

## LETTERS TO THE EDITOR

### Amaurosis fugax in young adults

EDITOR,—We read with interest the article by O'Sullivan *et al.*<sup>1</sup> The nine presented patients went through extensive investigations, including echocardiograms in all cases. Yet, no evidence of an embolic or atheromatous aetiology was found. Unfortunately, the authors failed to mention whether the echocardiography was transthoracic or transoesophageal.

We had experience with a 29-year-old woman, with recurrent retinal artery branch occlusion in both eyes. A thorough investigation, including transthoracic echocardiography, failed to reveal any pathology. Transoesophageal echocardiography, however, disclosed mitral valve prolapse with a thickened mitral valve and numerous vegetations, which were probably the origin for the embolic retinal events.

It has been found that transoesophageal echocardiography leads to a significant improvement in the identification of potential cardiac sources for arterial embolism associated with cerebral ischaemic events when compared with results using transthoracic echocardiography.<sup>2</sup> Transoesophageal echocardiography has also been shown to be better in detecting mitral valve prolapse and other cardiac abnormalities in young patients.<sup>3</sup> Greven *et al.*<sup>4</sup> and Wiznia *et al.*<sup>5</sup> have reported that transoesophageal echocardiography was more helpful than transthoracic echocardiography in disclosing the pathology that led to retinal vascular occlusion.

It seems to us that in cases of 'amaurosis fugax in young adults', when no pathological findings can be found, transoesophageal echocardiography is warranted.

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### Reply

EDITOR,—We thank Dr Sadeh and colleagues for their comments. They are correct in assuming that transthoracic echocardiographic

studies were performed in the patients were reported. However, at the time our data were collected, transthoracic echocardiography was the standard non-invasive method widely available in general hospital practice and therefore the most appropriate modality for the investigation of our patients. It was carried out by experienced operators who obtained high quality images of the valvular structures in all cases. If inadequate images had been obtained, consideration would have been given to the more invasive investigation.

We accept that there is an increasing body of evidence which suggests that if a paradoxical source of embolism via the atrial septum is suspected and the valvular structures are normal on transthoracic echocardiography, then transoesophageal studies are indicated. As further experience is gained with this investigative technique, it will become more widely available and possibly applicable in cases such as ours.

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### Glaucoma, ocular hypertension, cataract, and glucose tolerance

EDITOR,—The excellent paper by Harding *et al.*<sup>1</sup> provides further evidence of the association between diabetes and cataract and confirms the clinical association of glaucoma with cataract.<sup>2</sup> Another recognised and relevant association links diabetes mellitus and glaucoma in both directions — that is, diabetics are more likely to have glaucoma and glaucoma patients are more likely to be diabetic.<sup>3,4</sup>

In the Department of Clinical Pharmacology, St Thomas's Hospital, London, I studied glucose tolerance in 21 ocular hypertensives (as defined by Hollows and Graham<sup>5</sup>) and 21 age and sex matched control subjects. Subjects with known diabetes were excluded. Each group contained 14 women and seven men. Ages (years, mean (SD)) were 63.8 (7.3) for ocular hypertensives and 63.1 (8.31) for controls. Testing was performed according to WHO recommendations.<sup>6</sup> After a 12 hour overnight fast a venous blood sample was taken and subjects drank 82.5 g hydrous glucose dissolved in 300 ml tap water in less than 5 minutes. A further venous sample was taken 2 hours later. One patient in each group was excluded from final analysis for technical reasons.

Patients were classified as normal glucose tolerance (fasting glucose <6.7 mmol/l, 2 hour glucose <6.7 mmol/l), impaired glucose tolerance (IGT) (fasting glucose <6.7 mmol/l, 2 hour glucose >6.7 mmol/l but <10.0 mmol/l), or diabetes mellitus (DM) (fasting glucose ≥6.7 mmol/l or 2 hour glucose ≥10.0 mmol/l). Results (see Table) were analysed by  $\chi^2$  using Yates' correction. The  $\chi^2$  value was 7.152 and  $p < 0.05$ . This study demonstrates a significantly higher incidence of abnormal glucose tolerance (IGT or DM) in ocular hypertensives than age and sex matched controls.

These findings and the evidence from Hard-

	Normal	Impaired glucose tolerance	Diabetes mellitus
Ocular hypertension (n=20)	12	6	2
Controls (n=20)	19	1	0

ing and coauthors imply a triangular relationship between abnormal glucose tolerance (IGT or DM), raised intraocular pressure (ocular hypertension or primary open angle glaucoma), and cataract. The presence of any one of these disorders (and perhaps the 'strength' of that presence) is influential in determining the presence of either of the other disorders in the same individual. The clinical relevance of these findings in the ophthalmic clinic is simple; patients with raised intraocular pressure or cataract must have their urine tested to screen for diabetes mellitus, with additional investigation where glucose is detected in the urine.

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### Reply

EDITOR,—I am grateful to Dr Brazier for his interest in our paper. His study is small but appears to show a clear association between abnormal glucose tolerance and ocular hypertension. Thus our work and his, combined with earlier reports, associate diabetes (and abnormal glucose tolerance), cataract, and glaucoma (and ocular hypertension). The extension from diabetes to abnormal glucose tolerance is important in that estimates of the proportion of an eye disease, for example, cataract, due to diabetes underestimate the role of glucose per se. At least three of the four major eye diseases are associated with diabetes so that a large proportion of total visual impairment in Western countries must be caused by one small molecule, glucose, and its metabolites.

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### Pleomorphic adenoma of the lacrimal gland

EDITOR,—I read with interest the article by Rose and Wright<sup>1</sup> regarding their recommendations for diagnosis and treatment of pleomorphic adenomas of the lacrimal gland. The authors emphasise that biopsy of these lesions is often associated with recurrence and spread of the tumour, and they recommend against a biopsy in any case suspected of being a pleomorphic adenoma. Although I agree with them that a biopsy of the lesion, followed by termination of surgery with definitive surgery of the tumour at a later date is likely to result in recurrence of the tumour, it is unclear to me whether or not biopsy of the lesion for frozen section diagnosis, followed by definitive treatment of the lesion at the same time has the same probability of resulting in a recurrent tumour. I