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

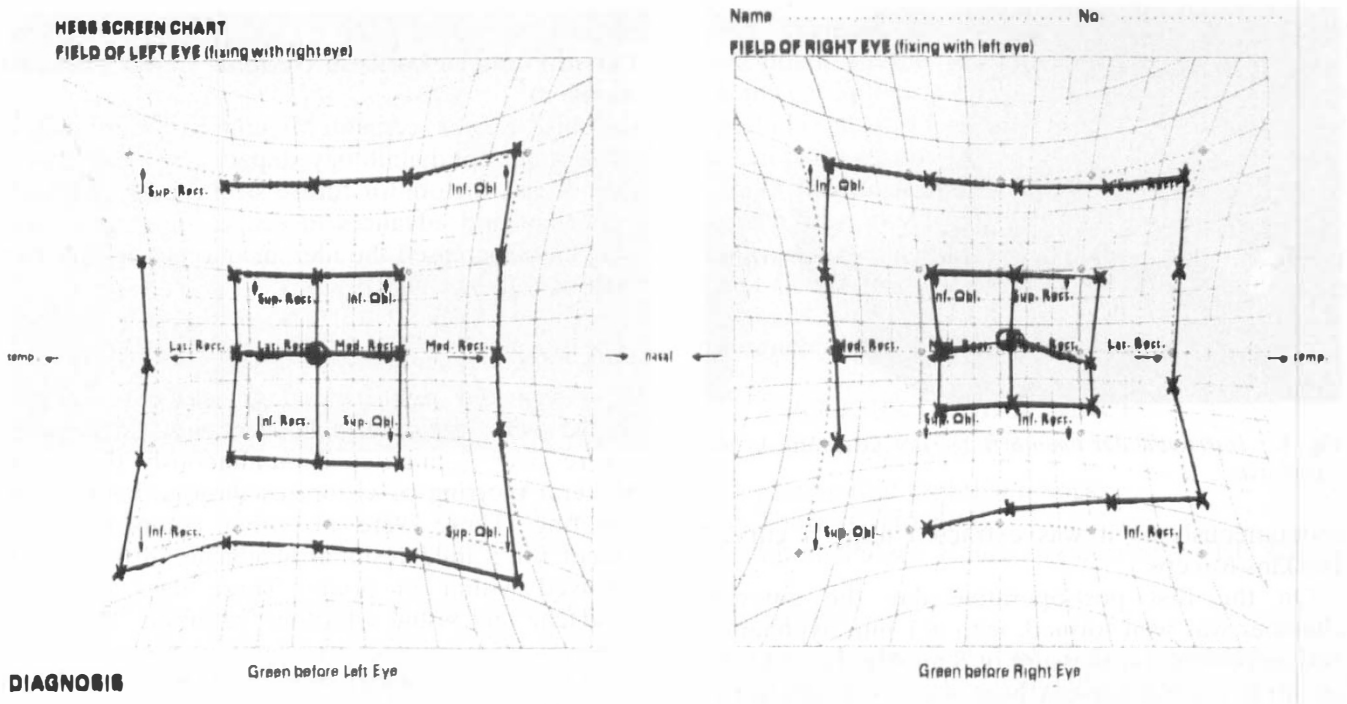

Fig. 1. *The left eye shows overaction of the superior oblique and the right eye underaction of the inferior rectus.*

inspection of the eyes at close range when assessing disorders of ocular movement.

The typical features of superior oblique myokymia are intermittent vertical diplopia and monocular oscillopsia.[1,2] The aetiology is unknown although two mechanisms have been proposed.[1] Tetanic bursts of action potentials may arise from the vicinity of the trochlear nucleus due to previous injury, inflammation or irritation. Alternatively it may be a reinnervation phenomenon following trauma to the IVth nerve with subsequent enlargement of superior oblique motor unit size.[4,5] Midbrain astrocytoma has been associated with the disorder and if there is no history of trauma a CT scan is advisable.[3]

This case also emphasises the value of propranolol in some cases of superior oblique myokymia and indicates that success with the drug may depend on experimentation with the dose.[6]

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Intralenticular Haemorrhage Complicating Trabeculectomy

Trabeculectomy is a commonly performed operation for glaucoma. The complications are well known and include hyphaema, hypotony, flat anterior chamber, and formation or acceleration of cataract.[1] We report a case in which this procedure was complicated by intralenticular haemorrhage.

Case Report

A 48-year-old man underwent a right trabeculectomy for primary open angle glaucoma not controlled by conventional medical treatment. A standard procedure was performed at the 12 o'clock position under local anaesthesia using 1 ml of 2% lignocaine injected subconjunctivally. The iris did not prolapse