obliterated by fibrosis. Pharyngeal and oesophageal ulceration, and fistula formation, have also been recorded (Levowitz *et al.* 1963, Winter & Duvall 1967, Miglets *et al.* 1978).

Because of this, some authorities recommend radical surgery to remove thorotrast from the neck, in order to prevent complications or to stop them progressing, and to spare the tissues from further exposure to radiation (Winter & Duvall 1967, Muzaffar & Nichols 1975, Chalat & Zane 1966).

Against this argument, there is the considerable problem of operating on a neck where normal anatomy has been obliterated by fibrosis, and where the blood supply to the carotid arterial wall may be compromised; several cases of carotid 'blow-out' following radical excision have been described. Because of this, it is nearly impossible to eradicate all thorotrast from the neck. Furthermore, as Novik (1960) has pointed out, penetration of alpha particles in tissue is less than 1 mm, and since the thorotrastoma comprises largely avascular connective tissue, the incidence of radiation damage is probably extremely small (von Leden 1970, Miglets *et al.* 1978, Brady *et al.* 1960). In addition, whatever management is pursued in the neck, thorotrast deposits will remain in the liver and spleen, where the carcinogenic potential is greatest. Therefore a conservative policy and careful follow up are advised.

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Retinopathy after irradiation and hyperbaric oxygen[1]

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A case is reported of retinopathy following irradiation to a paraocular tumour where hyperbaric oxygen was used in the treatment schedule. An unexpectedly severe vaso-occlusive response occurred for the dose of irradiation given and it is suggested that this may have been due to the synergistic action of hyperbaric oxygen.

Case report

The patient was a 59-year-old Caucasian woman. She originally presented in 1969 with left-sided visual failure and anosmia, and was found to have a large subfrontal meningioma which was resected. Postoperatively she recovered well and, apart from slight left disc pallor, vision returned to 6/5 in both eyes with full visual fields.

She presented again in 1974 with blurring of vision in both eyes, which was worse on the right. At that time best visual acuities were RVA 6/36, LVA 6/18. She had some temporal field loss on the right. There was a 6 mm right axial proptosis but eye movements were unrestricted. The right optic disc showed some swelling and there was segmental optic atrophy of the left optic disc. A skull X-ray showed typical meningiomatous hyperostosis involving the body of the sphenoid, the medial wall of the right middle cranial fossa and the posterior walls of both orbits. An exploratory craniotomy revealed an extensive soft tissue mass and biopsy showed a recurrent meningioma. Postoperatively visual acuities were measured as RVA counting fingers, LVA 6/36 and she had developed a right afferent pupillary defect. She was referred for radiotherapy, receiving 3600 rad of cobalt teletherapy via temporal fields while breathing oxygen at three atmospheres. The radiation was delivered in six fractions over eighteen days. She was subsequently followed by the radiotherapy department and was discharged in 1978, at which time she was registered as partially sighted.

The patient was referred to the Medical Eye Unit at St Thomas' Hospital in April 1983. She gave a three-month history of blurring of what little vision she had left and of persistent floaters in front of her right eye. There was no other relevant past medical or ocular history. General examination revealed a fit woman with no evidence of hypertension. She had bilateral

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anosmia. Visual acuities at that time were hand movements on the right and counting fingers on the left. She had a right afferent pupil defect. There was some mild lenticular nuclear sclerosis. The fundi showed a florid retinopathy. On the right there was marked optic atrophy (Figure 1). Hard exudates were present above and below the macula, surrounding the temporal vessels. Some retinal arterioles were absent below and showed marked sheathing towards the posterior pole. Retinal veins showed beading in parts and sheathing. Ghost vessels were present in the periphery. There were a number of microaneurysms and abnormal capillary buds inferotemporally. Two separate areas of subhyaloid haemorrhage showing fluid levels were present. On the left there was optic atrophy. A large circinate exudate almost completely surrounded the macula. Sheathing, microaneurysms and vessel closure were all present. A fluorescein angiogram showed the features of extensive ischaemia on both sides. An early venous phase picture on the right (Figure 2) showed gross capillary closure. There appeared to be a reduction in the number of superficial capillaries supplying the optic nerve head, with early leakage of dye. Retinal arterioles ended abruptly and an inferior vessel was completely obliterated. There was an early leakage from the capillary buds previously described and a masking defect caused by the subhyaloid haemorrhage. Late pictures showed diffuse microvascular leakage in both eyes.

Investigations at that time showed a normal blood picture. There was no evidence of diabetes. Skull X-rays showed no new features and a CAT scan showed no recurrence of tumour. An inferotemporal photocoagulation was carried out with the argon laser on the right to treat the new vessels. She remains under follow up.

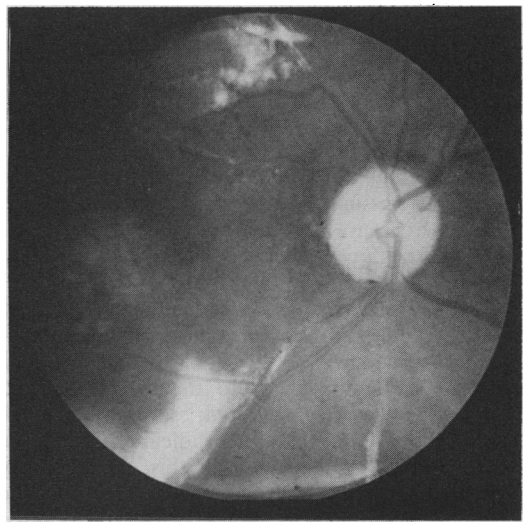

Figure 1. Fundus photograph of the right eye showing optic atrophy, hard exudation, large vessel sheathing and microaneurysms

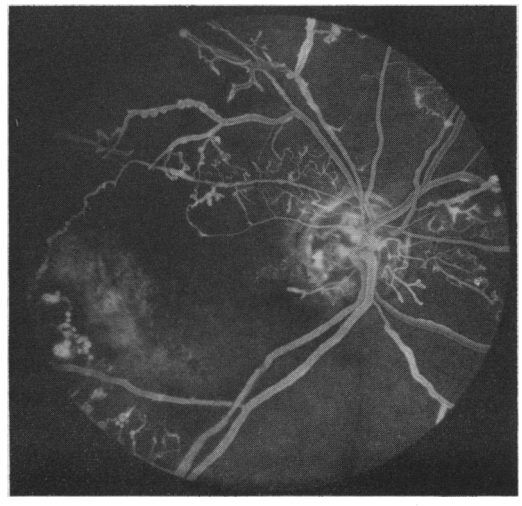

Figure 2. Venous phase fluorescein angiogram of the right eye showing extensive vaso-occlusion with early leakage from the disc and inferotemporal quadrant. Venous beading is prominent superiorly

Discussion

Radiation retinopathy was first reported by Foster Moore (1935), who described a number of cases where a circinate retinopathy appeared in patients who had been treated with radon seeds for the control of intraocular tumours. Since then the features of the retinopathy have been elucidated macroscopically, microscopically and by fluorescein angiography. It may occur after local irradiation to the eye, or after irradiation of paraocular structures. It occurs in an acute or chronic form, the latter being characterized by a variable latent period. The main abnormality found in radiation retinopathy appears to be a selective destruction of the retinal vascular endothelial cell, which causes occlusion of retinal arteries and, subsequently, an ischaemic vasoproliferative response. The main features have been described (Hayreh 1970), most of which appear in this patient. As with any ischaemic retinopathy, hard exudates, microaneurysms, vessel ghosting, peripheral and disc neovascularization are present. The fluorescein study shows widespread capillary closure and microvascular leakage. Sheathing of large vessels, though not pathognomonic, is a typical feature of the retinopathy, and may represent a previous perivasculitis. A reduction in the arterial supply

to the optic nerve head does not usually cause infarction, as the posterior ciliary vessels appear to be relatively radioresistant and usually remain patent.

Those eyes that have been examined pathologically support the concept of an ischaemic process. There is inner retinal layer necrosis with loss of ganglion cells and hyaline degeneration in the wall of the retinal arterioles (Egbert *et al.* 1980). The severity of the retinopathy appears to be dose-related. There is some debate about the exact dose of radiation that will produce change. Duke-Elder (1972) suggests that more than 3000 rad to the posterior segment will lead to some degree of retinal damage. Reported doses of irradiation to paraocular structures causing a retinopathy are generally more than 5000 rad (Shukovsky & Fletcher 1972, Chan & Shukovsky 1976, Chee 1968). It is now becoming apparent that though the total dose of irradiation is important in this respect, other factors such as fractionation, type of irradiation and the time course of delivery all play a part (Aristizabal *et al.* 1977).

Hyperbaric oxygen also produces marked effects on the eye (Nichols & Lambertsen 1969) which may be reversible or irreversible. A high partial pressure of oxygen causes retinal vasoconstriction. However, the retina becomes hyperoxygenated due to the increase in dissolved oxygen in plasma, and this is seen clinically as the venous circulation becomes arterialized. A reversible constriction of the visual fields has been observed in all patients undergoing treatment. There are now several reports of a reversible index myopia (Lyne 1978).

Irreversible effects of hyperbaric oxygen depend on the maturity of the retina. It may induce retrolental fibroplasia in the young of most mammals including man. In the adult experimental animal hyperbaric oxygen causes a toxic inactivation of respiratory enzymes, selective rod cell death and reduction of the electroretinogram. Prolonged administration leads to iritis, hypotony and exudative detachments in dogs. More recently it has been associated with the formation of nuclear cataracts in man (Palmquist *et al.* 1984).

This is the first case of radiation retinopathy reported where hyperbaric oxygen was used in the treatment schedule. Compared with other cases reported in the literature, it presents a particularly severe vaso-occlusive picture, despite the relatively low dose of irradiation given. It has been postulated since 1954 (Gerschman *et al.* 1954) that hyperbaric oxygen and X-irradiation may have a synergistic effect, and both have been shown to affect the retina through a selective metabolic blockade of respiratory enzymes. It is concluded that the retinopathy produced in this unique case is, in part, due to this synergistic action.

Acknowledgment: I would like to thank Mr M Sanders for permission to report this case.

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Polyarteritis nodosa presenting as posterior ischaemic optic neuropathy[1]

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An account is given of a patient who presented with a posterior ischaemic optic neuropathy (PION) and was later diagnosed as suffering from polyarteritis nodosa. It is rare for patients with this disease to present with ocular symptoms in the absence of systemic features of the disorder; furthermore, no documented record of PION occurring in polyarteritis could be found in the English literature.

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