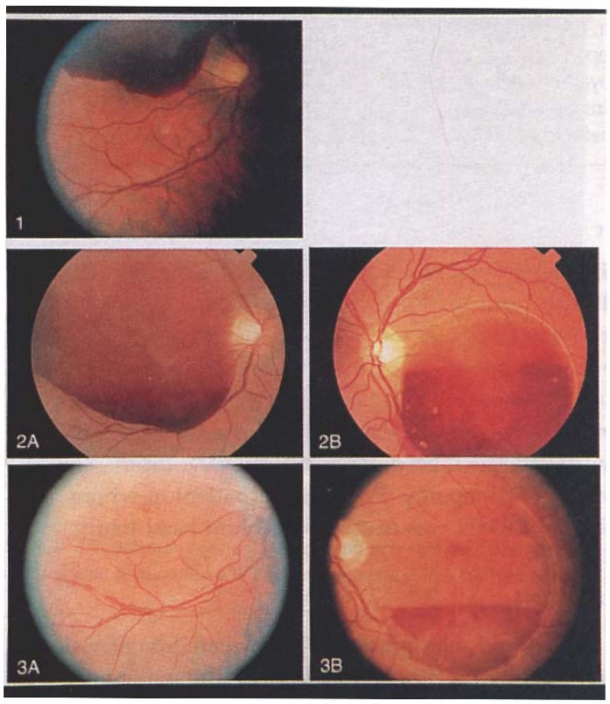

Fig. 1. *Photograph of right pre-retinal macular haemorrhage (PRMH) showing initial foveal sparing.*

Fig. 2. *(A) Right PRMH after 2 weeks, showing the haemorrhage has moved down over the fovea. (B) Left PRMH after 2 weeks, showing RBC sedimentation.*

Fig. 3. *More rapid absorption of PRMH in the right eye (A) than the left eye (B) after 2 months.*

the internal limiting membrane allows a more rapid recovery which occurs within days. In our described case there was evidence of leakage of RBCs into the vitreous in the right eye only, indicating some spontaneous membrane disruption. This was associated with a more rapid resolution than on the left, although still taking several months.

Previous descriptions of PRMH excluding proliferative diabetic retinopathy have indicated a central position. This case was unusual in that the source of haemorrhage was eccentric in one eye, although pre-retinal haemorrhage nasal to the optic disc has been described following bungee jumping;[8] this was curiously unilateral despite a similar causation to the described case.

We thank Southampton Eye Unit

References

1. Mushin AS. Ocular damage in the battered-baby syndrome. BMJ 1971;III:402–4.
2. Friedman SM, Margo CE. Bilateral subinternal limiting membrane hemorrhage with Terson syndrome. Am J Ophthalmol 1997;124:850–1.
3. Gomez-Ulla F, Fente B, Torreira MG, *et al.* Choroidal vascular abnormality in Purtscher's retinopathy shown by indocyanine green angiography. Am J Ophthalmol 1996;122:261–3.
4. Keyser BJ, Ferguson JB. Retinal venous beading with recurrent preretinal hemorrhage. Am J Ophthalmol 1997;123:696–8.
5. Duane TD. Valsalva hemorrhagic retinopathy. Trans Am Ophthalmol Soc 1972;70:298–313.
6. Gass JDM. Stereoscopic atlas of macular disease diagnosis and treatment. 2nd ed. St Louis: CV Mosby, 1977:320.
7. Gabel VP, Birngruber R, Gunther Koszka H, *et al.* Nd:YAG laser photodisruption of hemorrhagic detachment of the internal limiting membrane. Am J Ophthalmol 1989;107:33–7.
8. Jain BK, Talbot EM. Bungee jumping and intraocular haemorrhage. Br J Ophthalmol 1994;78:236–7.

Victoria J.M. Barrett ✉
R.G. Collyer-Powell
Department of Ophthalmology
Salisbury District General Hospital
Salisbury SP2 8BJ, UK

Sir,

Treatment of aqueous misdirection by trans-scleral diode laser photocoagulation

'Malignant glaucoma' describes a rare, difficult to manage condition characterised by elevated (often greatly) intraocular pressure (IOP), a flat or very shallow anterior chamber (AC), failure to respond to a patent peripheral iridectomy (PI), exacerbation by miotic therapy and the presence of a degree of misdirection of aqueous humour within the vitreous gel.[1] Current best practice recommends[2] medical management with intensive cycloplegic therapy, proceeding to a pars plana procedure should topical therapy fail. Unfortunately such radical surgery is technically demanding and this, together with the severity of the condition, has meant that outcomes have often been poor. We describe two cases where aqueous misdirection has been successfully managed by trans-scleral diode laser photocoagulation.

Case reports

Case 1. A 77-year-old woman presented with left angle closure glaucoma. Visual acuity was 6/9, the right eye being blind following unsuccessful management of malignant glaucoma 8 years previously. She failed to respond to medical therapy and following a repeat laser PI developed features of aqueous misdirection. With cycloplegic agents the AC was still only one-quarter corneal thickness when she was referred to us for further management. She underwent diode laser sector ciliary ablation, laser applications being applied in three rows aligned along the underlying ciliary processes (as shown in Fig. 1) with a 1 mm space between applications. One day later AC had deepened to 1.8 mm.[3] She went on to have cataract surgery and is currently well controlled on monotherapy.

Case 2. An 89-year-old woman was referred 6 weeks following right phacotrabeculectomy for uncontrolled chronic angle closure glaucoma. She had an IOP of 25 mmHg, a failed bleb, flat AC, patent PI and 360° of peripheral anterior synechiae. A diagnosis of aqueous misdirection was made but intensive cycloplegia, aqueous suppressants and Nd:YAG capsulotomy were unsuccessful in controlling the IOP. Trans-scleral photocoagulation was performed superonasally in a

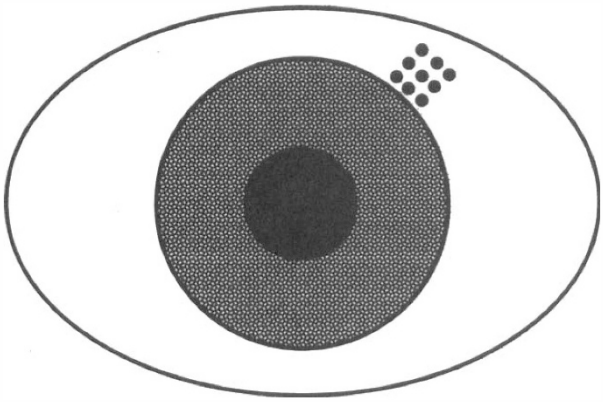

Fig. 1. *Case 1, left eye. Nine applications of the diode laser of 2 s duration varying in power from 1.5 to 3 W.*

pattern similar to that of Fig. 1 (nine applications of 2 s at 2 W), following which the AC deepened nasally. The temporal AC remained shallow, however, and further treatment was applied (superotemporally, same parameters), together with an inferior 180° cycloablation in view of the failed drainage surgery (11 applications of 2 s at 2 W). Following this the AC also deepened temporally. At latest review the AC depth was in excess of 2.5 mm and the IOP was 17 mmHg on topical therapy.

Comment

Malignant glaucoma has been reported following a variety of incisional and laser surgical procedures.[1,4–6] The block may occur at a number of sites resulting in iridolenticular, iridovitreal, ciliolenticular or ciliovitreal block, giving rise to the alternative nomenclature of aqueous misdirection, ciliary block glaucoma and direct lens block glaucoma. The initiating event is unknown. Shaffer[7] proposed posterior diversion of aqueous humour into the vitreous cavity, while Epstein *et al.*[8] postulated a forward displacement of the anterior vitreous with apposition of the anterior hyaloid face against the lens and ciliary body preventing the normal anterior flow of aqueous humour which then accumulates in the vitreous cavity. In addition, biochemical and conductivity changes result in a condensed anterior vitreous and anterior hyaloid face with reduced permeability of these structures to aqueous. The accumulation of aqueous in the vitreous cavity increases pressure in the posterior segment pushing the iris lens diaphragm forwards with shallowing of the AC.[8]

The aims of treatment are to address the site of the block and allow the release of intravitreal aqueous. Therapeutic options include medical and laser or incisional surgical procedures. The mainstay of medical therapy is atropine cylcoplegia, osmotic agents and aqueous suppressants.[1,2] Miotics should not be used in this scenario as they may result in ciliary muscle contraction and shallowing of the AC.[5] Medical therapy has been reported as ineffective in approximately 50% of cases.[6] If medical therapy fails, surgical intervention in the form of a laser or an intraocular procedure is required. Pars plana vitrectomy with anterior hyaloid face disruption – following previous lens extraction[9] and a combined lens extraction, primary posterior capsulectomy and surgical vitrectomy for phakic eyes[10] – have been advocated. To avoid such major intervention various laser procedures have been described. Nd:YAG laser capsulotomy and/or vitreous disruption has proven to be of benefit in selected cases where the hyaloid face is accessible via PI or pupil.[11] Argon laser shrinkage of ciliary processes through a PI has also been successful.[12]

Our use of the diode laser in aqueous misdirection is a logical extension of the above treatments. A possible explanation in case 1 is of relief of ciliolenticular block by sector cyclectomy. Case 2 probably represents anterior hyaloid face disruption with sequential release of separate collections of loculated aqueous. Lowering of IOP following cycloablation may in turn have increased the permeability of the anterior vitreous face, allowing re-establishment of the normal bidirectional flow of aqueous.

Diode laser cyclophotocoagulation is well established in the treatment of glaucoma.[13] This straightforward extraocular procedure can be performed under local anaesthesia and is associated with only minor degrees of pain and inflammation in most eyes. It can be performed in the absence of clarity of the ocular media and does not compromise the conjunctiva with respect to future glaucoma drainage surgery. We would therefore advocate its consideration in cases of aqueous misdirection unresponsive to medical therapy.

References

1. Luntz MH, Rosenblatt M. Malignant glaucoma Surv Ophthalmol 1987;32:73–93.
2. European Glaucoma Society. Secondary angle closure glaucomas. 1998 Terminology and guidelines for glaucoma. Savona: Dogma, 1998:119.
3. Smith RJ. A new method of estimating the depth of the anterior chamber. Br J Ophthalmol 1979,63:215–20.
4. von Graefe A. Beitrage zur Pathologie und Therapie des Glaucomas. Graefes Arch Ophthalmol 1869;15:108.
5. Simmons RB, Montenegro MH, Simmons RJ. Primary angle closure glaucoma. In: Tasman W, Jaeger EA, editors. Duane's clinical ophthalmology. Hagerstown: Lippincott 1999;3(53):1–43.
6. Liebmann JM. Malignant glaucoma: cases in controversy. J Glaucoma 1999;8:149–53.
7. Shaffer RN. The role of vitreous detachment in aphakic and malignant glaucoma. Trans Am Acad Ophthalmol Otol 1954;58:217–31.
8. Epstein DL, Hashimoto JM, Anderson PJ, Grant WM. Experimental perfusions through the anterior and vitreous chambers with possible relationships to malignant glaucoma. Am J Ophthalmol 1979;88:1078–86.
9. Byrnes GA, Leen MM, Wong TP, Benson WE. Vitrectomy for ciliary block (malignant) glaucoma. Ophthalmology 1995;102:1308–11.
10. Tsai JC, Barton KA, Miller MH, Khaw PT, Hitchings RA. Surgical results in malignant glaucoma refractory to medical or laser therapy. Eye 1997;11:677–81.
11. Brown RH, Lynch MG, Tearse JE, Nunn RD. Neodymium-YAG vitreous surgery for phakic and pseudophakic malignant glaucoma. Arch Ophthalmol 1986;104:1464–6.

12. Herschler J. Laser shrinkage of the ciliary processes: a treatment for malignant (ciliary block) glaucoma. Ophthalmology 1980;87:1155–9.
13. Spencer AF, Vernon SA. Cyclodiode': results of a standard protocol. Br J Ophthalmol 1999;83:311–6.

Rita Sengupta
Michael Austin ✉
Department of Ophthalmology
Singleton Hospital
Swansea SA2 8QA, UK

James Morgan
Cardiff Eye Unit
University Hospital of Wales
Heath Park
Cardiff CF14 4XW, UK

Sir,

Teaching junior ophthalmologists phacoemulsification under topical anaesthesia

Topical anaesthesia for cataract surgery is now reasonably commonplace. It has been well documented that patients prefer topical anaesthesia to retrobulbar or peribulbar anaesthesia, mainly due to the avoidance of the injection involved.[1,2] In addition, topical anaesthesia results in fewer intraoperative and post-operative complications.[3] Adjunction of topical pre-operative treatment with intracameral lignocaine has been reported to be safe[4,5] and effective; in particular it improves patient co-operation and reduces discomfort caused by tissue manipulation for phacoemulsification.[6] It has been an anecdotal idea that topical anaesthesia is not ideal for teaching trainee ophthalmologists phacoemulsification. We report the results of a prospective audit set up to ascertain whether patients felt excessive discomfort or movement while their cataract operation was being performed by a trainee ophthalmologist, compared with an experienced surgeon.

Methods

Patients attending for day case phacoemulsification surgery were divided into two groups: one to be operated upon by the consultant alone and the other to be operated on by a trainee ophthalmologist with assistance from the consultant. The trainee ophthalmologists had varying levels of surgical experience, from novices to senior registrars. All patients had the same topical anaesthetic pre-operatively: topical 0.75% bupivacaine supplemented by 1 ml of 2% intracameral lignocaine, combined with 1 ml of balanced salt solution. All cataract surgery was performed using a temporal, clear corneal approach, 3.2 mm incision, bimanual phacoemulsification and foldable intraocular lens insertion into the capsular bag. In all supervised cases, the consultant would initiate the procedure up to and including hydrodissection. The trainee would then continue and would proceed until they experienced difficulty progressing to the next stage. The level of operating surgeon and duration of surgery and any per-operative complications were noted.

Immediately after surgery, patients were asked by an independent observer to state whether they felt there had been any discomfort experienced during surgery and, if so, whether this was tolerable or severe. They were also asked at this time whether they felt excessive movement of their eye during surgery. The duration of surgery was compared between groups with the Student's *t*-test. Pain reported after surgery was compared between groups with the Mann–Whitney *U*-test and any difference in the proportion reporting excessive movement during surgery was compared using chi-square analysis.

Results

A total of 74 patients were studied. Thirty-seven patients underwent surgery by the consultant and 37 patients by a trainee ophthalmologist. In 2 cases the trainee was a Senior Registrar, in 27 a Specialist Registrar and in 8 a Senior House Officer. More than one trainee surgeon was involved at each level. No patient required additional sedation. The duration of surgery in the group operated on by the trainees was significantly longer (17.9 min) compared with the group operated on by the consultant (11.8 min) ($p < 0.005$). There was no significant difference between the amount of movement or discomfort perceived by the two groups of patients during surgery. The supervised trainees' per-operative complication rate was 2.7%. This appeared to be similar to the rate for the consultant alone but a larger study would be required to verify this.

Table 1. *Complications of surgery*

Operating surgeon	Consultant		Trainee	
Surgeon's perception of akinesia:				
Adequate		37 (100%)		37 (100%)
Inadequate		0		0
Per-operative complications:	None	36 (97%)	None	36 (97%)
	Posterior capsule tear/vitreous loss	1 (3%)	Posterior capsule tear/vitreous loss	1 (3%)
Mean duration of surgery:	11.8 min		17.9 min	
Post-operative complications:	None	35 (95%)	None	35 (95%)
	Uveitis	1 (3%)	Uveitis	1 (3%)
	CMO	1 (3%)	CMO	1 (3%)

CMO, cystoid macular oedema.