Long Tube Implants in the Management of Glaucoma

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SUMMARY

The design, surgical insertion and results of a plastic draining implant for severe glaucoma are reported. The need for pharmacological control of bleb inflammation is stressed and the favourable long-term outlook for patients with such implants is discussed.

S. Afr. med. J., **50**, 1062 (1976).

The senior author has previously reported methods of treating severe and advanced cases of glaucoma by means of an acrylic draining implant.[1-4] Subsequently we combined this method with drug therapy to limit bleb fibrosis.[5] This communication reports the results achieved in 112 patients who were treated with a modification of the implant. The original implant consists of an acrylic translimbal tube which opens onto the upper surface of a thin, curved episcleral plate sutured to the anterior portion of the globe. In most cases the initial implant provided adequate drainage via a large, unilocular anteriorly situated bleb, formed by the action of aqueous on the tissues covering the implant (Figs 1 and 2).

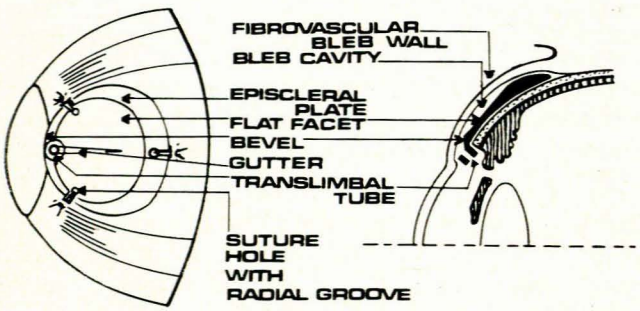

Fig. 1. Diagram to show principles of original implant.

METHODS

The Implant

The modified implant consists of a translimbal tube and an episcleral plate, but the episcleral plate is sutured to the sclera so as to lie entirely behind the equator. The translimbal tube, of fine-bore Silastic, enters the deeper layers of the sclera well behind the fornix and runs forward to enter the angle of the anterior chamber where it is perforated to form 5 mm of 'artificial trabeculae'. The implant thus lies largely within the scleral envelope until it emerges behind the fornix to form a bleb deep in the orbital tissues, where it is safe from the risks of hypotonia rupture and infection for the patient's lifetime (Figs 3 - 7).

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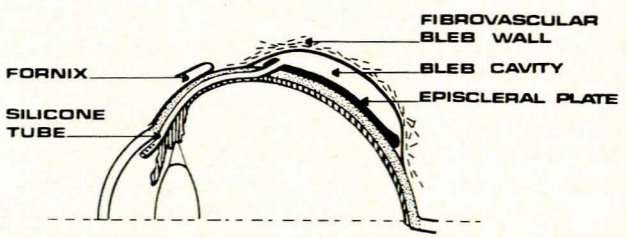
Fig. 2. Aphakic eye with uveitis and secondary glaucoma showing distended thick-walled bleb covering implant 4½ years after operation. (Normal intra-ocular pressure and coefficient of facility of outflow values off treatment.)



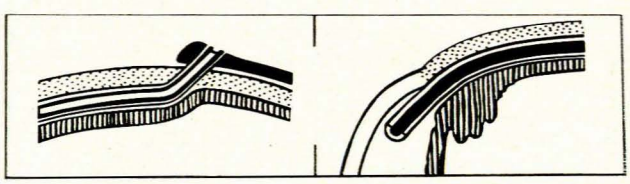


Fig. 3. Diagram to show principles of modified implant.

Patient Selection

At present patients are selected for insertion of implants after it has been shown that the disease cannot be controlled by treatment with Diamox (250 mg 4 times per day, supplemented with potassium), eserine (0,5% twice a day), Epitrate (*l*-adrenaline 2% 4 times per day), and, in some cases, glycerol by mouth (150 ml every 6 hours).

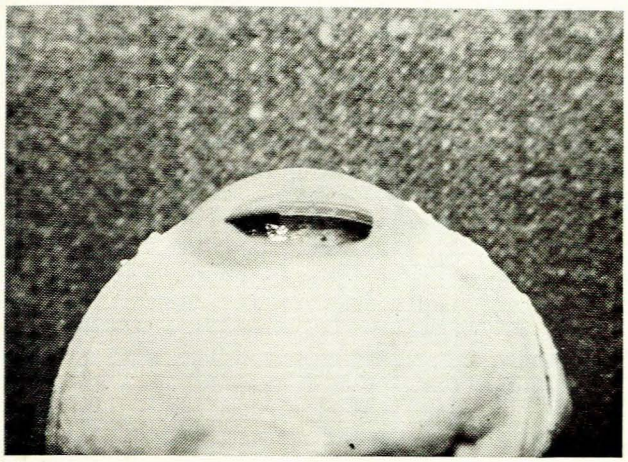


Fig. 4. Implant inserted into an enucleated eye to show perforated end of tube lying in the iridocorneal angle.

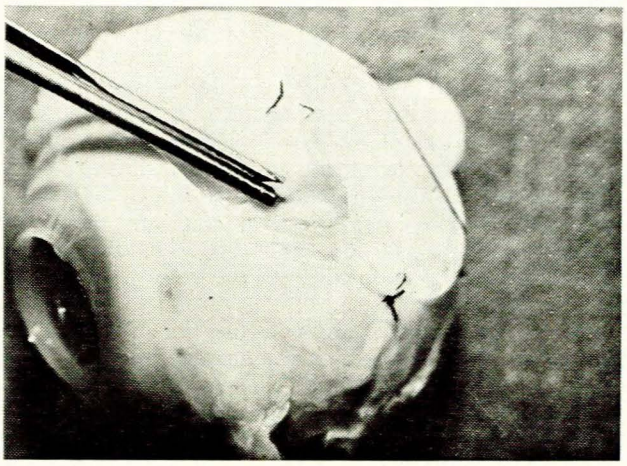


Fig. 6. Posterior scleral flap lifted to show attachment of silicone tube to acrylic episcleral plate.

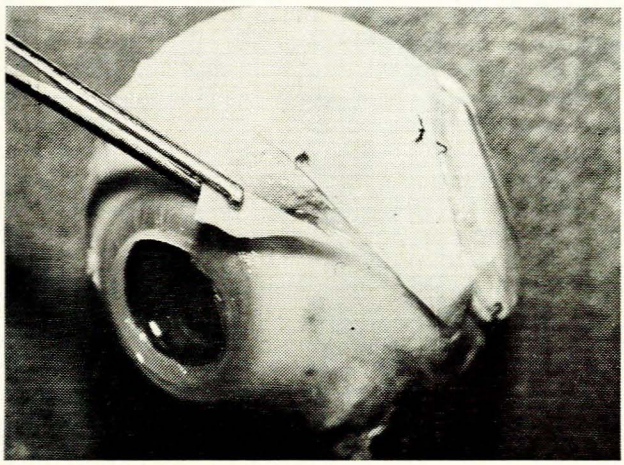


Fig. 5. Anterior scleral flap lifted to show course of silicone tube through deeper layers of sclera.

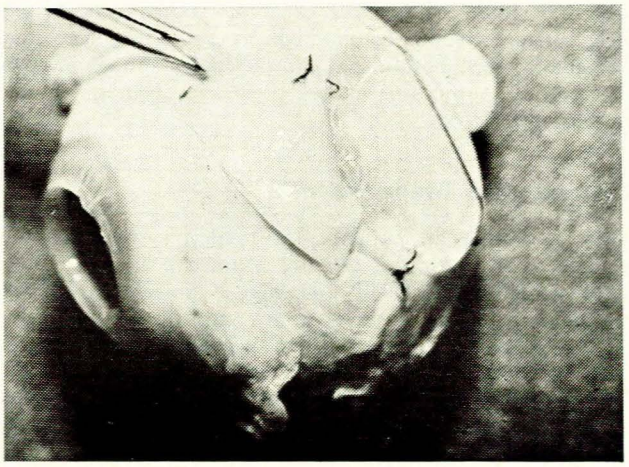


Fig. 7. Acrylic episcleral plate sutured to posterior portion of globe, showing zig-zag incision through outer three-quarters of the sclera.

Indications for insertion of implants as a primary procedure in preference to conventional surgery are still being worked out, but so far they include advanced buphthalmos, glaucoma secondary to uveitis, and aphakic and thrombotic glaucoma. Implants are not used in primary glaucoma unless the disease is terminal or conventional surgery has failed.

Preparation for Operation

Four days before the operation, treatment of patients is started with prednisone (10 mg 3 times a day), fluphenamic acid (200 mg 3 times a day), and colchicine (0,25 mg 3 times a day). This is the regimen for an adult weighing 70 kg and is administered by mouth.

Operative Technique

The operation is designed to place the perforated portion of the tube in the angle *at a tangent* to the limbus, so as to avoid injury to the corneal endothelium, and to place the tube in the deepest layers of the sclera. Early extrusion is avoided by placing the suture lines away from the tube.

Exposure of the superonasal quadrant of the globe is obtained by an incision into the conjunctiva and Tenon's capsule, from the superior rectus insertion to below and anterior to the medial rectus insertion.

After bleeding vessels have been cauterised and the sclera has been cleaned up to the limbus, a turn-point midway between the superior and medial rectus insertions and 7 mm from the limbus is marked with a cautery, and two triangular flaps of sclera are dissected up. The incision for the anterior flap extends from the turn-point along the anterior border of the medial rectus insertion to its inferior limit and then radially forward to the limbus, but *not* into the anterior chamber. The flap is raised by splitting the deepest layers of sclera until its base extends from the turn-point along a tangent to the limbus (Fig. 5).

The incision for the posterior flap extends from the turn-point to the medial margin of the superior rectus muscle and then back to the equator of the globe. This flap is also raised by splitting the deeper layers of the sclera until its base stretches radially back from the turn-point to the equator of the globe (Figs 6 and 7).

The episcleral plate of the implant is sutured to the sclera by means of two anterior holes only, the tube is laid along the base of the posterior flap, and the flap is sutured back into position. The tube is laid along the base of the anterior flap to select the point of entry into the anterior chamber.

After a stab incision through the limbal tissues, the tube is fed into the anterior chamber (iridectomy is not necessary). If correctly placed the perforated portion of the tube will occupy the inferonasal portion of the angle (Fig. 4). If entangled by the iris, the tube is freed by means of Ringer's solution syringed down the lumen, and the scleral flaps are firmly closed by multiple interrupted non-absorbable sutures (6,0 or 7,0 silk).

Tenon's capsule is drawn forward and sutured to the anterior flap of sclera by 2 or 3 interrupted sutures, and after closure of the conjunctiva by a continuous suture of 7,0 silk, the operation is completed by subconjunctival injection of a mixture of cephalosporin, gentamicin and methylprednisolone.

Postoperative Management

As a routine all patients are treated systemically for 6 weeks with prednisone 10 mg 3 times a day (for 70-kg adult), fluphenamic acid 200 mg 3 times a day, and colchicine 0,25 mg 3 times a day. Epitrate 2% drops 4 times per day, atropine 1% drops twice daily, and Sofradex drops 4 times per day are applied topically for 6 weeks.

This regimen blocks most of the known mediators of the inflammatory response. Prednisone has a wide range of actions which include stabilisation of cell membranes and inhibition of kinin formation. Fluphenamic acid blocks prostaglandin synthesis while colchicine blocks the hydroxylation of proline and thereby prevents the conversion of procollagen into collagen.[6]

Epitrate counteracts the action of the enzyme catechol *O*-methyltransferase which is increased in inflamed tissues; atropine blocks the effects of the vasodilator acetylcholine; while Sofradex contains the fluorinated prednisone derivative dexamethasone, which is an anti-inflammatory steroid with the side-effect of inducing atrophy of connective tissue. This regimen was administered meticulously for 6 weeks, while all the patients, except those who could be trusted to treat themselves, were hospitalised. These agents exhibit a very marked synergism in reducing bleb inflammation and thus limiting deposition of fibrous tissue around the episcleral plates of implants.

RESULTS

Local Complications

These were difficult to define with certainty owing to the advanced stage of the disease in the eyes selected for implants. However, the following complications were definitely ascribable to the implants:

(i) corectopia (due to iris becoming attached to the tube in the angle) was significant in 6 eyes but caused no visual deficit;

(ii) exposure of tube occurred in 3 of the earlier cases, before suitable scleral flaps were devised to overcome this problem; in 2 patients the sclera was resutured, in the third a preserved scleral graft was used to bury the tube;

(iii) tube blockage — where new blood vessels extended into the tube end — occurred in 2 patients with thrombotic glaucoma;

(iv) vitreous strands entered the tube in 8 instances, but have not interfered with drainage so far;

(v) endophthalmitis developed in 1 patient and was successfully treated, but it left an unduly thick bleb with poor drainage;

TABLE I. RESULTS OF INSERTION OF LONG TUBE IMPLANTS IN 64 PATIENTS (18 MONTHS TO 3 YEARS FOLLOW-UP)

Diagnosis	Cure (IOP 20 mmHg and C^0 = 0,15)	Control (IOP 20 mmHg on treatment and C^0 = 0,15)	Failure (IOP 20 mmHg and C^0 = 0,15)
Primary open-angle glaucoma			
Infantile	5	1	
Juvenile	2	3	
Adult	6	2	
Chronic closed angle glaucoma		2	1
Glaucoma secondary to congenital defects, surgery, trauma, displaced lenses			
Infantile	3	5	
Juvenile	2	4	1
Adult	11	4	2
Glaucoma secondary to uveitis	3	1	
Thrombotic glaucoma		4	2
	32	26	6

IOP — intra-ocular pressure; C^0 — coefficient of outflow.

(vi) the iris occluded the tube in 4 cases, where it was placed too deeply in the iridocorneal recess; in 3 cases the tube end was surgically removed and reinserted without complications, in the fourth a second implant was inserted.

Systemic Complications

The combination of prednisone, fluphenamic acid and colchicine administered by mouth caused remarkably few side-effects, considering the potencies and actions of the drugs. Gastric discomfort was the commonest side-effect and was observed in 8 patients. It was avoided by giving medication after meals and was readily treated with oral antacids, without the necessity of stopping treatment. Perforation of a pre-existing peptic ulcer occurred in 1 patient. Diabetes was unmasked in 2 patients by the use of steroids, while an Addisonian crisis occurred in 1 patient who developed pneumonia 5 weeks after discontinuing drugs to suppress fibrosis.

Over-all Results

We have performed 112 long tube implant operations over the last 3 years. Of 64 patients who have been followed-up for more than 18 months the present status is as follows: 32 are cured, in 26 the glaucoma is under control, and in 6 the operation failed (Table I).

Details of our first 24 patients (with more than $2\frac{1}{2}$ years follow-up) are summarised in Table II.

TABLE II. DETAILS OF RESULTS IN 24 PATIENTS (30 EYES) ($2\frac{1}{2}$ - 3 YEARS' FOLLOW-UP)

			Before operation (on drug treatment)			After operation			
Eye	Age of patient	Diagnosis	IOP (mmHg)	C^0	Vision	IOP	C^0	Vision	Drug therapy
1	25	OAG	45	0,003	PL	16	0,38	PL	Nil
2	55	Aphakic	42	0,008	PL	15	0,23	CF 2 m	Nil
3	59	Aphakic	27	0,05	6/8	20	0,23	6/18	Nil
4	33	OAG	38	0,10	6/5	20	0,24	6/5	E
5 (R)	44	Trauma	30	0,06	CF 2 m	15	0,18	CF 2 m	Nil
6 (L)		Trauma	38	0,06	CF 2 m	19	0,24	CF 2 m	Nil
7	10	Juvenile glaucoma (12 previous operations)	45	0,002	3/60	25	0,16	2/60	P, E
8 (R)	34	Trauma	35	0,06	CF 2 m	16	0,19	CF 2 m	P, E
9 (L)		Trauma	50	0,06	6/60	22	0,16	6/36	P, E
10 (R)	32	OAG	38	0,11	CF 3 m	16	0,33	CF 3 m	D, P, E
11 (L)		OAG	33	0,13	6/12	17	0,27	6/12	D, P, E
12 (R)	64	OAG	33	0,08	6/9	24	0,26	6/9	Nil
13 (L)		OAG	38	0,09	6/6	22	0,24	6/6	Nil
14	69	Aphakic	42	0,04	PL	16	0,17	PL	Nil
15	4	Buphthalmos	48	—	PL	15	—	No PL	Nil
16	57	Aphakic	43	—	CF 3 m	22	0,16	CF 3 m	Nil
17	42	Thrombotic	60	—	HM 1 m	35	0,11	CF 3 m	E*
18	61	Aphakic	30	0,01	PL	14	—	PL	Nil
19	43	Uveitis	38	0,03	PL	17	0,20	PL	E
20	13	Aniridia	33	—	PL	16	—	PL	E
21	22	OAG	60	0,00	No PL	22	0,15	No PL	E
22	46	Aphakic	28	0,10	6/20	17	0,47	6/20	D, E
23	48	Aphakic	45	—	HM 1 m	16	0,25	HM 1 m	Nil
24	61	Aphakic	50	—	PL	18	0,58	HM 1 m	Nil
25	47	Chron. angle closure	82	—	PL	30	0,20	6/9	E†
26 (R)	51	OAG	28	0,09	CF 2 m	15	0,28	CF 2 m	Nil
27 (L)		OAG	30	0,08	6/40	14	0,24	6/40	Nil
28	58	Uveitis	45	0,04	6/60	14	0,18	6/18	E
29 (R)	16	Buphthalmos	38	—	PL	20	—	PL	E, D
30 (L)		Buphthalmos	33	—	No PL	10	—	No PL	E, D

C^0 — coefficient of outflow; CF — counting fingers; D — Diamox; E — Epitrate; HM — hand movements; IOP — intra-ocular pressure; OAG — open-angle glaucoma; P — pilocarpine; PL — perception of light.

* Implant operations done twice.

† Surgical complication.

DISCUSSION

Precise indications for long tube implants are still being investigated, but at present several definite indications are recognised:

In buphthalmos which is too advanced for goniotomy, gratifying results have been obtained with the long tube implant.

In glaucoma due to smouldering uveitis, anterior-type implants gave good results but the long-term use of steroids to control uveitis usually caused undue thinning of blebs, with danger of perforation.[4] The new implants are easier to insert and the long-term prognosis is much better, since steroids can be used without danger of exposing the deeply buried implants.

Traumatic glaucomas, aphakic glaucomas, and neglected cases of angle-closure glaucoma form a mixed group, which includes buphthalmos and uveitis, and pose very difficult surgical problems. Implants are, however, indicated where conventional surgery seems hopeless, especially in young patients (Tables I and II).

Thrombotic glaucoma is another indication for long tube implants. The earlier anterior implants drained thrombotic glaucomas for a few months only before becoming blocked by new blood vessels invading the lumen of the tubes Our experience with 5 patients, who retained vision for the 3 - 10 months during which the intra-ocular pressure was controlled, suggested that it was worth while to treat this condition. So far, 7 eyes have been drained by long tube implants. The problem of new vessels which rapidly clogged the side end of the tube was overcome by using a simple open-ended tube and performing an iridectomy to keep new vessels away from the tube. The eyes of all 7 patients remain pain-free, with intra-ocular pressures varying from 10 to 40 mmHg on Epitrate alone (follow-up 6 months to $2\frac{1}{2}$ years).

We have described the use of implants with suppression of fibrosis in desperate cases where no other treatment seemed likely to be effective. However, long tube implants worked magnificently in advanced cases of primary glaucoma, whether buphthalmos, juvenile or adult types In such relatively normal phakic eyes they are inserted easily and without trauma. In addition, fibrosis suppression is less important in elderly patients and can be reduced somewhat, so that in patients over 40 years old with primary glaucomas one can almost guarantee that intra-ocular pressure will be controlled without hypotensive medication and without much risk of serious side-effects.

Long-Term Outlook

The development of blebs over implants has been observed in detail in 180 anterior implants, both by serial slit lamp photography and by histological examination of biopsy and autopsy material.

An initial subacute inflammatory reaction led to the formation of a thick-walled fibrovascular bleb, lined by an inner membrane of avascular fibrous tissue. Degeneration of this layer began approximately 3 months after the operation, the collagen gradually swelled and lost its staining properties, while the fibroblast nuclei became pyknotic and then disappeared. The inner fibrous layer became thinner and eventually disappeared approximately 5 years after insertion of the implant. Lymphatics were first noticed 18 months after operation and became gradually more numerous and prominent — while in some cases new aqueous veins appeared abruptly in the deeper layers of the bleb. These changes, accompanied by a gradual fall in intra-ocular pressure and increase in outflow, have been followed for up to 5 years in many cases, and in 2 eyes for 7 years.

The long tube implants behave in a similar fashion insofar as intra-ocular pressure is concerned, while 4 histological specimens which were available confirmed that the bleb histology was identical to that of anteriorly situated blebs.

A patient in whom a bronchogenic carcinoma developed after insertion of a long tube implant, provided a clear demonstration of the roles of fibrous tissue, lymphatics and the episcleral veins in bleb function. The carcinoma caused rapid wasting of body connective tissue, including that of the bleb, as well as slowly increasing obstruction of the superior vena cava. During the last 5 months of life the patient's coefficient of facility of outflow rose from $C^{o} = 0{,}23$ to $C^{o} = 0{,}53$, while at the same time his intra-ocular pressure rose from 11 to 18 mmHg. These findings can be interpreted as loss of fibrous tissue, causing an improved outflow, at the same time as increasing superior vena-caval obstruction which caused elevation of episcleral venous pressure and hence of intra-ocular pressure.

At present 54 patients with anterior implants, inserted during 1970 - 1972, and 93 patients with long tube implants, inserted from January 1973 to September 1975, attend regularly for follow-up at intervals of 3 - 6 months. Continuous follow-up of these patients has shown that the long-term result of these implants is a gradual fall in intra-ocular pressure, until the decay of fibrous tissue with the development of lymphatics and new aqueous veins has advanced to the stage where essentially free communication exists between the bleb cavity and the episcleral venous system. In these circumstances the intra-ocular pressure is likely to remain very slightly higher than the episcleral venous pressure (8 - 12 mmHg), without the need for hypotensive medication for the rest of the patient's life.

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