Sir,

I enjoyed reading the article by Lyons *et al.* 'Acute acquired comitant esotropia: a prospective study.'[1] In this context I find it of interest to give selected data from a Danish series of 12 patients, presented to the Danish Orthoptic Society, March 1986. All were referred for eye examination because of diplopia.

While 2 of the cases were associated with obvious VIth nerve palsies (in males aged 12 and 18, after trauma), 10 were regarded as clean comitant esotropias. Most were considered 'neurological'. Among them was an 8-year-old boy with a benign posterior fossa tumour similar to the 4-year-old female tumour case presented by Lyons *et al.* The 20 pd esotropia in our patient, however, did not require subsequent strabismus surgery. His binocularity was re-established within 6 months after surgical removal of his cerebellar astrocytoma. No patients had abducting nystagmus recorded.

The seven clearest cases of estropia are briefly summarised in Table 1.

All patients were seen in the regional Hillerød Hospital 1984–5. The accumulation of cases was embarrassing. Since then we have been on the look-out for similar cases in younger patients referred for diplopia, but did not find new comitant cases.

Reference

1. Lyons CT, Tiffin PAC, Oystreck D. Acute acquired comitant esotropia: a prospective study. Eye 1999;13:617–20.

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Sir,

We thank Dr Fledelius for his helpful and interesting letter describing a series of 12 patients presenting with diplopia of 'neurological' origin. Seven of these had a comitant deviation and are clearly described here. This adds to our paper by pointing out the fact that a comitant esodeviation may accompany other central nervous system (CNS) disease such as encephalitis or as a complication of trauma. Hydrocephalus is another CNS problem which may present in this way.

In these circumstances, the significant underlying factors would usually be identified by a thorough history and examination. In some patients (case 3), trauma or encephalitis allowed breakdown of a pre-existing phoria. Many of these patients returned to orthotropia spontaneously. However, few of us would accept influenza (case 4) as a precipitating factor of comitant esotropia without VIth nerve palsy in the absence of significant underlying hypermetropia. The question remains: Which of these patients can we afford not to scan?

The clinical details of patient 2 are not given but Dr Fledelius' letter is further support for the assertion that acquired diplopia with a comitant esodeviation, particularly in the absence of other underlying factors associated with strabismus (high hypermetropia, family history of strabismus), should be treated cautiously. Abducting nystagmus, previously described in the context of comitant esotropia as a useful pointer to identify patients harbouring a CNS tumour, may represent a 'fixation duress' in cases with very early VIth nerve involvement. Dr Fledelius did not find abducting nystagmus in his series, and we found this sign to be absent in the one patient who did have a large midline cerebellar tumour.

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Table 1. *Summary of seven cases of comitant esotropia*

Patient no.	Sex, age (years)	Neurological diagnosis	Clinical course
1	f, 7	Brain contusion, bilateral papilloedema	30 pd esotropia days 10 and 22 Orthophoria on day 35
2	m, 8	Cerebellar astrocytoma	Diplopia for 10 weeks. Papilloedema at admittance. Esotropia cured after brain surgery (see text)
3	f, 13	Encephalitis, papilloedema	Previously +4.0 sph and esophoria. Now breakdown into 18 pd esotropia. Spontaneous cure after 10 weeks
4	m, 14	Severe influenza	30 pd comitant esotropia. No effect of +1.75 sph. Recurrences after repeated squint surgery
5	f, 15	Brain stem radiculitis	10 pd esotropia and diplopia. Spontaneous cure
6	f, 22	Brain contusion, occipital bone fracture	Diplopia noticed after 7 days. +18 pd esotropia. Spontaneous cure after 50 days. Stereopsis restored
7	f, 41	Brain stem disease	Diplopia after 1 week with headache and vomiting. No papilloedema. A-pattern 20 pd esotropia. Orthophoria restored with 60 days

Sir,

I read with great interest the article by Patel and Rosen entitled 'Pre-operative malposition of foldable implants', which they termed IOL flip.[1] The authors advocate the use of a closed folder to support the optic if the IOL starts flipping. A second instrument through the side port can be introduced into the anterior chamber to achieve the same effect. The use of a second instrument facilitates gradual unfolding of the intraocular lens and can be used to disengage the snagged trailing haptic. In my experience, this bimanual manoeuvre is more controlled and prevents inadvertent corneal endothelial touch by the lens folder.

In tropical countries, during the winter Acrysof intraocular lenses become harder. They pose a difficulty in folding and are predisposed to lens flip. At times the lens may get folded in the reverse direction. Keeping the Acrysof intraocular lens in a warmer place, such as under a microscope light, makes the lens pliable and facilitates its proper folding.

Reference

1. Patel CK, Rosen PH. Pre-operative malposition of foldable implants (IOL flip). Eye 1999;12:255–8.

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