CORRESPONDENCE

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 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 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 esodeviations.[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 systematic analysis of the problem and in judging the seriousness of the situation.

History	*Examination*
D=diplopia	P=papilloedema
O=ophthalmic symptoms	A=anisocoria (unequal pupils)
N=neurological and general symptoms	N=nystagmus
T=trauma	I=incomplete visual fields
	C=corneal hypoaesthesia

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Reply

EDITOR,—We thank Cruysberg, 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 comments.

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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[2] 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 Klyce[3] 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 keratoconus. 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 decentred 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.

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Reply

EDITOR,—I wholeheartedly agree with Mr Rosen's attitude; we seem to share s 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.

I agree that high spending laser clinics need to treat 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 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 prescrip-

tion (HESP) forms to permit a visit to an optician in order to obtain a low vision aid is not only notoriously expensive and wasteful, but is also not very helpful for the patients. The editorial suggested that reports of alternative models of low vision aid (LVA) services are 'essential' so that we can all learn from each other when setting up new clinics. The paper by Shuttleworth *et al*[2] in the same issue of the *BJO* is one such encouraging report. From Reading, a paper is in preparation reporting our experiences. Interestingly, our LVA clinic was set up as a result of a senior house officer's audit into our existing 'service' (HESP forms). The surgical business unit manager, who was genuinely motivated by a desire to establish a better service for the patients (as well as the obvious financial implications), worked closely with the SHO and the hospital audit department. The fact that the manager had been involved from the start, and had participated in the original audit meeting, undoubtedly helped in the process of the establishment of the LVA clinic. The presentation to the whole department at the audit meeting enabled interested colleagues, such as the orthoptists, to state what role they wanted to play in the training of the patients to use the LVAs. Subsequently, the orthoptists, in fact, liaised with the sensory needs department and the new clinic now also has input from the sensory needs service.

This was an example of a junior's audit project conducted in collaboration with a hospital manager which helped to change clinical practice in a district general hospital.

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Patient selection may be the key for the success of shared care for glaucoma

EDITOR,—We agree with Hitchings' views that shared care for glaucoma has, at least on the face of it, much to recommend it.[1] Despite the growing enthusiasm, it has not yet received critical evaluation for national adoption. Shared care is a way of integrating primary and secondary care services and is one of the targets set for the NHS in the 1990s.[2]

Shared care schemes have been in operation for patients with other chronic diseases such as diabetes,[3] asthma,[4] and hypertension for the past few years and such schemes claimed that shared care is clinically as effective as hospital outpatient department, cost effective, acceptable to the patients and general practitioners, and allows more new patients to receive specialist advice.[5]

Glaucoma patients constitute the bulk of the follow up patients in any general ophthalmic clinic. Our recent departmental audit revealed that 47% of all follow up patients are, in fact, the previously diagnosed chronic glaucoma sufferers. Any scheme that would reduce the work load of busy outpatient department without compromising the quality of care is welcomed by one and all.

We would like to highlight three important issues that need to be considered before embarking on any such ventures. Firstly, overcautious opticians running such schemes may result in more re-referrals to the hospitals defeating the basic objective. Secondly, the prerequisite to run a glaucoma shared care clinic is to have the same instrumentation and examination methods at the base hospital and the shared care centre. At present pneumatic 'applanation' tonometry, direct ophthalmoscopy, and a variety of visual field analysis techniques are popular with opticians in contrast with Goldmann tonometry and binocular non-contact funduscopy lens used by hospital doctors. Use of different methods might also mean unnecessary additional hospital referrals.

Finally, we think proper patient selection could be the single most important factor on which the success or failure of shared care schemes may depend. Understandably not all glaucoma sufferers are suitable for the shared care system. Ideally, only those patients with stable intraocular pressures and visual fields, and whose glaucoma is unlikely to need change of management should be recruited into such ventures in order to avoid inappropriate recycling of patients between hospitals and opticians. There is a need to identify the type of patients that are most suitable for shared care or otherwise. Currently, there are no established guidelines regarding patient selection for these new schemes and the outcome of pilot studies mentioned by Hitchings is eagerly awaited.

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Reply

EDITOR,—I thank Rao and O'Donnell for their kind comments on my commentary in the *BJO*.

They raise a number of useful points. It needs to be remembered that shared care has two aspects – diagnosis and follow up. For diagnosis the most cost effective method with the highest sensitivity and specificity possible is required. This will need to be nationally agreed and standardised. For follow up, there is greater latitude in the methods chosen although the same rigid protocol as that which constitutes change will be required.

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Use of glycopyrrolate as a prophylaxis for vaso-vagal syncope during retinal photocoagulation

EDITOR,—Application of a fundus contact lens for retinal photocoagulation may, though uncommonly, result in a vaso-vagal syncope as a result of oculocardiac reflex bradycardia. Preoperative intramuscular anticholinergic agents were found to be partially protective against oculocardiac reflex bradycardia in adult squint surgery.[1] We report on three patients in whom we used intramuscular glycopyrrolate to diminish the oculocardiac reflex so that retinal photocoagulation could be carried out without resorting to retrobulbar or general anaesthesia.

PATIENT 1
A 26-year-old woman who previously suffered a vaso-vagal syncope after 23 burns of retinal photocoagulation was able to receive between 100 and 1250 burns per session when 0·2 mg of glycopyrrolate was given intramuscularly 30 minutes before each procedure.

PATIENT 2
A 31-year-old woman had had one vaso-vagal syncope and complained of feeling faint in subsequent treatment sessions when she received between 20 and 350 burns of retinal photocoagulation. She was able to receive up to 421 burns without any complaints with intramuscular glycopyrrolate before treatment.

PATIENT 3
A 47-year-old man, who previously had a vaso-vagal syncope after only four burns, was able to receive between 360 and 1002 burns of retinal photocoagulation with pretreatment intramuscular glycopyrrolate.

We did not encounter any of the anticholinergic side effects such as sinus tachycardia and dry mouth in our patients. Glycopyrrolate was used as the anticholinergic agent because it has been found to be associated with less tachycardia and dysrhythmias than atropine.[1–3] It also produces less central nervous systemic effect as it penetrates the blood-brain barrier poorly.[4]

In view of the above reports, we would suggest that intramuscular glycopyrrolate before retinal photocoagulation could be useful in preventing a vaso-vagal syncope in susceptible patients. Though a randomised, double blind, controlled study would be desirable, it is impractical owing to the rarity of the problem. Patients receiving intramuscular glycopyrrolate should be observed and monitored after treatment.

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