When is acute onset comitant esotropia a sign of serious neurological disease?

Editor,—We read with interest the paper by Hoyt and Good in which they outlined the differences between patients with acute onset comitant esotropia with coexisting central nervous system pathology and those who were otherwise neurologically intact.1

We fully agree with the authors that the vast majority of cases will have no obvious underlying neurological cause, making it of the utmost importance to have good clinical criteria for use in the selection of those patients who will need immediate neurological and neuroradiological investigation. As the authors state, the patient who presents with diplopia should prompt careful consideration of whether the strabismus is a sign of serious central nervous system pathology. The ophthalmologic history (especially that of previous strabismus and occlusion therapy) and neurological findings (such as headache, papilloedema, clumsiness, etc) are helpful in distinguishing ophthalmic from neurological causes of strabismus. Enquiry about previous head trauma is most important.2 The authors reached the quite correct conclusion that the presence of nystagmus in cases of acute comitant esotropia should be considered an abnormality that warrants neurological investigation.

Furthermore, we do not agree that a history of monocular visual loss need cause little worry for the clinician. Unilateral reduced visual function is one of the various factors that may be a cause of comitant esodeviation.3 Both tumours of the optic nerve and chiasm region may be responsible for this unilateral reduced visual function. In such cases associated with visual loss, examination of pupilary reactions and visual fields is indispensable. Both should be normal in uncomplicated esotropia.

In summary, we believe that in cases of acute onset comitant esotropia, the same risk factors for serious neurological disease should be considered as in cases of paralytic strabismus. Recently, those risk factors were summarised in the mnemonic: DON'T PANIC with ocular motor palsy.4 Using the mnemonic should help in the systematic analysis of the problem and in judging the seriousness of the situation.

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Reply

Editor,—We thank Cruysberg, Draijer, and Sellar for their thoughtful and important comments on our paper. We do not disagree with the concern about the possibility esotropia associated with afferent visual pathway disease. However, we were only addressing acute esotropia presenting with diplopia. Our experience has been that the esotropia associated with monocular visual loss and tumours of the optic nerve and chiasm is more indeterminant in its onset and rarely associated with diplopia. This is not meant to minimise the importance of these tumours and the associated esotropia, but to say that this group of patients usually falls outside the clinical profile that we were addressing. We thank the authors again for their comments.

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Refraction and visual results and patient satisfaction after excimer laser keratectomy for myopia

Editor,—I would like to take issue with some of the points raised in the paper by Brett L Halliday.1

In the discussion there is a statement 'Excimer laser surgery is still a relatively new procedure. It appears to be safe, especially when compared with other refractive surgical procedures, etc'. This statement is unrefereenced. The only comparable procedure for low degrees of myopia with which there is a fair comparison for photorefractive keratectomy (PRK) is radial keratotomy (RK). The data on RK are much more extensive in time than data for PRK, and the 10 year PERK study2 shows at least comparable results with the 1–6 dioptre range for RK. I note that patients when interviewed with regard to the potential treatment for their myopia were only offered the one solution! I further note that nowhere in the article is corneal topography mentioned, neither preoperatively nor postoperatively, when the results can be monitored, as by Wilson and Klyce3 that a representative cohort of patients attending for refractive surgery revealed a significant incidence of corneal shape abnormalities including contact lens warpage and previously undetected and early keratomalacia. The author therefore shows disregard for the comprehension of corneal shape when a procedure designed to alter shape is about to be performed. Were the dissatisfied patients or geons and laser clinics through the civil courts.

I agree that high spending laser clinics need to treat large numbers of patients in order to generate profit. In comparison, low budget radical keratotomy never became very popular. This was not because radial keratotomy was perceived by the public as dangerous or unpredictable, but that, in the absence of massive capital investments, there was no need for the professionally generated, high profile, media campaigns and expensive advertising which have become the hallmark of so many private laser clinics. This promotional attitude is a dreadful way to present a surgical technique to the public.

Corneal scarring and irregular astigmatism may ultimately lead some patients to require corneal grafts. Other patients, justifiably angry about their inadequate preoperative counselling are choosing to take action against surgeons and laser clinics through the civil courts.

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Low vision

Editor,—We read with interest the editorial 'Low vision: a parochial view'.1 As Dickinson said, it is becoming increasingly recognised that the use of the hospital eye service presup-
