Implants for draining neovascular glaucoma

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SUMMARY The implant design, surgical technique, and pharmacological methods of controlling bleb fibrosis, used to treat neovascular glaucoma, are described, together with the results of 14 operations performed on 12 eyes.

Established glaucoma with iris neovascularisation and a medically uncontrollable intraocular pressure carries such a poor prognosis that treatment is usually limited to the retrobulbar injection of alcohol or enucleation—even in the presence of slight residual vision. One of us (A.C.B.M.) developed a series of draining implants for use in severe and complex cases of glaucoma (Molteno, 1969a, b; 1970; 1973a, b; Molteno *et al.*, 1976a, b). This communication reports the design of implant and the surgical technique currently used to treat cases of neovascular glaucoma at the Tygerberg Hospital glaucoma clinic and at the University of Edinburgh.

Principles of implant

Among the first attempts to drain cases of glaucoma by an artificial device was that of Zorab (1912), who connected the anterior chamber to the sub-conjunctival space by means of a silk thread which acted as a 'conducting wick'. This operation of aqueoplasty gave long-term control of intraocular pressure in some cases (Martin and Zorab, 1974). This work has been followed by numerous attempts to drain glaucomatous eyes by a wide range of devices, which can be classified as follows:

(1) Translimbal tubes of various materials connecting anterior chamber to subconjunctival space.
(2) Sheets of gel film and other materials placed beneath the conjunctiva over a conventional drainage operation fistula (e.g., cornea-scleral trephine hole or iridencleisis opening) in order to promote the formation of a large bleb.
(3) Implants of various materials inserted to maintain the patency of cyclodialysis clefts.

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(4) Devices draining vitreous from eyes with absolute glaucoma.

Glaucoma implants are reviewed by Ellis (1960), and other recent publications in this field include those of Epstein (1959), La Rocca (1962), MacDonald and Pierce (1965), and Blumenthal *et al.* (1970).

Specifications and mode of action of implants

Our current implant utilises an artificial translimbal tube combined with bleb spreading plate. The translimbal tube of silicone rubber is 16 mm long and has an internal diameter of 0·30 mm and an external diameter of 0·63 mm. It is firmly attached to and opens on to the upper surface of a thin circular acrylic episcleral plate (Stellon brand polymethyl methacrylate) 13 mm in diameter, which has a thickened rim 0·7 mm in height around the circumference. This rim is perforated for attachment of the translimbal tube and to allow passage of sutures which fasten it in position on the globe (Fig. 1).

When used to drain a case of glaucoma, the episcleral plate is sutured to sclera and buried beneath a thick flap of Tenon's capsule and conjunctiva. The translimbal tube is trimmed to the correct length and inserted into the anterior chamber after being buried under a lamellar scleral flap. During the first few days after operation the episcleral plate becomes surrounded by a thin layer of vascular connective tissue which impedes the flow of aqueous away from the plate and causes the intraocular pressure to rise. The increasing pressure causes this fibrovascular 'bleb lining' to become distended and to be stretched by a force that is directly proportional to the product of intraocular (= to intrableb) pressure and radius of curvature of the 'bleb lining' (Young, 1805; Laplace, 1807).

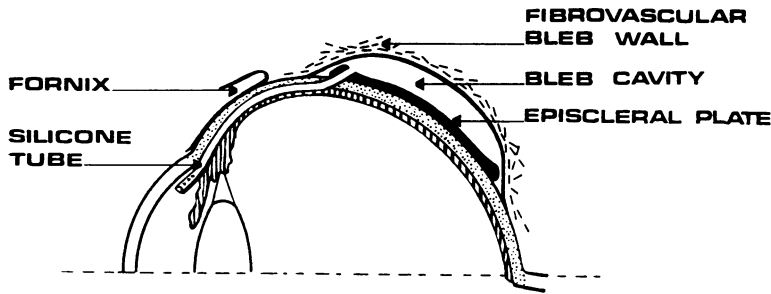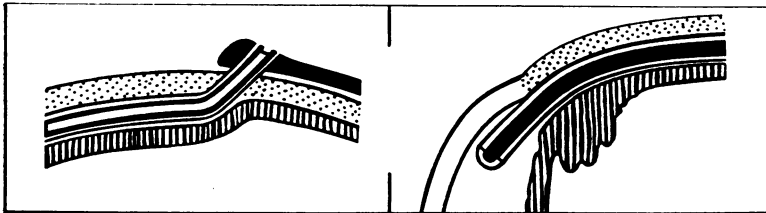Consequently, if this 'bleb lining' can be prevented

Fig. 1 *Situation and principle of draining implant*

from becoming too thick (by administration of a combination of anti-inflammatory agents), and if the radius of curvature of the bleb lining can be made large through the presence of a large episcleral plate, then a normal intraocular pressure causes progressive stretching and enlargement of this bleb lining until the increased area available for drainage of aqueous so lowers the intraocular (and intrableb) pressure that the bleb lining no longer stretches. This situation results in long-term control of intraocular pressure at a level which depends directly on the area of bleb available for drainage and inversely on the thickness of the fibrous inner bleb lining.

Case selection

We hesitated to operate on eyes with neovascular glaucoma, since a drainage operation with insertion of foreign material (albeit inert plastic) into an inflamed eye containing infarcted retina is a situation which, in theory, is likely to cause sympathetic ophthalmitis. However, after being forced to operate on two only eyes and on a patient with bilateral diabetic retinopathy in whom the glaucomatous eye saw better than the normotensive eye we have gradually relaxed our criteria for operation, so that we operate in the presence of a good fellow eye if the eye has light perception and if the patient chooses an implant after having the dangers pointed out to him and after having been offered a quick and safe end to his pain through enucleation.

All 12 eyes drained by implants had iris neovascularisation, corneal oedema, and severe pain. Intraocular pressures on full doses of acetazolamide, glycerol, and gutt. adrenaline tartrate (Epitrate) 2% varied from 42 to 75 mmHg, while in 6 of the cases free blood was present in the anterior chambers. Visual acuity of these eyes varied from light perception only in 9 eyes through counting fingers at 2 m (1 eye) at 5 m (1 eye) to 3/60 (1 eye).

In 2 cases the underlying pathology was considered to be chronic angle closure glaucoma with secondary central retinal vein occlusion. In 5 cases vein occlusions occurred without obvious local cause, while in the remaining 5 cases iris neovascularisation was associated with grade IV diabetic retinopathy. Both patients with primary angle closure glaucoma were healthy, but of the remaining 10 cases, 9 patients had serious general disease including myocardial ischaemia in 3 cases, vascular hypertension in 1 case, and severe diabetes in 5 cases. Of the diabetics 3 had markedly elevated blood urea levels and proteinuria as a result of advanced diabetic nephropathy.

Surgical technique

Cases were prepared for operation with a combination of gutt. adrenaline tartrate 2% every 30 minutes for 2 hours before operation, acetazolamide 250 to 500 mg intravenously 1 hour before operation, and mannitol 10% solution 100 to 400 ml by slow intravenous infusion, starting 30 minutes before operation, to lower the intraocular pressure as much as possible.

Fig. 2 *Insertion of implant. A deep lamellar scleral flap has been dissected up to the limbus (patient 8)*

Anaesthesia

General anaesthesia was preferred, but when the general condition was poor operations were done under neurolept analgesia and local injection of 2% lignocaine retrobulbarly and over the condyle of the mandible.

Dissection and placing of implants

After inserting a speculum and passing a suture beneath the superior rectus muscle near its insertion the globe was rotated so as to expose the supero-nasal or supero-temporal quadrant. The conjunctiva and Tenon's capsule were separated from the limbus and a large fornix-based flap raised and dissected posteriorly to expose the insertions of superior and medial (or lateral) rectus muscles and gain access to Tenon's space.

After cleaning sclera and cauterising all bleeding points in the exposed quadrant a large square lamellar flap of sclera was dissected up and reflected at the limbus, care being taken not to enter the anterior chamber (Fig. 2). The implant was then placed in position and the episcleral plate sutured firmly to sclera, using two anterior suture holes and interrupted mattress sutures of 7·0 silk. The next step was to cut off the tube so that the end overlapped the cornea by 2 to 3 mm (Fig. 3). The anterior chamber was entered by a circumferential limbal incision under the lamellar flap using a no. 11 Bard Parker blade and a broad or large peripheral iridectomy performed without any special precau-

tions to prevent bleeding. The free end of the silicone tube was then inserted into the anterior chamber, and after it was arranged to lie free in the centre of the iridectomy it was firmly fastened in this position by means of a 7·0 silk suture passed around the tube, through the lamellar flap, and tied tightly so as to prevent the tube becoming displaced sideways, being withdrawn from the anterior chamber, or sliding further into the anterior chamber and touching the central corneal endothelium (Fig. 4).

Closure

After the scleral flap had been sutured into position by 4 interrupted sutures of 6·0 silk, Tenon's capsule was drawn forward over the episcleral plate of the implant and sutured to sclera near the limbus, while the conjunctiva was held in apposition to the limbus by a 7·0 silk suture at each corner of the flap. The operation was completed by injecting a mixture of gentamycin, cephalosporin, and methylprednisolone (Depot Medrol) beneath the conjunctiva away from the implant.

Postoperative management

The operated eye only was padded, and patients were mobilised the following day in spite of the presence of a hyphaema in every case.

Local medication consisted of gutt. atropin. 1%, gutt. adrenaline tartrate 2%, and gutt. dexamethason. 0·1% all given 3 times a day for 6 weeks, after
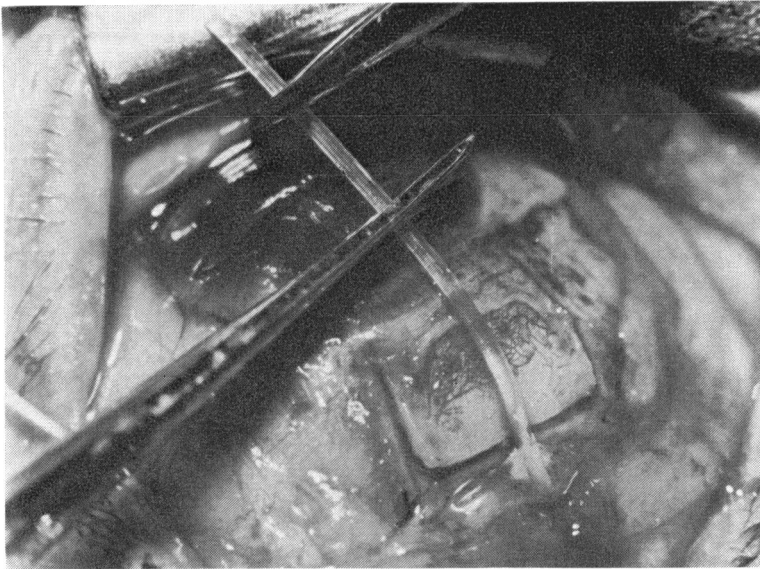
Fig. 3 *The implant has been sutured to sclera and the silicone tube laid across the eye so as to cut off the end, 2 mm inside the limbus*

which atropine and dexamethasone were stopped while adrenaline tartrate was continued in twice daily dosage until the intraocular pressure fell below 15 mmHg.

All patients received a combination of a steroid, fluphenamic acid, and colchicine to control inflammation and limit fibrosis round the implant. As many of these patients were unable to tolerate large doses of systemic steroids and others had severely reduced renal function, our usual regimen of prednisone 10 mg t.i.d., fluphenamic acid 200 mg t.i.d., and colchicine 0·25 to 0·5 mg t.i.d. for 6 weeks

was modified where necessary by replacing systemic steroids by a weekly subconjunctival injection of 0·25 ml of methylprednisolone and adjusting the dose of fluphenamic acid and colchicine according to renal function.

Postoperative behaviour of intraocular pressure

After insertion of an implant the eyes remained soft for up to 3 weeks, after which the intraocular pressures rose gradually to a peak of between 35 and 60 mmHg (6 to 9 weeks after operation) and
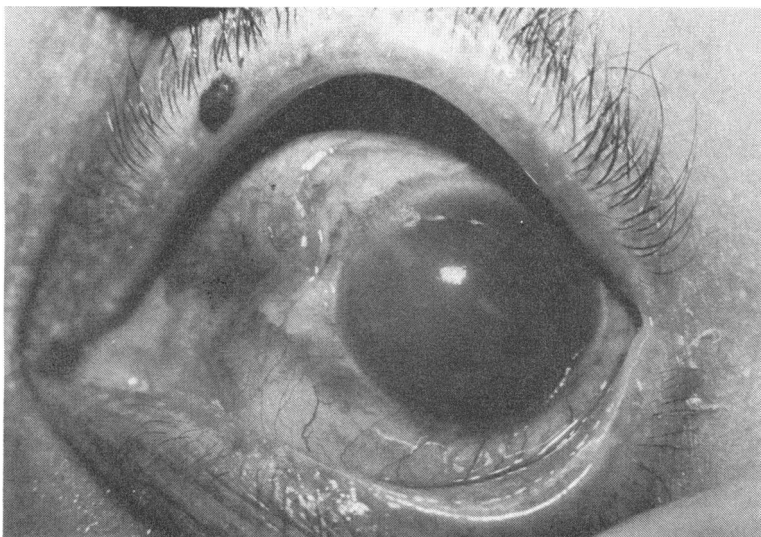

Fig. 4 *Eye of Patient 8, 7 months after operation, showing broad iridectomy and distended bleb over implant (intraocular pressure 15 mmHg on adrenaline tartrate 2% b.d.)*

Table 1   *Effect of implants on IOP*

Patient	Sex	Age	Preop. IOP	Present/final IOP	Treatment	Follow-up (months)	Remarks
1	Male	45	60	10–30	E	38	Late bleb thickening Second implant after 24 months Recurrent vitreous haemorrhage
2	Male	54	64	30	E	16	Iris blocked tube Second implant after 3 months Persistent low-grade iritis
3	Male	82	45	10	—	16	Eye quiet
4	Male	72	55	30	E	14	Eye quiet
5	Female	55	53	15	—	14	6/12 vision Eye quiet
6	Female	70	42	5	—	10	Bleb perforation Implant removed after 8 months Poor general state due to rheumatoid arthritis
7	Female	68	67	12	E	8	Eye quiet Died at 8 months Uraemia and diabetes
8	Female	58	75	15	E	9	Eye quiet
9	Female	42	50	18	E	9	No rubeosis Eye quiet
10	Male		70	30	E A	9	Eye quiet
11	Male	48	80	20	E	8	Ectropion uvea ┬ ┬ ┬ Eye quiet
12	Female	55	52	17	E A S	6	Eye quiet

E = Epitrate (adrenaline tartrate). A = Atropine. S = Sofradex (framycetin, dexamethasone, and gramicidin)

then fell rapidly at first and then more gradually to a stable value some 3 to 6 months after operation. The 'stable stage' pressures of 11 eyes with implants are between 10 and 20 mmHg in 6 eyes and between 20 and 30 mmHg in 5 eyes, while follow-up varies from 6 months to 38 months (Table 1).

Visual results

The visual acuity improved from 3/60 to 6/12 in one patient whose central vein occlusion was precipitated by chronic angle closure glaucoma. However, in the remaining cases, although measurable improvement did not occur, all patients but one have retained their preoperative vision so far.

Behaviour of iris neovascularisation

The abnormal neovascular formations on the iris persisted as long as the intraocular pressure was elevated, but once it fell towards normal values (below 35 mmHg) the vessels collapsed and became very inconspicuous. However, they did not disappear, for where the intraocular pressure again became elevated

or if the anterior chamber became active (Table 1) they filled with blood and became obvious once more.

Early complications

The postoperative course of these eyes was characterised by a hyphaema in every case ranging from a small collection of blood that took 6 days to clear to a full hyphaema which took 3 weeks to clear. The presence of blood in these cases did not alter the treatment in any way and was not associated with severe pain or elevation of pressure.

A more serious complication was iris blocking the drainage tube. This occurred in 3 of the earlier cases and was cleared surgically in 1 while in the remaining 2 cases a second implant was inserted.

Late complications

In 1 patient with severe rheumatoid arthritis and abnormally fragile tissue the anterior chamber remained very shallow with hypotony for 8 months, after which the implant became partially exposed

by breakdown of the overlying conjunctiva and was removed. The only other late complication has been a late rise in pressure associated with recurrent massive vitreous haemorrhages. This occurred 2 years after insertion of the initial implant and was treated by insertion of a second implant, which has given reasonable control of pressure up to the time of writing (14 months).

Our results can be summed up by the statement that after 14 operations carried out on 12 eyes with neovascular glaucoma all 12 eyes are pain-free and have a pressure of less than 30 mmHg without any systemic hypotensive medication.

Discussion

The use of implants to treat thombotic glaucoma is the culmination of a long series of animal experiments and clinical investigations, during which the implant technique has been extended to treat increasingly severe and complex cases of glaucoma so that a series of more than 330 operations with up to 10 years' follow-up is available for study. In spite of this experience the authors have found the treatment of neovascular glaucoma a difficult and demanding field, because, in general, the patient's state is poor, the use of steroids, fluphenamic acid, and colchicine is essential for control of bleb fibrosis, and the postoperative behaviour of these eyes is stormy. However, the ultimate results are surprisingly good, and even better results will be obtained in future now that the need for a broad iridectomy to remove iris from the vicinity of the tube is recognised. Many more cases are needed to assess the risks of sympathetic ophthalmitis and to decide the ultimate visual prognosis of these eyes.

However, at present we consider that insertion of an implant with pharmacological control of bleb fibrosis is the treatment of choice for neovascular glaucoma in only eyes, in cases with bilateral predisposing disease, and where significant vision is present in the glaucomatous eye.

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