are the first to demonstrate such a prognostic significance for abnormalities found on electro-physiologic testing. In so doing they help ophthalmologists faced with ever mounting numbers of ocular hypertensive patients to decide which patients are at risk of developing glaucoma and therefore worthy of prophylactic treatment. However, before adopting their test strategy as a routine it would be helpful if they could amplify the precautions they took in this prospective study to truly identify those ocular hypertensive patients with an abnormal VEP who progress and develop a visual field defect.

In their article they describe the results of twice testing 49 ocular hypertensive patients with VEP and perimeter, first between 1984 and early 1989, and secondly in late 1989. They noted that 7/24 (29%) reverted to normal. It would be helpful to know what was the test-retest variability in their laboratory.

Secondly, these patients had only two visual field tests analysed. Assuming that they had had previous tests and were ‘experienced’ patients, there are still many pitfalls found on drawing conclusions from analysing two visual fields, especially in glaucoma patients, for the visual field can progress from ‘normal’ to ‘abnormal’ (again) and ‘back again’ in consecutive fields. The authors should let us know the precautions taken to rule out such false positive results occurring in their patients.

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Reply

Sir,—We would like to thank Mr Hitchings for making a number of important observations. With regard to the false positive rate of the VEP, as we explain in discussion, intertest comparisons enable us to detect deterioration of VEP parameters, and thus abnormality, even when they still fall within the normal range. So, while it is true that the VEP of seven of our ocular hypertensive (OH) patients were reported as ‘normal’ on repeat testing, we are able to qualify these observations. In four of the seven cases such comparisons revealed an increase in latency in the fellow eye, ‘normalising’ a previously interocular difference suggesting the presence of ‘atrophy.’ Of the other three, one was found to have consistently normal intraocular pressures during subsequent follow-up and in retrospect we feel should have been eliminated from the trial, and one who became ‘normal’ when he changed age control range (59 to 60 yr). This leaves one false positive for whom we have no explanation. Notwithstanding this, the true false positive rate of 3/24 (12.5%) is similar to that observed in the control group (2/26 or 7-7%) and within the degree of statistical precision one would expect from a trial of this size. Test-retest variability was investigated in four control subjects who were subjected to daily VEPs for three days. Mean intertest latency for this group was found to be 1.8 ms. Intertest interocular variability is insignificant, as it is less than the measurement accuracy of VEPs.

In relation to the second question, we acknowledge the current view that more than 15% of patients,24 from the control group, were truly normal on testing. These patients,24 however, were ‘pristine’ fields and thus would have been difficult to rule out had they been included in the group. This group demonstrates such a prognostic value of VEPs being both a measure of uncertainty.

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OBITUARY

E G ATKINSON, MBChB, MSc, MRCP

Edmund Atkinson had just resumed his clinical training in ophthalmology, after three years in research, when he died in a paragliding accident in France in June 1990. He had been an invaluable and enthusiastic member of the retinal vasculitis research group at St Thomas’s Hospital, and presented his work both at the OSUK and ARVO. He qualified in medicine at Bristol University in 1982, and obtained his MRCP after working in local hospitals. He then moved to London to start his career in ophthalmology at St Bartholomew’s Hospital. As he wished to develop his research interests as soon as possible he moved to St Thomas’s Hospital, where in 1986 he became an MRC Training Fellow. He developed two new animal models of uveitis, and extended earlier work on its pharmacological modulation. Later he worked with the pharmacology department to develop FITC-dextran angiography as a method for investigating permeability changes in human uveitis. At the time of his death, he was continuing his studies on cloning vitreous T-cells from uveitis patients. He was always a potent source of ideas, support, and enthusiasm and always worthy of talents to the full. It was tragic that his latest enthusiasm should also be the cause of his early death.

NOTES

The 4th Annual Meeting of the Retinological Society (Retinologische Gesellschaft)

The 4th Annual Meeting of the Retinological Society (Retinologische Gesellschaft) will be held on 1 to 2 November 1991, at the Kupferbau-Building of the University of Tubingen, Gmelinistraße 8, W-7400 Tubingen, Germany. Details from: Professor J Kreissig MD, Ophthalmology III, Schleichstrasse 12, W-7400 Tubingen, Germany. (Tel: 07071/293741, Fax: 07071-293730.)

Optical engineering

A meeting on Ophthalmic Technologies II (Part 2 of Biomedical Optics) will be held by the International Society for Optical Engineering on 19-24 January 1992. It will be at the Los Angeles Marriott Hotel, Los Angeles, California, USA. Details from the society at PO Box 10, Bellingham, Washington 98227-0010, USA.

Golden Jubilee Conference of All India Ophthalmological Society

The Golden Jubilee Conference of All India Ophthalmological Society will be held in New Delhi from 2 to 6 February 1992, at the Ashok/Samarahot Hotel. Further details: Dr R B Jain, organising secretary, Mohan Eye Institute, 11-B Ganga Ram Hospital Marg, New Delhi-110 060, India. (Tel: 587655, 5728969. Fax: 91-011-5728969).