Gaze evoked amaurosis in dysthyroid orbitopathy

EDITOR,—Gaze evoked amaurosis is an uncommon symptom usually associated with intracranial masses such as cavernous haemangioma or optic nerve sheath meningioma.1 Patients describe transient loss of vision in eccentric positions of gaze with full recovery of vision on returning to the primary position. The cases previously published have all been unilateral. We describe a case of bilateral gaze evoked amaurosis in a patient with dysthyroid orbitopathy.

CASE REPORT
A 62 year old smoker gave a 12 month history of transient bilateral loss of vision on upgaze associated with supraorbital discomfort. His hobby was flying radio controlled model aeroplanes but after crashing two of these during amaurotic episodes he presented to an ophthalmologist. Two years previously he had required admission for cardiac failure associated with elevation of the eyes. The only orbital pathology was only moderate and persisted after treatment with iodine-131 and carbimazole his thyroid status had been stabilised.

Ophthalmic examination revealed symmetrical axial proptosis (26 mm), restriction of upgaze, and lid signs consistent with dysthyroid orbitopathy. In primary position the corrected distance acuities were 6/6 in both eyes with 17/17 Ishihara colour plates seen and full visual fields. On upgaze the distance acuities were reduced to less than 6/60 with none of the Ishihara colour plates seen. The intraocular pressures (IOP) increased from 20 mm Hg (both eyes) in primary position to 30 mm Hg (right eye) and 31 mm Hg (left eye) in elevation. Pupil examination using infrared pupillography showed 0.20 mm dilation to 30 mm Hg (right eye) and 31 mm Hg (left eye) in elevation. Pupil examination using the primary position. The disc and retina both in primary position. The only orbital pathology was only moderate and persisted after treatment with iodine-131 and carbimazole his thyroid status had been stabilised.

Figure 1 Pattern VEPs from the left eye (left) and from the right eye (right) before treatment (A) and after treatment (B). In each case the upper trace was recorded with the eyes in primary position, the lower trace with the eyes in upgaze.

Figure 2 Computed tomograph of orbits (coronal view).

COMMENT
The visual loss in this patient was transient, reversible, and related to the position of the globe in the orbit. The only orbital pathology found on imaging was enlargement of the extraocular muscles related to his dysthyroid orbitopathy. The mechanism of this visual failure remains intriguing. Its rapid onset and reversibility suggest vascular compromise but fluorescein angiography showed normal disc and retinal perfusion on upgaze. Vascular compromise of the retrobulbar optic nerve cannot be ruled out. Of interest, in the rat model retrobulbar ischaemia produces a depolarising conduction block which takes minutes rather than seconds to develop and which would generate photopsia before the visual failure.2 In previous reports, raised IOP has been invoked as the cause of the visual loss.3 However, in our patient the rise in IOP was only moderate and persisted after steroid treatment whereas the vision improved. A third possibility is that compression of the optic nerve by enlarged extraocular muscles or stretching of the nerve as a result of dural tethering produced a mechanical conduction block in upgaze. This has been described in peripheral nerves4 but it classically takes several days for full recovery of function; whether optic nerve axons behave similarly is not known. Gaze evoked amaurosis has not been previously described in thyroid eye disease but was noticed by this patient because it interfered with his hobby of flying radio controlled model aeroplanes.

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Epiphora due to Kaposi’s sarcoma of the nasolacrimal duct

EDITOR,—Excessive watering of the eye may be due to lacrimation, mechanical obstruction of the tear drainage, or lacrimal pump failure. We report a case of obstructive epiphora due to Kaposi’s sarcoma of the nasolacrimal duct.

CASE REPORT
A 34 year old man who was recently diagnosed as HIV seropositive was referred to the eye department complaining of watering of his left eye. At the time of diagnosis Kaposi’s sarcoma lesions were noted on his face, left lower lid, and scalp and the patient was commenced on triple antiretroviral therapy (zidovudine, lamivudine, saquinavir). On examination a Kaposi’s sarcoma lesion of the left lower lid with swelling over the lacrimal sac was noted (Fig 1). The patient had a left sac washout, which confirmed left nasolacrimal duct obstruction. At the time of sac washout a blood stained nasal discharge was noted. Examination of the left nostril showed a raised lesion involving the nasal septum, inferior turbinates, and the nasolacrimal duct. Biopsy under local anaesthesia was performed and examination of the specimen confirmed the diagnosis of Kaposi’s sarcoma (Fig 2). The patient was commenced on liposomal daunorubicin and his symptoms of epiphora resolved completely.

COMMENT
Epidemiological evidence suggests that Kaposi’s sarcoma is caused by a transmissible
Figure 2: Histological section shows a cellular spindle cell lesion forming vascular channels within the uvea (Kaposi’s sarcoma).

Figure 1: Vitreous haze with a lobulated white mass in the inferior vitreous.

venous catheter for 1 month. Mitral valvuloplasty had been performed in October 1997. His visual acuity was right eye 20/20 and left eye 20/100. The left eye had anterior uveitis and treated with oral prednisolone. As the inflammation had not resolved, a transitory intravitreal flucnazole injection was performed on 14 April 1998. The next day he had severe pain in his left eye and headache. Left visual acuity reduced to light perception. Intraocular pressure was 42 mm Hg, and marked inflammation with hypopyon was observed. A bacterial endophthalmitis was suspected, he underwent the second vitrectomy on 16 April 1998 with intravitreal imipenem irrigation. Vitreous cultures grew O. anthropi. The isolate was sensitive to cefmetazole, cefbuperazone, imipenem, minocycline, levofloxacin, gentamicin, tobramycin, and amikacin, and resistant to other conservative or surgical medications.

COMMENT
The natural habitat of O. anthropi has not been established. It is commonly found in environmental and hospital water sources. It has been isolated from clinical specimens, including blood, urine, faeces, and sputum. Most cases of O. anthropi sepsis were reported to relate to indwelling catheters. In a case series, 10 of 11 patients had intravenous catheters or other permanent medical devices. As for the infectious routes, there are two possibilities in our case. One is contamination during mitral valvuloplasty. Indeed, a lobulated white mass in the vitreous occurred within 3 weeks after placement of a central venous catheter. Another possibility is that the patient had an intravenous access or other permanent medical devices.

Ochrobactrum anthopri endophthalmitis after vitreous surgery

EDITOR—Ochrobactrum anthopri is a non-fermentative, motile, strictly aerobic, oxidase positive Gram negative bacillus. In 1980, the first case of human infection with O. anthopri was described. Since then, there have been some reports and this bacillus has been considered as a possible cause of opportunistic infection. There are only two reports of O. anthopri endophthalmitis, one was metastatic endophthalmitis in a patient with a central venous catheter, and the other was after cataract surgery. We describe a case of unilateral endophthalmitis caused by O. anthopri, which was diagnosed after two vitreous surgery procedures.

CASE REPORT
A 64 year old man complained of visual loss in his left eye in January 1998. He was diagnosed with uveitis and treated with oral prednisolone, topical betamethasone and atropine, and subconjunctival injection of dexamethasona. As the inflammation had not resolved, he was transferred to our institution. He had a medical history of bacterial endocarditis caused by Staphylococcus haemolyticus in April 1997 and underwent placement of a central venous catheter for 1 month. Mitral valvuloplasty had been performed in October 1997. His visual acuity was right eye 20/20 and left eye 20/100. The left eye had anterior uveitis and treated with oral prednisolone. As the inflammation had not resolved, a transitory intravitreal flucnazole injection was performed on 14 April 1998. The next day he had severe pain in his left eye and headache. Left visual acuity reduced to light perception. Intraocular pressure was 42 mm Hg, and marked inflammation with hypopyon was observed. A bacterial endophthalmitis was suspected, he underwent the second vitrectomy on 16 April 1998 with intravitreal imipenem irrigation. Vitreous cultures grew O. anthopri. The isolate was sensitive to cefmetazole, cefbuperazone, imipenem, minocycline, levofloxacin, gentamicin, tobramycin, and amikacin, and resistant to other conservative or surgical medications.

COMMENT
The natural habitat of O. anthopri has not yet been established. It is commonly found in environmental and hospital water sources. This organism has been isolated from clinical specimens, including blood, urine, faeces, and sputum. Most cases of O. anthopri sepsis were reported to relate to indwelling catheters. In a case series, 10 of 11 patients had intravenous catheters or other permanent medical devices.

As for the infectious routes, there are two possibilities in our case. One is contamination during mitral valvuloplasty. Indeed, a lobulated white mass in the vitreous occurred within 3 weeks after placement of a central venous catheter. Another possibility is that the patient had an intravenous access or other permanent medical devices.

REFERENCES

Scleral perforation following trans-scleral cyclodiode

EDITOR—In recent years, the diode laser utilising 810 nm wavelength has emerged as an increasingly popular and effective tool for treating severe cases of glaucoma which are resistant to other conservative or surgical therapeutic options. The desired effect (as with other lasers used in this field) is thermal heating and coagulation necrosis of the ciliary epithelium in a trans-scleral cyclodiope photocoagulation). However, the laser scleral transmission is increased by the contact method (compared with the non-contact method), allowing for less total energy application while obtaining the same desired effect. The side effects of trans-scleral cyclodiode laser range from common ones such as mild irritis to rare ones including phthisis bulbi. To the best of our knowledge, there has been only one reported case of scleral perforation due to trans-scleral cyclode ciclophotocoagulation. This was following contact delivery of the laser utilising the original quartz G-probe (diameter 600 µm) and settings of 2 W for 2 seconds per application. The patient in question had scleral thinning following previous cataract surgery. It was thought that the sharp edge of the probe had cut conjunctival vessels causing bleeding and contamination of the probe head. Thus adherent debris was then carbonised allowing the laser tip temperature to rise to 300°C, sufficient to cause scleral perforation. This case report led to the redesigning of the laser probe tip in order to protect the vascular structures from its sharp edges.
scleral thinning was the common risk factor. Parma et al. looked at the effect of cyclodiode therapy on cadaver eyes. They showed that approximately 40% less energy is needed to achieve ciliary photocoagulation in thin sclera (that is, half to a third full thickness) compared with normal thickness sclera.

Our patient was being treated as part of a standard protocol in our unit as part of a prospective trial of cyclodiode therapy. We had previously treated other eyes with thin sclera without incident at the energy levels utilised for the case described above although none has had such extensive thinning as this case. Similarly thinned areas in the same eye had been treated before the event which occurred on the final scheduled application. No conjunctival or corneal perforations had been noted before the perforation and the probe tip had not been inspected between applications. It is difficult to know whether the perforation in our case was due to mechanical pressure, ciliary decompensation, chemical irritation, or a combination of these. We now warn against treating areas of an eye with severe scleral thinning as there are no known “correction factors” that can be utilised at present. If treatment is absolutely necessary, a lower laser power setting should be used (we would suggest 50%) and minimal pressure applied with the G-probe. Furthermore, care should be taken to ensure that the probe tip is clean before each application in such eyes in order to prevent carbonisation of debris.

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COMMENT
Botulinum injection is regarded as a procedure with a low incidence of morbidity. Post injection ptosis and diplopia are transient complications. No eye has been reported to have lost vision as a result of botulinum injection. Accidental perforation of the globe is an acknowledged complication of peribulbar and retrobulbar anaesthesia and strabismus surgery. High myopia is a strong risk factor for globe perforation in peribulbar anaesthesia. One case shows that globe perforation can occur with botulinum injection as with any peribulbar surgical procedure. In our patient the injection was performed by an experienced surgeon and the procedure

Figure 1 Disc and nasal retina of the right eye showing retinal haemorrhage following botulinum injection.
was performed in a standard fashion using EMG control. Before injection there was good muscle signal. It has been shown that EMG signals may be recorded in most cases once the needle contacted the conjunctiva. Using a monopolar electrode needle, the reference electrode is located centimetres away from the active electrode and the needle can record signals that are closer to the needle electrode than the reference electrode even if they are several millimetres away from the needle electrode. In our patient it is likely that the tip of the needle was in the vitreous cavity at some stage during the procedure and an EMG response was present at that time. The most common potentially vision impairing complication of globe perforation is retinal detachment. Retinal detachment has been reported in globe perforations associated with peripheral anaesthesia and after strabismus surgery. The theoretical risk of causing a globe perforation is greater with botulinum injection into an extraocular muscle than it is with peripheral injection. The needle enters the muscle just behind the insertion and the sclera at its thinnest at this point (0.3 mm). Any movement of the eye by the patient, with the needle in this position may result in an inadvertent perforation of the globe, especially in patients who are likely to have thin sclera, such as high myopes. Demonstration of increased signal by movement of the eye into the field of action of the muscle to be injected should probably be avoided for this reason.

Management of patients with scleral perforations is controversial. Some authors recommend that they should be treated with indirect diode laser or transscляр cryotherapy regardless of the depth of perforation, to reduce the incidence of retinal detachment. However, animal experiments have found a higher incidence of retinal detachment following heavy cryothapy, and suggest cryotherapy should be used only if there is vitreous haemorrhage or the patient has a predisposing risk factor for retinal detachment. Our patient was a high myope and she had a small retinal and vitreous haemorrhage which increased her risk of developing a detachment. Globe perforation, although rare, is a complication that can occur with botulinum injection into an extraocular muscle and surgeons doing the procedure and their patients should be aware of this. The risk is higher in myopic eyes, as the equator of the globe is more posterior than usual, and the sclera thinner.

Botulinum toxin injection to an extraocular muscle should be approached with extreme caution in highly myopic eyes, and all movement of the eye should be avoided during the procedure.

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and fungi were negative. These findings suggest that apoptosis occurs in choroidal granuloma and plays a regulatory role in limiting ocular inflammation.4

Upon diagnosis of ocular sarcoidosis and treatment with azathioprine, the patient's vision improved to 20/40 in her right eye and has remained stable for 4 years.

COMMENT

This case illustrates several points: ocular sarcoidosis can occur in the absence of pulmonary signs or symptoms and causes a plethora of ocular findings; the disease eludes accurate diagnosis5—in this case, a chorioretinal biopsy suggested some parallels in ocular and systemic corticosteroids and cyclosporine, but responded to azathioprine. Azathioprine has been used successfully to treat sporine, but responded to azathioprine. Azathioprine can occur in the absence of pulmonary diseases and ophthalmic manifestations, special caution is warranted. Dysregulative phenomena might well alter various vascular beds in a comparable fashion, but local factors will always influence regional blood flow. Consequently, further studies will have to confirm these results and unravel the exact basis for such a relation.

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Relation between blood flow velocities in the ophthalmic artery and in nailfold capillaries

Editor—Studies on visual field and peripheral circulation had led to the hypothesis that the eye might be involved in the vasospastic syndrome, a potential contributor to glauco-

matous optic neuropathy.2 In some patients with peripheral vasospasms, visual field defects worsened after cold provocation, and, often, both peripheral vasospasms and visual field defects improved after calcium channel blocker treatment.1 Because these and newer observations3 suggest some parallels in ocular and digital blood flow regulation, the relation between ophthalmic artery and nailfold capillary blood flow velocities was evaluated.

Fifty patients with primary open angle glaucoma with a mean age of 67 (SD 15) years were examined. Excluded were patients with previous filtering surgery or systemic and cardiovascular diseases. Blood flow velocity in nailfold capillaries was assessed in one randomly chosen finger of the right hand (totally arbitrary choice) by means of nailfold capillaroscopy.1 The velocities measured in each visible vessel were averaged. Blood flow velocity in the ophthalmic arteries was assessed by means of colour Doppler imaging (CDI). The ophthalmic artery was traced nasal to the optic nerve, 10–15 mm posterior to the globe. CDI measurements of the right eye of each patient were considered for further analysis (same side as nailfold capillaroscopy).1 The velocities measured in each visible vessel were averaged. Blood flow velocity in the ophthalmic arteries was assessed by means of colour Doppler imaging (CDI).

The mean blood cell velocity in nailfold capillaries was 0.45 (SD 0.32) mm/s. The mean PSV was 37.11 (7.33) cm/s. The mean EDV was 7.7 (3.06) cm/s. The average MV was 15.30 (7.80) cm/s. The mean RI was 0.79 (0.07). EDV (R=0.31; p=0.034) and MV (R=0.38; p=0.007) correlated with blood cell velocity in nailfold capillaries. PSV (R=0.14; p=0.35) and RI (−0.25; p=0.09) did not correlate with nailfold capillary blood velocity. Systemic blood pressure (mean systolic pressure 120.5 (11.4) mm Hg; mean diastolic blood pressure 73.4 (13.6) mm Hg) did not vary significantly during blood flow assessments.

COMMENT

The results suggest a relation between nailfold capillary blood cell velocity and blood flow velocity in the ophthalmic artery. Although the ophthalmic artery is the only routinely assessed retorubular vessel in CDI which does not enter the eye, its contribution to choroidal blood flow is important.4 Because vasospastic dysregulation seems to be much more common in the ciliary circulation than in the retinal vasculature,4 it appeared reasonable to evaluate this vessel, especially because this vessel is assessed much more reliably than ciliary vessels during CDI. RI and PSV did not correlate with nailfold capillary blood cell velocity. However, RI is not a measure of velocity. PSV represents a unique event in arterial blood flow, and, possibly, capillary blood flow fluctuations may not be related directly to very brief moments during the cardiac cycle.4 Although the measurement of EDV is less reproducible compared with PSV and RI, a fact which is expected to alter a potential correlation, nailfold capillary blood cell velocity correlated with EDV and MV in the ophthalmic artery. The relatively constant blood flow during the diastole might predict more closely blood cell velocity in the capillary bed. Mean velocity is not related to unique moments in the cardiac cycle and seems to reflect even more closely blood cell velocity in capillary vessels. Although the present study suggests some common alternations in various vascular beds in glaucoma patients, special caution is warranted. Dysregulative phenomena might well alter various vascular beds in a comparable fashion, but local factors will always influence regional blood flow. Consequently, further studies will have to confirm these results and unravel the exact basis for such a relation.