Fig. 3. *A titanium retinal tack, on a 1p coin to show the scale.*

ocular toxicity enabling them to be safely retained in the eye. This has been demonstrated by work on an animal model[5] in which minimal tissue response was found histologically following tack insertion. In addition published results report no adverse effects after tacks have been left in (human) eyes, for up to 18 months.[6] Various modifications of the original design have been devised, of which the inclusion of a barb is the most notable. This serves to anchor the tack to the sclera, minimising the risk of extrusion. Retinal tacks may be used either temporarily as a means of manipulating mobile retina intraoperatively or permanently to achieve prolonged (mechanical) retinal fixation.

Complications arising from the use of retinal tacks are recognised and instances of tack intrusion are documented in the literature. Lewis *et al.*[7] described cases in which retinal tacks became displaced, into both the subretinal and preretinal spaces as well as the anterior chamber. The ensuing complications included retinal phlebitis, vitreous haemorrhage, focal corneal damage and corneal oedema. In Lewis's series, the commonest cause of tack intrusion was found to be persistent tissue proliferation. This resulted in displacement of tacks into the globe and frequently coexisted with retinal redetachment. It was suggested that other factors contributing to intrusion were the design of the tacks and the surgical technique used. Non-barbed tacks, often of short shaft length, had been employed. Whilst the absence of a barb facilitates tack insertion it cannot prevent inadvertent dislocation of the tack into the globe. Likewise a shorter shaft precludes complete scleral penetration resulting in a more tenuous scleral anchorage for the retinal tack. Similarly surgical technique may be of importance as non-perpendicular tack insertion will result in incomplete scleral penetration. Placement of an explant after insertion of non-barbed tacks may displace tacks intraocularly and should be avoided.

In summary, retinal tacks are useful in certain circumstances, although permanent insertion may result in late complications as illustrated by this, the first described case of consecutive tack dislocation.

O. J. Lehmann, FRCOphth
C. R. Canning, FRCOphth

Southampton Eye Unit
Southampton General Hospital
Tremona Road
Southampton SO16 6YD UK

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Sir,

Encysted Tenon's Bleb over Superiorly Placed Single-Plate Molteno Implant Causing Proptosis and Strabismus

Molteno implants are often used to control intraocular pressure when other treatment modalities have failed. They are associated with a number of complications, including encapsulation of the filtering

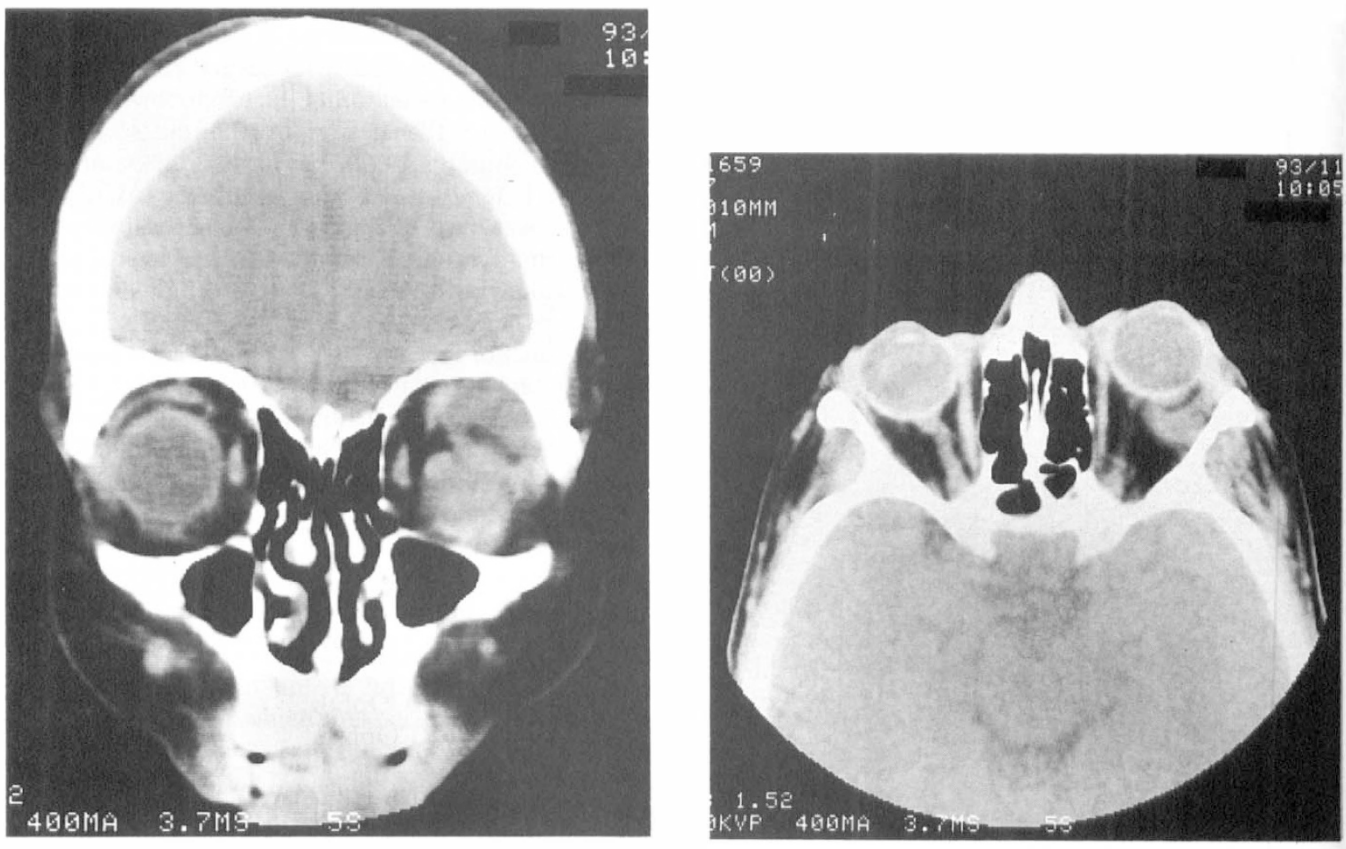

Fig. 1. *CT scans of the orbits showing the size and posterior extent of the encysted bleb. The radiolucent area within the bleb is the Molteno plate.*

bleb and Tenon's cyst formation.[1–4] A previous report of ocular motility problems caused by inferiorly placed double-plate Molteno implants has suggested that superiorly placed implants do not run the same risk because of the greater capacity of the upper orbit.[3] We report a case of encysted Tenon's bleb over a superiorly placed single-plate Molteno implant causing proptosis and strabismus.

Case Report

In October 1987, the iris-clip implant in the left eye of a 67-year-old woman dislocated posteriorly. A soft contact lens was tolerated until 1990 when she developed superficial punctate staining of the cornea. During the course of assessment for secondary anterior chamber implant, her intraocular pressure was found to be 43 mmHg. She was started on pilocarpine 2% q.i.d. and given two sessions of argon laser trabeculoplasty in 1991. The pressure remained elevated. By January 1992 the pressure had risen to 40 mmHg despite acetazolamide. We proceeded to left trabeculectomy and secondary anterior chamber implant. By January 1993 the intraocular pressure had risen to 25 mmHg with an encysted drainage bleb and by May 1993 the pressure had reached 29 mmHg on pilocarpine 4% q.i.d. The drainage bleb was refashioned in June 1993 and following 6 weeks of normal intraocular pressure the pressure rose acutely to 50 mmHg in mid-July 1993.

A single-plate Molteno implant was placed in the superotemporal quadrant posterior to the equator of the globe with initially satisfactory control of pressure. By September 1993 the pressure started to rise once more to 33 mmHg, this time associated with horizontal diplopia. The cyst around the plate was deroofed and most of the fibrotic capsule excised. Within 2 months the diplopia had returned, this time with a degree of proptosis and intraocular pressure of 28 mmHg. A CT scan of the orbits showed the size and posterior extent of the encysted bleb. The radiolucent area within the bleb is the Molteno plate (Fig. 1). On 23 December 1993 the tube was withdrawn from the anterior chamber. Two weeks later trabeculectomy was repeated with 5-fluorouracil. When the patient was last seen on 23 February 1994 intraocular pressure was 22 mmHg on betaxolol 0.5% b.i.d. and acetazolamide slow-release 250 mg b.i.d. She is developing intolerance to acetazolamide and cycloablative procedures are being considered.

Comment

The diagnosis of Tenon's cyst formation is based mainly on clinical observation.[1,4] Tenon's cyst

formation and bleb encapsulation are common complications following glaucoma filtering surgery.[1,2,4] Significant ocular motility disturbance following placement of Molteno implants has been reported previously, especially when the implant has been placed inferiorly.[3, 5] Hypertropia after superior placement of the implant has also been reported,[6] possibly due to a fat adherence syndrome, but not associated with proptosis. Possible risk factors for the development of encysted bleb include prior argon laser trabeculoplasty, previous trabeculectomy complicated by Tenon's cyst formation, male gender, female gender with age over 50 years, prolonged use of sympathomimetic antiglaucoma medication and black race.[1, 4]

Treatment of Tenon's capsule cyst causing raised intraocular pressure includes repeated needling through the conjunctiva, excision and marsupialisation of the cyst, and use of a second Molteno implant without excision of the cyst. Mild displacement of the globe may be adequately treated by the use of prisms. A recurrence of the cyst or sustained rise in pressure may require cycloablative procedures.[1,3]

J. Pandit
M. Graham

Ophthalmology Department
Torbay Hospital
Torquay, Devon TQ2 7AA
UK

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Sir,

Prevalence of Chronic Hypokalaemia Amongst Elderly Patients Using Acetozolamide and Diuretics

Chronic open angle glaucoma is a common condition in old age, affecting 5% of those over 65 years of age, and is responsible for 15% of registrations for blindness in this population group.[1–3] Acetozolamide is a carbonic anhydrase inhibitor and has been used to treat glaucoma for many years. It also acts on the renal tubules where it increases the excretion of bicarbonate and cations, chiefly sodium and potassium. Thus it promotes an alkaline diuresis and a metabolic acidosis. It has been shown that long-term therapy results in a compensatory increase in bicarbonate resorption by the proximal tubule. This limits the acidosis and the diuresis induced by acetazolamide. Overall in humans there is an initial risk of hypokalaemia and/or hyponatraemia when taking a therapeutic dose of acetazolamide, but there is little risk of hypokalaemia in the long term.[4] However, in elderly patients there are several reasons for concern, namely that acetazolamide accumulates in elderly people,[5] that there is a decline in renal function with age (glomerular filtration rate shows a linear fall)[6] and that elderly patients frequently have multiple medical problems which require multiple therapies some of which may also have an effect on serum potassium levels (e.g. other diuretics, digoxin and ACE inhibitors).[7] Finally, the elderly may eat a diet low in potassium.[8] We were, therefore, particularly concerned that chronic hypokalaemia could develop in an elderly population on chronic acetazolamide therapy.

We undertook a prospective control study at Mayday Hospital, Croydon, of consecutive patients attending all ophthalmology clinics over a 1 month period who were over 60 years of age and on long-term acetazolamide therapy (defined as treatment for >6 months). Each patient was reviewed in clinic and the following details recorded: age, sex, ophthalmological diagnosis, duration of acetazolamide therapy, history of cardiac and/or renal disease and the number and dose of other medications. Blood was taken for estimation of plasma sodium, potassium, urea and creatine levels. A control group matched for age and sex was recruited from amongst patients about to undergo routine surgical procedures. The same details were recorded and blood tests performed.

We recruited 16 patients (8 women and 8 men) with an age range of 60–85 years (mean 70.9 years). They had used acetazolamide for a mean of 4.7 years (range 0.5–16 years). Seven used the sustained release preparation. Six of the study group were

Table I. Mean values of electrolytes in study and control groups

	Study group ($n = 16$)	Control group ($n = 16$)	p value (Student's t-test)
Sodium (mmol/l)	139.6 SD 2.69 Range 135–144	138.3 SD 3.44 Range 132–143	NS
Potassium (mmol/l)	4.28 SD 0.32 Range 3.8–4.9	4.15 SD 0.34 Range 3.6–4.7	NS
Urea (mmol/l)	7.63 SD 2.36 Range 5.0–13.6	7.09 SD 1.94 Range 4.4–12.3	NS