Amaurosis fugax in young adults

EDITOR,—We read with interest the article by O'Sullivan et al.1 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 significant abnormality. 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.2 Transoesophageal echocardiography has also been shown to be better in detecting mitral valve prolapse and other cardiac abnormalities.3 Green et al.4 and Wiznia et al.5 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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Glucoma, ocular hypertension, cataract, and glucose tolerance

EDITOR,—The excellent paper by Harding et al.6 provides further evidence of the association between diabetes and cataract and confirms the clinical association of glaucoma with cataract.6 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.7

In the Department of Clinical Pharmacology, St Thomas's Hospital, London, I studied glucose tolerance in 21 ocular hypertensives (as defined by Hollows and Graham8) 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.9 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), impaired glucose tolerance (IGT) (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 >7.0 mmol/l or 2 hour glucose >10.0 mmol/l). Results (see Table) were analysed by y' using Yates' correction. The y' 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 Harding 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 their presence) is influencing 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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Pleomorphic adenoma of the lacrimal gland

EDITOR,—I read with interest the article by Rose and Wright1 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
Letters to the editor

would appreciate it if the authors could address this issue.

NEIL R MILLER
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Reply

EDITOR.—We thank Dr Miller for his interest and commentary about our paper on pleomorphic adenomas of the lacrimal gland. We are unable to answer the question of whether the extent of surgical excision determines the presence of lid changes. Our study was focused on investigating the presence of surgical pathology in the vicinity of the tumour. We have no data to suggest that the extent of surgery is linked to the presence of lid changes. The presence of lid changes may be linked to factors such as the degree and duration of the symptoms, the type of tumour (e.g., pleomorphic adenoma, acinic cell carcinoma), and the patient's age and gender. We agree that further research is needed to determine the factors that contribute to the development of lid changes.

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