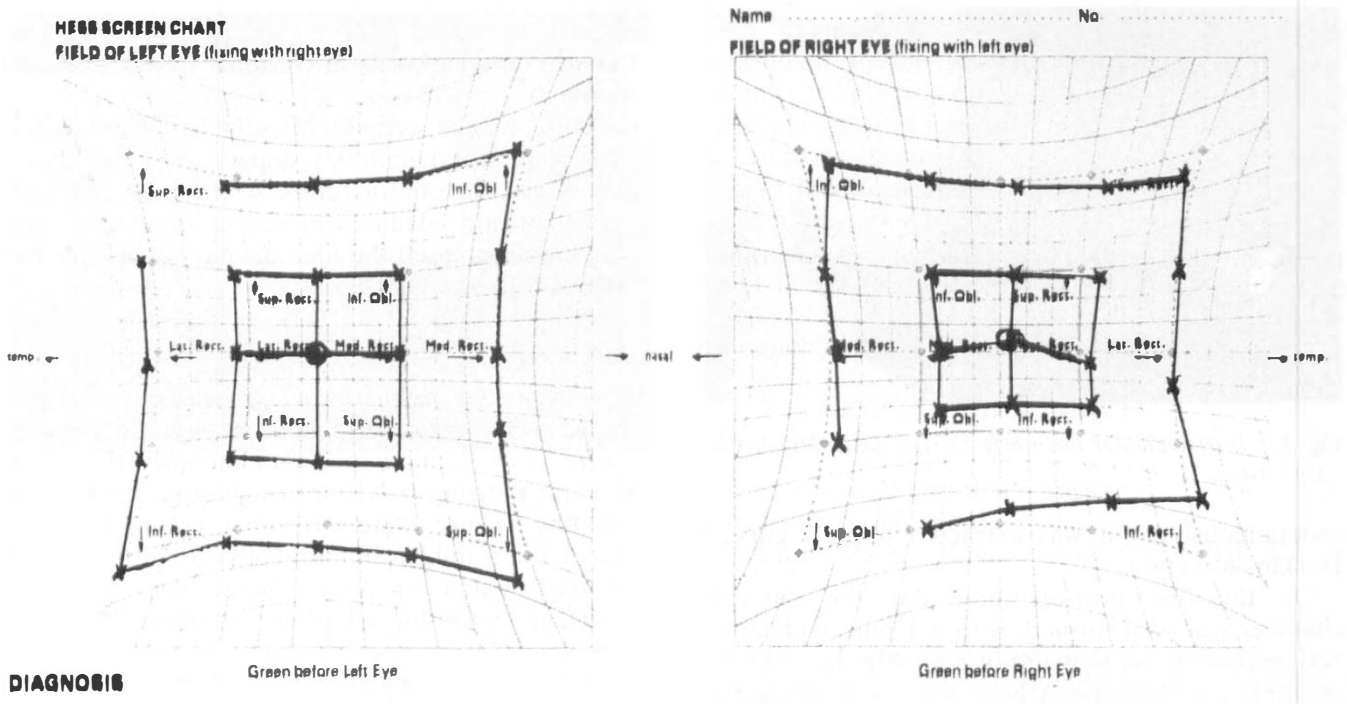

Fig. 1. *The left eye shows overaction of the superior oblique and the right eye underaction of the inferior rectus.*

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

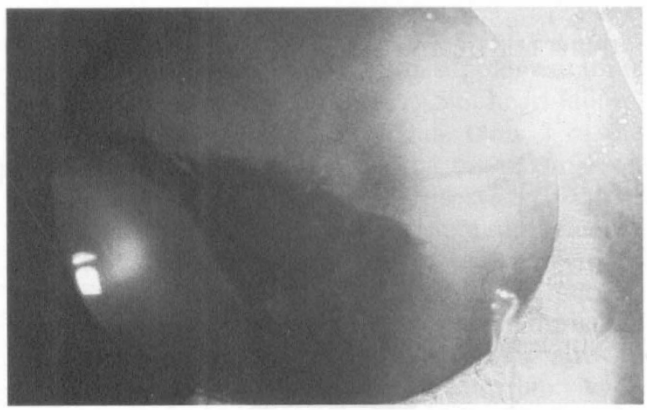

Fig. 1. *Intralenticular haemorrhage not extending to the visual axis.*

spontaneously so it was extracted using a curved Hoskins forceps.

On the first post-operative day the anterior chamber was well formed, with a 1 mm hyphaema, and an intraocular pressure of 8 mmHg. In addition, an intralenticular haemorrhage was noted superiorly, lying between the anterior lens capsule and the cortex. This did not extend to the visual axis (Fig. 1). This clot has reduced in size over an 8 month period, with no evidence of cataract formation. The visual acuity remains 6/6 uncorrected.

Discussion

We presume that, at the time of the peripheral iridectomy, the lens capsule was inadvertently lifted and breached, resulting in blood tracking downwards between the anterior lens capsule and the cortex. A capillary effect drawing blood into the lens as it flowed over the breach in the capsule is a possible mechanism.[2]

This case highlights the importance of careful extraction of the iris from the anterior chamber in cases where it does not prolapse spontaneously, in order to avoid inadvertent damage to the underlying structures.

To our knowledge this complication of intralenticular haemorrhage following trabeculectomy has not been reported previously.

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Sir,

Patent Foramen Ovale in Occipital Cerebrovascular Accident

Cerebrovascular accidents affecting vision frequently present to ophthalmology departments. Identification of risk factors for future stroke prevention are important and advances in echocardiographic imaging have increased the identification of cardiac risk factors.

Case Report

A 45-year-old right-handed Caucasian man presented with occipital pain becoming moderately severe over 2 minutes. Simultaneously there was bilateral blurring of vision, oscillopsia, nausea and vomiting. There were no other complaints and, except for a mild residual headache, all symptoms resolved within 24 hours. Three days later the headache and visual symptoms returned. This time, however, a few minutes after onset he noticed that he was unable to see objects in the right half of his visual field. The headache and oscillopsia again settled during the day but the visual field loss persisted.

There was no significant past medical history or family history, in particular no migraine, cardiac disease or cerebrovascular disease. He was on no medication, and was a non-smoker with moderate alcohol intake.

Visual examination was entirely normal apart from hypometropic saccades to the right and a dense right-sided homonymous hemianopia with macular sparing. General and neurological examination was otherwise normal; there were no cardiac murmurs or carotid bruits. His blood pressure was 130/70 mmHg.

All routine haematological and biochemical investigations were within normal limits. Serology for syphilis and antinuclear antibody was negative. Cholesterol was 5.06 mmol/l and triglycerides 1.96 mmol/l. An ECG showed sinus rhythm with normal axis and no ischaemic changes. His chest radiograph and a transthoracic echocardiogram (TTE) were normal. An MRI brainscan was also normal. Transoesophageal echocardiography (TOE) demonstrated a patent foramen ovale (PFO) with paradoxical shunting during a Valsalva manoeuvre (Fig. 1). There was no clinical evidence of deep venous thrombosis or pulmonary emboli. He was anticoagulated with warfarin and given low-dose aspirin. Within 48 hours the hemianopia had shrunk to a right upper quadrantanopia and had completely resolved when reviewed 3 weeks later.

Discussion

This case had a patent foramen ovale in association with a cerebral ischaemic episode. This may enable emboli of venous origin to cross into the arterial