

touch. Was the original Fort Bragg fever, therefore, erythema nodosum caused by *L. autumnalis*?

Although *L. canicola* is believed to infect a high proportion of unimmunised dogs, few infections have been described.³ In 1973 only 10 such cases were proved in the British Isles.⁵ Possibly only a minority of leptospiral infections are being detected, since feverish illnesses occur at home, where they are treated by family doctors, often with penicillin. Indeed, our patient was treated with oral penicillin 250 mg four times daily for 10 days.

¹ Turner, L H, *British Medical Journal*, 1969, 1, 231.

² Turner, L H, *British Medical Journal*, 1973, 1, 537.

³ Christie, D B, *Infectious Diseases*, 2nd edn, p 962. Edinburgh, Churchill Livingstone, 1976.

⁴ Heath, C W, Alexander, A D, and Galton, M M, *New England Journal of Medicine*, 1965, 273, 857, 915.

⁵ Turner, L H, *Special Report Part 1 Leptospirosis 1973*, Leptospirosis Reference Laboratory. Central Public Health Laboratory, Colindale, 1973.

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Vitrectomy in subarachnoid haemorrhage

Until recently, persistent vitreous haemorrhage was a blinding condition. We present a case of bilateral vitreous haemorrhage resulting from subarachnoid haemorrhage that developed suddenly but persisted without improvement of vision. Sight was successfully restored by vitrectomy.

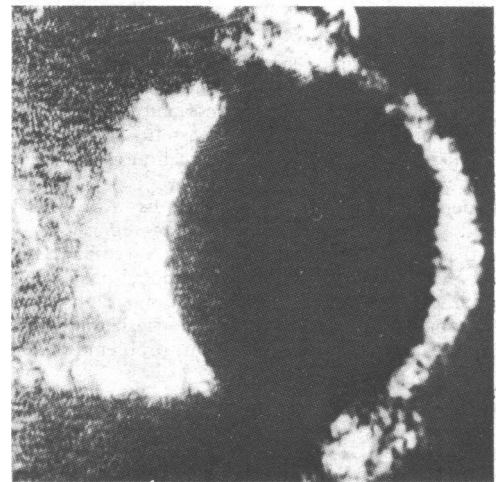
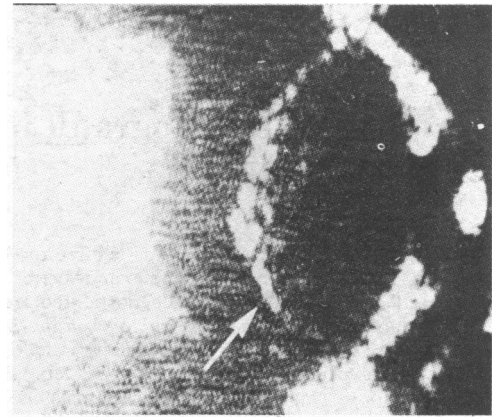
Case report

A previously healthy 34-year-old teacher presented in 1974 with sudden severe headache followed by unconsciousness for 48 hours. He also had bilateral vitreous haemorrhage, which, when consciousness returned, had reduced his vision to perception of hand movements only. His blood pressure was 150/90 mm Hg. Blood values—including full cell count, erythrocyte sedimentation rate, urea, electrolytes, and sugar—were normal, as were x-ray appearances of skull and chest, and the results of electroencephalography, electrocardiography, and bilateral carotid angiography. The cerebrospinal fluid was blood-stained. Vertebral angiography was not performed.

The patient made an excellent general recovery, and his wife said that his rather obsessional personality was more relaxed. The vitreous haemorrhages did not resolve spontaneously, however, and B-scan ultrasonography showed bilateral dense posterior vitreous membranes (see fig). Just over a year after the subarachnoid haemorrhage he underwent staged bilateral vitrectomies via the pars plana approach, the vitreous infusion-suction cutter being used.⁵ Subsequently the visual acuity in his right eye was 6/60 due to a juxta-papillary retinal fold and premacular fibrosis, but the rest of the retina was normal. This may have been due to a vitreoretinal adhesion resulting from a choroidal haemorrhage at the time of the subarachnoid episode, although this eye had always been the worse eye. He made a spectacular visual recovery in his left eye, however, with a visual acuity of 6/9, and is leading a normal life.

Discussion

Bilateral vitreous haemorrhage resulting from subarachnoid haemorrhage was first described by Terson.¹ Most cases are associated with rupture of congenital internal carotid or anterior circle of Willis aneurysms in young people with otherwise normal blood vessels.² A clot may have obscured the causative aneurysm in our patient. Other, much rarer causes are intracerebral and intraventricular haemorrhages.² A postulated mechanism for subarachnoid, intracerebral, and intraventricular bleeding causing intraocular haemorrhage is an acute massive increase of intracranial pressure. Sudden raised intracranial pressure embarrasses the cerebral circulation, and high-pressure carotid blood is shunted, without time for vascular compensation, through its first intracranial branch (the ophthalmic) into both



B-scan ultrasonograms of left eye showing (top) vitreous opacities and posterior vitreous membrane (arrowed) preoperatively, and (bottom) disappearance of vitreous opacities postoperatively

orbits.² This causes leakage and rupture of venous capillaries, associated with the relative compression of venous exit channels from the globe. The resulting intraocular haemorrhages either are confined to the nerve-fibre layer of the retina (retinal haemorrhages) or lift the internal limiting membrane (so-called subhyaloid haemorrhages), such as may occur after the Valsalva manoeuvre.³ Rupture through the internal limiting membrane causes vitreous haemorrhage.

Spontaneous return to normal vision may occur in these patients, but once a posterior vitreous membrane forms it cannot be absorbed and vision does not return.⁴ Vitrectomy, by means of an instrument inserted into the eye via the pars plana that will cut and aspirate abnormal tissue while maintaining the intraocular pressure by infusion of physiological fluid, offers a fairly simple sight-saving procedure and replaces previous operations, which have had a poor rate of success.⁵

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¹ Terson, A, *La Clinique Ophthalmologique*, 1900, 22, 309.

² Walsh, F, and Hoyt, W, *Clinical Neuro-ophthalmology*, 3rd edn, vol 2, p 1784. Baltimore, Williams and Wilkins, 1969.

³ Valsalva, A M, *De Aure Humana tractatus*, ch 5, sect 8, p 84. Utrecht, vande Water, 1707.

⁴ Blach, R K, *Transactions of the Ophthalmological Society of the United Kingdom*, 1975, 95, 407.

⁵ Machemer, R, Panel, J M, and Buettner, H, *American Journal of Ophthalmology*, 1972, 73, 1.

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