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]

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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

receiving diuretic therapy, 2 were receiving ACE inhibitors and 1 was on potassium supplementation. Ten gave a history of heart disease, 5 of heart failure, 4 of hypertension and 4 of angina. Only 2 of the study group had had their plasma potassium level measured in the year prior to the study. Only 1 patient was receiving regular potassium supplements.

We found no patient in the study or control groups to be hypokalaemic and no significant difference in mean potassium concentrations between the study and control groups (Table I). There was also no significant difference in mean potassium levels between those receiving acetazolamide and diuretics and those receiving acetazolamide alone (study group mean, 4.21 mol/l; control group mean, 4.18 mmol/l). The mean potassium level in those using the acetazolamide sustained release preparation was slightly higher than in those receiving the normal formulation (study group 4.37 (range 4.0–4.9); control 4.06 (range 3.8–4.5); $p = 0.047$).

We were reassured to find no difference in mean potassium concentrations between study and control groups and that no patient was hypokalaemic. That there was no significant difference in mean potassium levels amongst those receiving diuretic therapy is more important. That there was a small, but statistically significant higher mean potassium level amongst those receiving the sustained release formulation seems of little importance as no patient was hypokalaemic and the mean value was well within the normal range.

We conclude that we can find no evidence that long-term acetazolamide therapy given to elderly patients is associated with hypokalaemia, even if they are also receiving diuretic therapy. Regular electrolyte estimation would seem unnecessary.

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Sir,

Superior Oblique Myokymia Masquerading as an Inferior Rectus Palsy

A 44-year-old woman was referred by her optometrist because of a 5 year history of intermittent vertical diplopia and the finding of a variable right hypertropia. An associated 'shimmering' of vision in the left eye had been diagnosed as migraine but treatment with Sanomigran (Sandoz) had failed to relieve this. She was in otherwise good general health, on no regular medication and with no other past ocular problems or familial ocular disorders. There was no history of head trauma.

Unaided visual acuities were 6/6 in each eye. There was a slight and variable abnormal head posture with chin depression and tilt to the right. With this head posture a cover test showed a small exophoria and right hyperphoria for both distance and near gaze. On correcting the head posture the vertical deviation became manifest and in the primary position measured 4 prism dioptres (PD). Ocular movements suggested a mild underaction of the right eye on dextrodepression, supported by the Hess chart which showed underaction of the right inferior rectus (Fig. 1). However, close inspection of the eyes in good lighting revealed intermittent intorsion movements of the left eye and a diagnosis of left superior oblique myokymia (superior oblique microtremor).

A cerebral CT scan, with particular attention to the brain stem tectal plate, showed no abnormality. The patient was commenced on oral propranolol; a starting dose of 40 mg was raised in increments of 20 mg fortnightly and symptoms eventually resolved at a level of 80 mg daily (Half-Inderal LA, ICI).

This case of superior oblique myokymia was unusual in that it was associated with a sustained increased tone in the superior oblique resulting in an overaction of this muscle. This in turn gave the impression of an underaction of the contralateral inferior rectus. The case highlights the importance of