Correspondence

Cranial nerve palsy following retrobulbar anaesthesia

SIR, Retrobulbar injection of anaesthetic agents is seldom associated with serious sequelae. Complications such as retrobulbar haemorrhage, central retinal artery occlusion, scleral perforation, grand mal seizures, and cardiopulmonary arrest have, however, been described. We wish to report an additional complication of retrobulbar anaesthesia: temporary contralateral second and third cranial nerve dysfunction accompanied by a depressed sensorium.

Case report

A 66-year-old white male without significant past medical history complained of sudden loss of his inferior visual field in his left eye. Best-corrected visual acuity was 20/30 in the right eye and 20/40 in the left eye. Pupils were equal round and reactive to light and versions were full. Slit-lamp biomicroscopy of the anterior segments revealed early nuclear sclerosis. Tensions by applanation tonometry were 14 mm Hg in both eyes. Funduscopy examination was remarkable for lattice degeneration in both eyes and a superior retinal detachment in the left eye. While dilated, retrobulbar anaesthesia was administered to the left eye using 3 ml of 2% lignocaine without epinephrine and a disposable ultrasharp tip needle. Air was then injected into the left eye to tamponade the retinal break located at the 12 o'clock meridian. After the procedure was completed the patient complained of decreased vision in his other eye and became disorientated. Visual acuity in the right eye was hand motion. The patient was unable to elevate, depress, or adduct the eye.

Right cranial nerves four and six were intact. Blood pressure, pulse, and respiratory rate were stable, and there were no other neurological findings except for an altered mental status. He was both incoherent and disorientated to time and place. Approximately one hour after the onset of symptoms the patient's visual acuity improved to 20/40 in the right eye, and versions were again full. He became coherent and orientated to time and place.

Discussion

Injection of local anaesthetics into the optic nerve sheaths and subsequently into the intracranial cavity has been proposed as the mechanism of central nervous system complication following retrobulbar anaesthesia. In five patients who underwent retrobulbar injection, orbitography demonstrated radio-opaque dye penetrating the optic nerve sheaths and tracking into the subdural space.¹ The contrast material outlined the optic nerve and extended through the optic canal into the middle cranial fossa. In all cases the injection was believed to be uncomplicated. This pathway has similarly been demonstrated in cadavers.²

Animal studies have shown that direct injection of anaesthetic into the optic nerve results in nerve fibre degeneration.³ Paulter *et al.* reported two cases of marked

visual loss resulting from retrobulbar injections presumably into the optic nerve sheath.⁴ Callahan suggests that the likelihood of penetrating the optic nerve is increased when disposable needles are used.⁵ These needles are ultrasharp, and consequently it is difficult to appreciate the resistance of the optic nerve sheath.

The patient presented illustrates contralateral second and third cranial nerve dysfunction as a complication of retrobulbar anaesthesia induced with an ultrasharp needle. We believe a significant concentration of anaesthetic reached the optic nerve chiasm and/or contralateral optic nerve to block nerve conduction. The contralateral nerve palsy resolved once the anaesthetic was metabolised. The patient's disorientation suggests that the anaesthetic agent also entered the subarachnoid space and the cerebrospinal fluid. Recently, Antoszyk and Buckley reported three cases of contralateral second and third nerve palsy,⁶ and Friedberg and Kline described two cases of contralateral second nerve palsy⁷ following retrobulbar anaesthesia using ultrasharp needles. Many authors recommend the use of blunt tip needles and having the patient look straight ahead or down and outward during retrobulbar injection to minimise the chance of penetrating the optic nerve sheath.^{5,6} We feel this will reduce the incidence of complications following retrobulbar injection.

RAND RODGERS
JUAN ORELLANA

Mount Sinai School of Medicine
and Beth Israel Medical Center,
New York,
USA

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Branch retinal arteriolar occlusion with chicken-pox

SIR, A 19-year-old male who had developed the typical rash of chicken-pox three days previously was referred by his general practitioner after noticing a 'haze' in the nasal field of his left eye that day. On examination his visual acuity was 6/4 in each eye unaided. Examination of his visual fields to confrontation revealed a left inferonasal quadrantic defect. The eyes were quiet and the pupillary responses were normal. Fundal examination revealed a left superotemporal branch arteriole occlusion, with a corresponding area of

Fig. 1 Left fundus on admission.

Fig. 2B Late fluorescein angiogram of left fundus.

Fig. 2A Early fluorescein angiogram of left fundus.

Fig. 3 Left fundus five months later.

oedema (Fig. 1). Immediate treatment with intravenous acetazolamide and eye massage proved unsuccessful. General examination was unremarkable, apart from a just palpable liver which resolved within five days. The following investigations gave normal results: full blood count and platelets, erythrocyte sedimentation rate, plasma viscosity, protein electrophoresis, fasting glucose and lipids, serology for syphilis, and urine analysis. Liver function tests revealed raised aspartate transaminase and γ -glutamyl transferase levels, but these fell markedly in five days and were entirely normal after a further week. Fluorescein angiography (Figs. 2 A, B) confirmed the arteriolar obstruction with delayed filling of the corresponding vein. Late films demonstrated a

slight leakage of dye around that vein, but we did not consider this signified a focal vasculitis.

The retinal oedema settled over the subsequent four months, though the appearance of the arteriole remained the same (Fig. 3), and a visual field test after five months showed that the defect remained unchanged (Fig. 4). Throughout this period his central visual acuity was unaffected.

Discussion

Serious complications following chicken-pox are fortunately rare¹ and the published reports of visual disturbances

Fig. 4 Left visual field five months later.

occurring with it are few.² One case was associated with unilateral³ and two with bilateral^{4,5} optic nerve involvement, and one other was in a patient with bilateral papillitis and a unilateral macular lesion,⁶ but all reported a good visual recovery. The only report of a permanent visual field defect⁷ was in a 16-year-old girl, occurring on the fourth post-eruptive day. This is similar to our patient, who developed a field defect three days after the onset of his rash. In that report the field defect was associated with segmental optic disc pallor followed by pigmentation, and the authors suggested that this was caused by a focal optic neuritis. This may have had an ischaemic basis and therefore could represent a similar process to the arteriolar obstruction in our patient. Although there are no reports of retinal arteriolar occlusion associated with varicella zoster, many complications of the closely related herpes zoster virus may have an ischaemic basis,⁸ either a retinal vasculitis or perivasculitis⁹ or a granulomatous angiitis affecting the brain.¹⁰ In addition the acute retinal necrosis syndrome, which includes an arteritic component, has been linked with a member of the herpes virus group.¹¹ However there was no evidence of a more widespread vasculitis in our patient, and no source for an embolus was found. The exact cause of the retinal arteriolar occlusion in this case remains unknown, but its coincidence with chicken-pox is interesting. To the best of our knowledge this is only the second reported case where a permanent visual field defect has occurred.

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C E HUGKULSTONE

L L WATT

Department of Ophthalmology,
Queen Mary's Hospital,
Frogna Avenue,
Sidcup,
Kent DA14 6LT.

Correspondence to Miss L L Watt, FRCS.

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Notes

Names omitted

SIR, We apologise for having omitted in error the names of two coworkers, Gisèle Soubrane, MD, and Peirre Dhermy, MD, in the paper entitled 'Adenocarcinoma of retinal pigment epithelium' *Br J Ophthalmol* 1987; **71**: 516–20. The correct list of authors should have been: C Ramahefasolo, G. Soubrane, P Dhermy, V Godel, L Regenbogen, G Coscas.

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