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 causes⁶ 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 Gass¹³ 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. Wise¹⁷ 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 Wise¹⁷ 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

small, fine, white keratic precipitates scattered on the endothelium of the cornea along with anterior chamber activity in both eyes. The intraocular pressures were found to be normal.

The betaxolol was stopped and the treatment changed to timolol 0.25% (Timoptol, MSD) twice a day and fluorometholone drops four times a day. The uveitis resolved over the next 4 weeks on the above treatment.

Granulomatous anterior uveitis has previously been reported with metipranolol (a non-selective beta blocker)¹ but not with other beta blockers, either selective or non-selective. Betaxolol is a cardioselective beta-1 blocker and the most notable feature of its adverse effect profile is transient local stinging and irritation.² Other reported ocular side-effects include decreased tear production,³ chronic conjunctival inflammation⁴ and dendritic ulcer.⁵

Our patient has rheumatoid arthritis, a condition making her more prone to developing uveitis. However, the uveitis started within 3 weeks of starting betaxolol, resolved promptly on stopping it in conjunction with weak steroid drops, and did not reappear on stopping steroids in the follow-up period of 6 months. This suggests that betaxolol is strongly associated with the anterior uveitis in this case.

This is, to the best of our knowledge, the first report of uveitis with betaxolol.

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Sir,

Retinal Changes in Scurvy

Scurvy is now rare in the United Kingdom and eye changes in scurvy are not extensively described.¹ We present a case demonstrating retinal haemorrhage and exudates, which resolved on treatment.

Case Report

A 48-year-old white unemployed divorcee presented with painful bruising in the leg muscles. He reported no visual disturbance but his gums had been bleeding and one tooth

had recently fallen out. For 2 years he had eaten no fresh fruit or vegetables; he subsisted mainly on pasties and tinned soups with some boiled eggs, white bread and milk. He admitted to 20 cigarettes a day and 28 units of beer weekly.

Examination revealed jaundice, poor dental hygiene with gum haemorrhage, petechial and perifollicular haemorrhages on the limbs and extensive muscle bruising particularly affecting the thighs. Funduscopy (Fig. 1) showed flame haemorrhage and areas of soft exudate.

Investigations included a normal platelet count, white blood cell (WBC) and international normalised ratio

(a)

(b)

Fig. 1. Retinal photographs of the left (a) and right (b) eyes at presentation demonstrating exudate and haemorrhage.