Discussion

Was this vitreous haemorrhage a coincidence or had the hyperbaric oxygen exposure affected the patient's retinal vasculature in some way? Elevated arteriolar oxygen tensions are known to cause retinal vasoconstriction in this autoregulated circulation, but this reactivity has been shown to be markedly decreased in hypertension and in diabetes. Arteriolar vasoconstriction on breathing 100% oxygen was decreased from 11.5% in normal subjects to approximately 3% in both hypertensive and diabetic subjects using fundus photography for measurement.[2] Laser Doppler techniques have shown that in normal subjects 5 minutes of 100% oxygen breathing produces a 63% decrease in retinal blood flow, while in diabetic patients blood flow decreases by only 36%.[3] It has also been shown that this lack of reactivity to oxygenation is partially dependent on the blood sugar concentration at the time and that restoration of blood sugar level to normal values will improve retinal reactivity.[4]

If vasoconstriction in the presence of hyperoxia is a protective response, either against oxygen-driven free radical damage or against the shear and circumferential stresses of increased blood flow,[4] then as diabetics are deficient in this response, the fragile neovascular fronds of proliferative retinopathy may be being placed at risk of haemorrhage when treated with hyperbaric oxygen. As a precaution we have adopted the procedure in our chamber whereby all patients with proliferative or pre-proliferative diabetic changes have their blood sugar levels normalised at the time of treatment and any hypertension controlled.

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Sir,

Epiretinal Membranes Presenting in Two Young Adults with Evidence of Persistent Primary Vitreous

Two males of 19 and 24 years presented with uniocular epiretinal membranes and evidence of persistent primary vitreous with a 'Mittendorf's dot' and 'Bergmeister's papilla' in the involved eye. There was no history of previous eye disease. There was little subjective or objective disturbance of visual function. Results of fundal examination and fluorescein angiography were otherwise normal. Such idiopathic epiretinal membranes are uncommon in young persons and are believed to be of a different aetiology from those occurring in older people, being thought to represent persistence of embryonic material. The occurrence of persistent primary vitreous remnants in the involved eyes of these cases supports this view.

Case Reports

Case 1. A 19-year-old male was referred by his general practitioner to the Eye Department complaining of mild headaches and intermittent blurring of the vision in the right eye since falling through a plate-glass window. He had sustained a small laceration to the right zygoma region but there had been no other trauma to the face or eyes. He was generally fit and well.

Examination showed unaided acuities of 6/6 right and 6/5 left. There was no perceived abnormality on observing an Amsler chart. Colour vision was normal using Ishihara plates and visual fields were full to confrontation. The right eye showed a mild opacity on the central posterior surface on the posterior capsule (Mittendorf's dot). The posterior hyaloid membranes remained attached in both eyes. There was a striking membrane overlying the supero-temporal retina and glial tissue on the right disc. There was no underlying macular pucker or distortion of the retinal vasculature, although there was some fine surface wrinkling of the highly reflective inner limiting membrane of the retina (Figs. 1, 2). There were no abnormalities of the left globe. Fluorescein angiography showed no underlying retinal abnormalities (Fig. 3). The membrane was not visible during angiography and therefore transparent to blue/green light.

Case 2. A 24-year-old male was referred to the Eye Clinic by his general practitioner complaining of gradual deterioration of the vision in the right eye. There was no past ocular history, no relevant family history and he was generally well.

Examination showed unaided acuities of 6/12 right (improving to 6/6 part with refraction) and 6/6 left. There was no perceived abnormality on viewing an Amsler chart. Colour vision was normal using Ishihara plates and there was no visual field defect on confrontation testing. There was a marked Mittendorf's dot in the right eye. The posterior hyaloid membranes remained attached in both eyes. There was some tissue overyling the right disc extending anteriorly (Bergmeister's papilla) and a membrane overlying the right macula (Figs. 4, 5). There were no underlying retinal abnormalities evident on funduscopy or fluorescein angiography (Fig. 6) and the membrane did not appear to block background choroidal fluorescence.

Discussion

These two cases show unilateral idiopathic epiretinal membranes. The absence of retinal exudates and peripheral avascular changes characteristic of 'familial exuda-

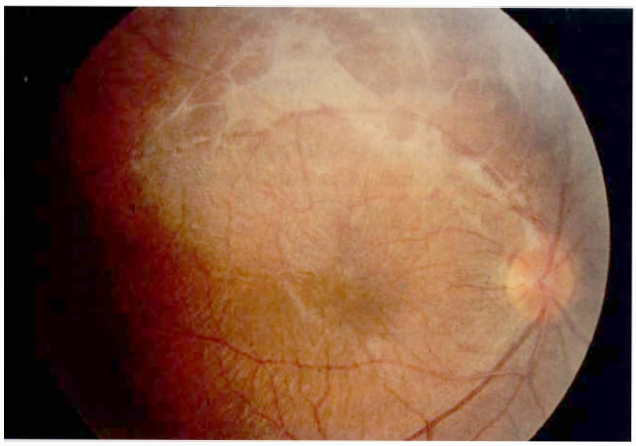

Fig. 1. *Case 1. Fundus photograph of affected eye.*

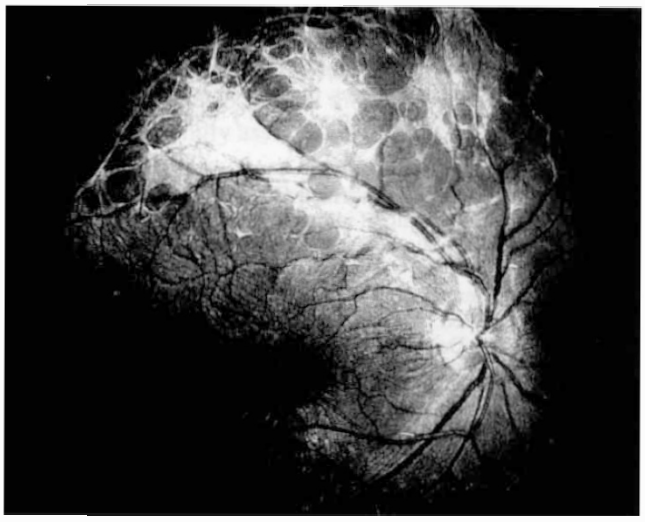

Fig 2. *Case 1. Red-free photograph of affected eye.*

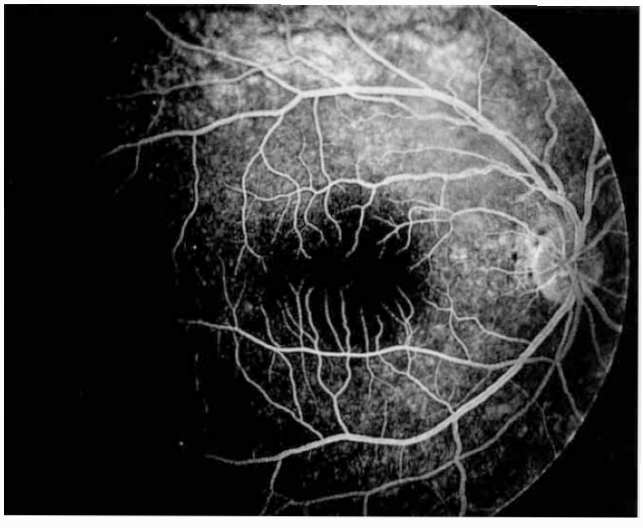

Fig. 3. *Case 1. Fluorescein angiogram (venous phase) of affected eye.*

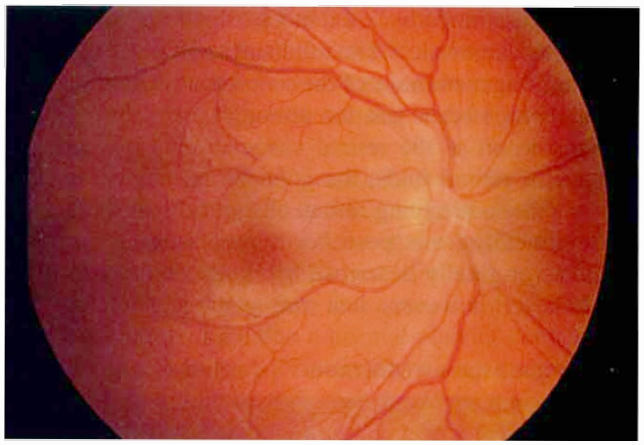

Fig. 4. *Case 2. Fundus photograph of affected eye.*

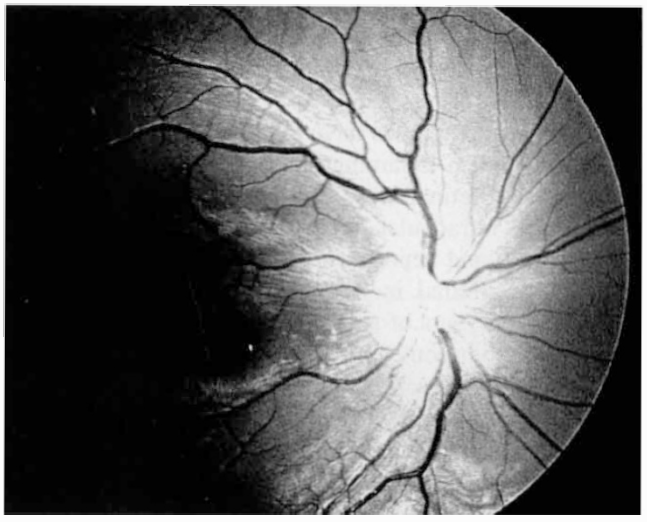

Fig. 5. *Case 2. Red-free photograph of affected eye.*

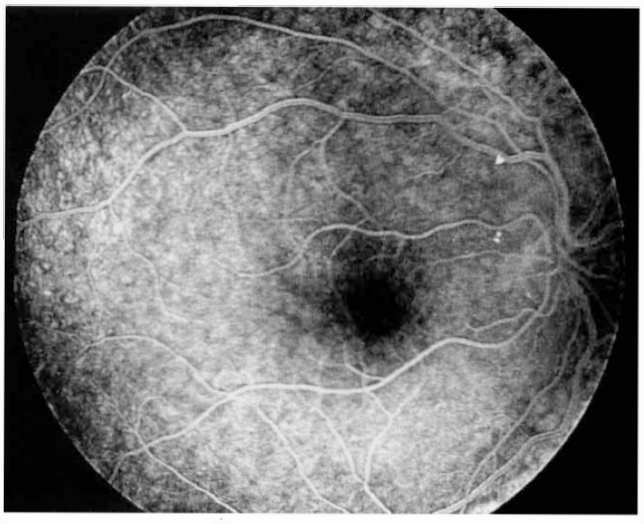

Fig. 6. *Case 2. Fluorescein angiogram (venous phase) of affected eye.*

tive vitreoretinopathy' excludes this entity as an alternative diagnosis.[1,2] The mild surface wrinkling of the inner limiting membrane of the retina in case 1 may represent early membrane traction although there is no associated distortion of the retinal vasculature. However, the absence of retinal folds, the good visual acuity, and the lack of other underlying retinal changes distinguish these cases clinically from 'posterior hyperplastic primary vitreous' as originally described by Pruett and Schepens.[3,4] We believe, however, that the pathogenesis may be similar.

Both cases show a Mittendorf's dot and a Bergmeister's papilla in the involved eye, signs believed to represent remnants of the embryonic primary vitreous. The primary vitreous appears following the formation of the optic vesicle at the 35 somite stage (36 days) in the embryo and forms the hyaloid vascular system to nourish the developing anterior segment. Other signs thought to be associated with it include Cloquet's canal, 'persistent hyperplastic primary vitreous'[5] appearing as a retrolental mass, and posterior hyperplastic primary vitreous' referred to above.

Epiretinal membranes have many causes[6] and include posterior segment inflammation, retinal vascular disease (e.g. CRVO), retinal tears and rhegmatogenous retinal detachment,[7] intraocular surgery, retinal photocoagulation and cryopexy and neurofibromatosis type II.[8] Primary or idiopathic epiretinal membranes are more common in adults over 50 years of age[9,10] and are usually associated with posterior vitreous separation.[9,11,12] The retinal appearances have been described and staged by Gass[13] and can show other associated signs[10,11,13] including macula oedema, pseudomacular holes and subretinal new vessels. Spontaneous regression has been documented[14,15] and vitrectomy and membrane peeling is an established treatment in progressive cases.[16]

Idiopathic epiretinal membranes occurring in young persons are less common than those occurring in adults[15,17,18] and are less often associated with posterior vitreous separation. Wise[17] proposed that such membranes were distinct from those occurring in older individuals, being a congenital abnormality due to persistent adherence of primary vitreous to the inner retina. Such a pathogenetic mechanism was disputed by others[15,18] who believed them to be an acquired abnormality due to glial cell proliferation through the inner retinal surface.

The authors are not aware of any reported cases of idiopathic epiretinal membranes that also show evidence of persistent primary vitreous, and in this respect these cases are unusual. They suggest that such membranes may be due to persistence of the primary vitreous on the inner retinal surface as originally proposed by Wise[17] and not to an acquired retinal process.

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Sir,

Betaxolol-Associated Anterior Uveitis

A 78-year-old woman with rheumatoid arthritis was referred by an orthopaedic surgeon to the Eye Department complaining of blurred vision for 6 months. She had no history of any past ocular problems. Her only systemic medication was ibuprofen 400 mg three times a day.

At the first outpatient visit she was noted to have bilateral blepharitis, early cataracts, accounting for blurred vision, and ocular hypertension, with intraocular pressures of 32 mmHg each eye. The anterior chambers of both eyes were noted to be quiet and with no activity present.

She was advised on lid hygiene and prescribed betaxolol 0.5% twice daily to both eyes to reduce the intraocular pressure. Three weeks later she returned with ocular pain and deterioration of vision. On examination there were