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Sir,

We thank Murray and her colleagues for their interest in our paper.[1] We are grateful to them for drawing attention to the practicalities of preparation where sterile manufacturing pharmacy units are not available. We undertook our study because we had already adopted a clinical treatment regime similar to that described by Murray *et al.* Also, just as they draw attention to potential retinal toxicity from aminoglycosides, we replaced vancomycin with teicoplanin because of the, admittedly theoretical, risk of damage from the very low pH inherent in maintaining even low concentrations of vancomycin in solution. The research that is now needed is a formal multicentre comparative trial to determine which intravitreal antibiotic(s) provide optimum first-line cover for endophthalmitis.

Reference

1. Briggs MC, McDonald P, Bourke R, Smith G, McGalliard JN, Wong D. Intravitreal penetration of teicoplanin. Eye 1998;12:252–5.

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Sir,

I read with interest the editorial by Griffiths *et al.* on empty sella and its possible relationship with visual field defects.[1] I and my colleagues have shown, on sella CT scans obtained with intrathecal injection of radio-opaque material in patients with visual field defect and primary empty sella, that the chiasm may flatten without herniation.[2] Therefore, although rare, empty sella may indeed cause visual field defects, without herniation, due to the filling effect of cerebrospinal fluid below the chiasm, which lifts it up. It is our belief that the chiasm is put under pressure by the liquid, from both below and above, causing ischaemic changes.

Although there is general acceptance that MR imaging for microadenomas is superior to CT scans, we believe that, in order to detect the location of the chiasm, it is not essential to use MR imaging.

References

1. Griffiths PG, Dayan M, Coulthard A. Primary empty sella: cause of visual failure or chance association? Eye 1998;12:905–6.
2. Kirkali P, Kansu T, Erzen C, Cila A. A radiological insight into primary empty sella syndrome with visual dysfunction. Neuro-ophthalmology 1989;9:259–65.

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Sir,

We thank Professor Aydin for his interest in our article and apologise for our oversight in not referring to his study. Although it is possible to image the anterior visual pathway with CT scans and radio-opaque contrast material, it is an invasive and time-consuming test which can not now be justified in the investigation of patients with visual failure when safer, non-invasive options are available.

Whilst the mechanism of visual loss in primary empty sella syndrome as proposed in Professor Aydin's paper is entirely plausible, the causal relationship between primary empty sella and visual loss was not established by that study, and has yet to be established. The proposed mechanism is therefore at best speculative.

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Sir,

We read with interest 'Glaucoma screening by optometrists: positive predictive value of visual field testing' by Newman *et al.*[1] There has been some recent interest in the British ophthalmological and optometric literature[1,2] on the subject of the positive predictive value of patients referred by their optometrist under suspicion of having glaucoma. The above paper identified a false positive referral rate of 34 in 86 referrals (39.5%) and cited references to support the underlying implication that there are too many false positive referrals.

Obviously *any* false positive referral is cause for concern given the load on outpatient departments. However, speaking from the optometrist's perspective, the underlying implication of over-cautious referral needs more detailed examination before simplistic conclusions are drawn.

Suppose an optometrist saw 10 000 patients over the age of 40 years, and assume the incidence of COAG amongst this group to be 2%. Were the optometrist to have at his disposal a screening test that conferred hitherto unheard of levels of 99% sensitivity and 99% specificity, the results would be as follows. A test that is 99% sensitive would detect 198 of the 200 glaucoma cases (true positive) and regrettably miss 2 (false negative). Similarly, a test that was 99% specific would show normal findings for 9702 patients (true negative) but would fail 98 normals (1% of 9800) as abnormal (false positive). The resultant false positive referral rate would be 98/198 (49.5%), which is not enormously different from the typical figures quoted in this paper and others. Given that it is commonly stated that 50% of cases are already detected,[3] the numbers requiring such opportunistic case detection may be significantly less than these assumed figures, which would lead to an even higher false positive ratio.

Fundamentally, the difficulty lies with glaucoma being a disease of relatively low prevalence that is difficult to diagnose unequivocally at a stage of minimal optic nerve head damage. Put alongside this the structure of the legislation circumscribing optometric practice and it can be seen that this mitigates directly counter to the clear need for more extensive and repeat testing prior to referral. In addition, the lack of nationally agreed referral criteria and good inter-professional communications including feedback make it difficult to see how any improvements are going to be made within the current system. Our professions need to work closer together so that optometrists can achieve acceptable false positive referral rates.