When is acute onset concomitant 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 concomitant esotropia with coexisting central neurological 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 ophthalmic 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 reach the quite correct conclusion that the presence of nystagmus in cases of acute concomitant esotropia should be considered an abnormality that warrants neurological investigation.

However, 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 concomitant esotropia.3 Both tumours of the optic nerve and chiasmal region may be responsible for this unilateral reduced visual function. In such cases associated with visual loss, examination of pupillary reactions and visual fields is indispensable. Both should be normal in uncomplicated esotropia.

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


**Reply**

**Editor,—**We thank Cruyberg, Draaijer, and Sellar for their thoughtful and important comments on our paper. We do not disagree with the concern about the sensory 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 indeterminate 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 comment.

**CRIEG S HOYT**
**WILLIAM V GOOD**
**University of California, UCSC Ophthalmology Department, Pediatric Ophthalmology, 400 Franklin Avenue, Ste 1290, Box 0344, San Francisco, CA 94143, USA**

Refractive 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 unreferenced. 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 study shows at least comparable results with the 1–6 dioptre range for PRK. 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. It has been shown by Wilson and Klyce2 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 keratocones. 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 the poor results a consequence of decreased ablation? Your readers should be aware that a professional approach to refractive surgery must include documentation of the preoperative status of the cornea, then questions of adverse reactions which arise later can be correctly investigated.

I return to the statement already quoted that PRK appears to be safe especially when compared with other refractive surgery procedures. One should remember the economic background to PRK. Lasers are extremely expensive and their obsolescence is rapid. The only way investment can be recouped is by a high volume of treatments. In other words treating a lot of patients as a result of marketing a procedure with a very short track record. Accordingly, even if the complication rate is relatively small, given a large volume of patients the actual number of patients so affected may be significant. Everyone involved in PRK has some sad tales to tell for this is not a reversible procedure and corneal replacement in whole or in part may be the only solution for some of the poorer results.

**EMANUEL ROSEN**
**10 St John Street, Manchester M3 4DY**

**Reply**

**Editor,—**I wholeheartedly agree with Mr Rosen’s attitude; we seem to share a similarly circumspect view of the excimer laser.

Time will ultimately prove the safety or otherwise of the excimer laser. My statement that the procedure ‘appears to be safe’ is based on the results reported in my paper. Inadvertent corneal perforation at the time of surgery is unknown with excimer laser treatment but well reported in radial keratotomy where it can lead to blindness.

It is correct to regard corneal topography as being mandatory before considering laser surgery for all the reasons stated. Mr Rosen may not realise that the patients reported in this paper were treated over a 17 month period starting in 1991. At that time corneal topography was in its infancy and Klyce’s excellent paper did not appear until 1994.2 I agree that high spending laser clinics need to get large numbers of patients in order to generate profit. In comparison, low budget radial 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 procedures, are choosing to take action against surgeons and laser clinics through the civil courts.

**B L HALLIDAY**
**Shrewsbury SY1 2QZ**

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 prescrip-