despite apparently normal vasculature the haemorrhage may recur during labour. Possible interventions included epidural anaesthesia (which considerably reduces central venous pressure) and/or operative delivery.

In the discussion following Duane's original paper[2] Norton described a case of Valsalva retinopathy in a pregnant woman. Neither in his case nor Callender's case nor our two cases did the retinopathy recur, despite the patients undergoing normal labour. We believe that there is no evidence at present that obstetric intervention is necessary in labour in order to prevent recurrence of the haemorrhage. It is perhaps surprising that in the extreme Valsalva manoeuvre of the second stage of labour this retinopathy has not been reported, though orbital Valsalva haemorrhages have.[5]

James S. Deane, FRCOphth
Department of Ophthalmology
University of Leicester
Clinical Sciences Building
PO Box 65
Leicester LE2 7LX

Nikolas Ziakas, DEBO
Royal Victoria Infirmary
Newcastle upon Tyne
UK

References

1. Callender D, Beirouty ZAY, Saba SN. Valsalva haemorrhagic retinopathy in a pregnant woman. Eye 1995;9:808–9.
2. Duane TD. Valsalva haemorrhagic retinopathy. Trans Am Ophthalmol Soc 1972;52:298–313.
3. Gass JDM. A stereoscopic atlas of macular disorders, 3rd ed. St Louis: CV Mosby, 1987.
4. Schipper I. Valsalvamanover: nicht immer Gutartig. Klin Monatsbl Augenheilkd 1991;198:457–9.
5. Geyer O, Wasserman D, Rothkoff L, Lazar M. Orbital haemorrhage induced by labour. Br J Ophthalmol 1990;74:242.
6. Van Rens E. Traumatic ocular haemorrhage related to bungee jumping. Br J Ophthalmol 1994;784:948.

Sir,

We read with interest the report by Meaney and Ogunsola of a child with orbital tuberculosis.[1] We report a similar case of an orbital mass in a young child due to tuberculosis.

A 10-year-old Somalian girl presented to the ophthalmic department with a right upper lid lump. This had first been noted 4 months previously following minor lid trauma. She complained of ptosis and occasional diplopia. She was systemically well and had no past medical history of note. On examination, she had a 30 mm × 25 mm tender, fluctuant, transilluminant mass in the superotemporal aspect of the right orbit (Fig. 1). Her visual acuities

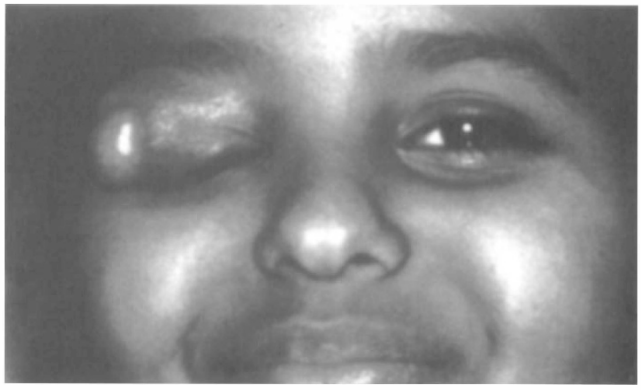

Fig. 1. *The patient at presentation, showing the right upper lid lump.*

were 6/9 bilaterally and 2 mm of right ptosis was observed. She did not have proptosis but abduction of the right eye was limited. The pupils were normal and visual fields were full. She was apyrexial and had no lymphadenopathy. The remainder of the examination was unremarkable.

Her erythrocyte sedimentation rate was raised at 28 mm/h but full blood count, urea and electrolytes, glucose, liver function tests and thalassaemia screen were within normal limits. A chest radiograph was normal but a tuberculin test was positive. An orbital CT scan showed a large bilobed soft tissue mass in the lateral wall of the right orbit displacing the globe downwards and medially. There was irregular destruction of the superior and lateral orbital walls with some hyperostosis (Fig. 2).

As the mass was about to discharge, a decision was made to perform a controlled surgical drain and the aspirate was cultured, growing *Mycobacterium tuberculosis.* Our paediatric colleagues advised consultation with a chest physician. As a result the child was treated with Rifinah 150 (Merrell; 3 tablets daily), pyrazinamide 20 mg/kg daily and ethambutol 25 mg/kg daily for the initial 60 days, followed by 15 mg/kg

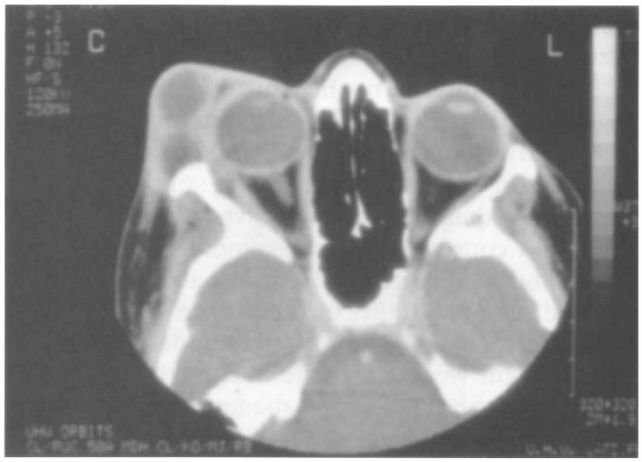

Fig. 2. *CT scan showing a large soft tissue mass in the lateral wall of the right orbit.*

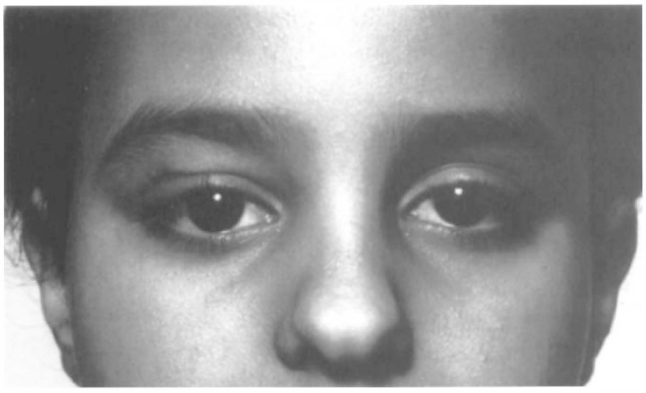

Fig. 3. *The patient at 18 months follow-up.*

daily. This treatment was continued for 9 months and she remains well at 18 months follow-up (Fig. 3).

Many cases of extra-pulmonary tuberculosis present to surgeons. However, chemotherapy is used to treat all types of tuberculosis, whatever the site. We would like to point out the British Thoracic Society's recommendation that the drug treatment of adult patients with extra-pulmonary tuberculosis should be shared with a chest physician and that of children with a paediatrician, unless the surgeon responsible is experienced in the use of modern anti-tuberculosis regimens.[2]

B. N. Roberts, MRCOphth
C. M. Lane, MD, FRCOphth, MRCP

Cardiff Eye Unit
University Hospital of Wales
Cardiff
UK

References

1. Meaney TPJ, Ogunsola AB. Tuberculosis presenting as an orbital mass lesion in childhood. Eye 1995;9:649–50.
2. Ormerod LP. Recommendations of the Joint Tuberculosis Committee of the British Thoracic Society. Thorax 1990;45:403–8.

Sir,

We read with interest the cases reported by Haslett *et al.*[1] and their pathogenetic and medico-legal implications. We would like to contribute to the discussion by describing the case of a patient who had a similar accident while not wearing a seat belt. This introduces an alternative pathogenetic hypothesis.

A healthy 49-year-old man had a car accident while not wearing a seat belt. He reportedly suffered a whiplash injury without any direct cranial trauma. Two weeks later he complained of visual field modification in his left eye. On physical examination he presented 20/20 vision in each eye. On fundus examination the right eye (RE) was normal but the left eye (LE) showed vitreous haemorrhage inferiorly and multiple retinochoroidal haemorrhages inferiorly and nasally to the optic disc (Fig. 1). A- and B-scan echography of the LE confirmed: (1) vitreous haemorrhage; (2) subhyaloid haemorrhage; (3) posterior detachment of the vitreous body; (4) intraretinal and choroidal haemorrhage below the disc; and (5) subretinal haemorrhage nasally. At his most recent examination, 8 months later, the last two lesions had been replaced by a large area of chorioretinal atrophy localised nasally and inferiorly to the optic nerve head of the LE (Fig. 2).

Trauma of various types, even at sites distant from the eye, may involve the retinal vascular system, as previously described by Purtscher[2] at the beginning of this century. Several reports of unilateral or bilateral retinopathies similar to those observed by Purtscher have been reported after compressive thoracic injuries (e.g. seat belt injuries), head trauma and violent deceleration.[3–5] The pathogenesis of these retinal vascular alterations has been attributed to various phenomena, such as a sudden rise in intrathoracic venous pressure,[3] arterial angiospasms, and retinal vessel occlusion by gas and lipid embolisms or aggregates of granulocytes.

In this case, the aetiopathogenesis that we hypothesise is: (1) transient blood flow arrest due to rapid bending of the neck or rapid movement of the head, as in whiplash, both resulting in direct trauma of the carotido-ophthalmic vascular system and retinal vasospasm; (2) acute thoracic compression due not to the use of a seat belt but probably to a rapid muscular contraction with closed glottis, resembling a Valsalva's manoeuvre. Such compres-

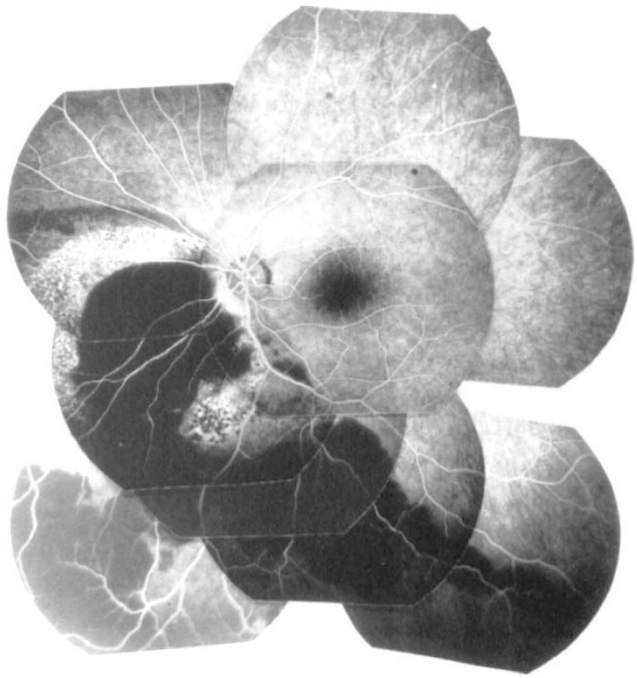

Fig. 1. *LE: multiple retinochoroidal haemorrhages inferiorly and nasally to the optic nerve head.*