under general or peribulbar anesthesia. However, up to four injections were required to control the disease, and recurrence occurred in one case during a mean follow-up time of 17.5 months. Three patients, including the one with recurrence, continued one systemic antitoxoplasmic medication during follow-up.

IVTA has been efficient in rapidly decreasing inflammation in acute sight-threatening noninfectious uveitis.[3,4] In our study, a single injection of IVTA was used as an adjunct in the treatment of severe ocular toxoplasmosis, with rapid and successful control of the intraocular inflammation in both cases and no signs of recurrence during an 18-month follow-up period. Trimethoprim-sulphamethoxazole three times a week was used as a prophylactic treatment against the parasite in the period in which some triamcinolone acetonide residue might still be present in the vitreous cavity, potentially suppressing the immune response.

Conclusion

We suggest that IVTA should be considered as a potential benefit to patients with severe ocular toxoplasmosis or in which the use of systemic corticosteroids is contraindicated and should be investigated further. To our knowledge, this is the first report of IVTA in the treatment of infectious uveitis.[4]

References

1 Nussenblatt RB. Ocular toxoplasmosis. In: Nussenblatt RB, Whitcup SM (eds). *Uveitis: Fundamentals and Clinical Practice*, 3rd edn. Mosby: Philadelphia, 2004, pp 214–234.
2 Kishore K, Conway MD, Peyman GA. Intravitreal clindamycin and dexamethasone for toxoplasmic retinochoroiditis. *Ophthal Surg Lasers* 2001; **32**: 183–192.
3 Benitez Del Castillo Sanchez JM, Garcia Sanchez J. Intravitreal injection of triamcinolone acetonide in non infectious uveitis. *Arch Soc Esp Oftalmol* 2001; **76**: 661–664.
4 Jonas JB, Kreissig I, Degenring R. Intravitreal triamcinolone acetonide for treatment of intraocular proliferative, exudative and neovascular diseases. *Prog Retin Eye Res* 2005; **24**: 587–611.

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Sir,

The natural history of Vigabatrin-associated visual field defects in patients electing to continue their medication

We read with interest the paper by Best and Acheson[1] regarding the natural history of Vigabatrin-associated visual field defects in patients electing to continue their medication. The first cases of concentric bilateral visual field defects in patients taking Vigabatrin were reported in the late 1990s and since then numerous cases have been reported worldwide. The association has grown stronger and is now a well-accepted adverse drug effect of Vigabatrin therapy.[2–4]

In 2001, the Royal College of Ophthalmologists (RCO) published screening guidelines entitled 'The ocular side-effects of Vigabatrin, information and guidelines for screening'.[5] For adults, they recommend pretreatment baseline visual field using either static suprathreshold 2 or 3 zone perimetry (Humphrey 120 point or Octopus 07) to at least 45 radius eccentricity, or Goldman kinetic perimetry (IIIe and I4e or I2e stimuli, as appropriate). All patients should have 6 monthly follow-up assessments for the first 3 years of treatment, which can then be extended to annually in patients in whom no visual field defects are found.

We used a questionnaire survey in the South-West of England and Wales to investigate views of consultant ophthalmologists on the RCO guidelines and to review current clinical practice.

Out of 97 consultants, 54 contacted responded to the questionnaire (response rate 56%). Consultant ophthalmologists were asked about their experience with Vigabatrin-related visual field screening in the year 2002–2003. More than a third (35%) of those surveyed were unaware that the RCO had published guidelines relating to Vigabatrin-associated visual field defects. Only 15% had received new referrals for baseline visual field documentation prior to patients starting on Vigabatrin and 41% of respondents had performed or arranged visual field screening for patients already on Vigabatrin. With respect to screening intervals, 51% thought that the screening interval should be 12 months or longer, 45% agreed with a screening interval of 6 months and 4% felt

that the interval should be 3 months for the first 3 years of treatment.

A lack of communication between the neurologists and ophthalmologists was stated, by 75% of respondents, as the most common reason for noncompliance with the screening guidelines. This probably relates to the failure of neurologists to refer patients for screening prior to commencing them on Vigabatrin and also to the lack of involvement of the ophthalmologist in the follow-up of these patients.

Surprisingly, a small proportion of respondents cited that an increase in workload as a reason for noncompliance with the RCO guidelines, in spite of the relatively small number of patients requiring screening. Those respondents who were involved in screening patients were performing less than five visual fields a year.

There is a disparity between current clinical practice and the RCO guidelines. None of the respondents had carried out an audit of their clinical practice regarding screening of patients on Vigabatrin. While patient numbers may be few, continued audit and clinical data collection should be encouraged in accordance with RCO guidelines. Although the exact pathogenesis of these field defects is not known, the need to screen patients on Vigabatrin is well established. Screening is important if the field defects are caused by an idiosyncratic reaction to the medication, particularly as only a certain group of patients will be affected.

It is disappointing that despite the RCO publishing guidelines, more than a third of the consultant ophthalmologists responding to the survey were unaware of their existence. Our survey of clinical practice indicates that there is only a moderate agreement with the current guidelines. A joint guideline issued by ophthalmologists and neurologists regarding screening for Vigabatrin-associated field defects may help bridge the gap between the specialties and achieve wider agreement and compliance.

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References

1 Best JL, Acheson JF. The natural history of Vigabatrin associated visual field defects in patients electing to continue their medication. *Eye* 2005; **19**(1): 41–44.
2 Eke T, Talbot JF, Lawden MC. Severe persistent visual field constriction associated with Vigabatrin. *BMJ* 1997; **314**: 180–181.
3 Appleton RE. Guideline for prescribing Vigabatrin. *BMJ* 1998; **317**: 1322.
4 Vigabatrin Paediatric Advisory Group. Guideline for prescribing Vigabatrin in children has been revised. *BMJ* 2000; **320**: 1404–1405.
5 http://www.rcophth.ac.uk/members/members-documents/Vigabatrin.pdf.

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Sir,

Reply to Kumar and Jivan—Vigabatrin-related visual field defects

The results of the postal survey by Kumar and Jivan on screening for Vigabatrin-related visual field defects has yielded some interesting data reflecting the application of guidelines in the real world. Busy clinicians are naturally averse to extra service load and there are always communication issues between hospital specialists. However, it is worth pointing out that Vigabatrin has now largely been replaced by newer agents without this side effect for the control of refractory epilepsy, so the number of epileptic patients with visual impairment from this phenomenom is limited. Although the field loss is not treatable, recognition and appropriate visual impairment registration is still beneficial to the patient.

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