

# Acute Onset Esotropia Associated with Neuro-Toxic Effects of Anticonvulsant Medication

**A**cute onset esotropia (brainstem esotropia) occurs as a result of a lesion within the posterior fossa. This results in a disturbance to the otolithic input pathways to the horizontal vergence centre. Clinically, a concomitant esotropic deviation is evident which is often associated with fine acquired nystagmus.<sup>1</sup> The authors report a case of acute onset esotropia in an epileptic patient occurring during the month of Ramadan.

## Case report

A 15-year-old female presented in October 2004, complaining of horizontal diplopia and variable oscillopsia. The patient was a known epileptic, taking sodium valproate 1600mg and lamotrigine 300mg daily. At the time of presentation the patient had been fasting for Ramadan for 14 days.

Orthoptic assessment revealed a concomitant 10Δ left / alternating esotropia for all distances with homonymous diplopia (Fig 1). No abnormality of ocular motility was found. Downbeat nystagmus was evident in all positions of gaze. Lees screen analysis verified orthoptic clinical findings, demonstrating a concomitant deviation (Fig 2a). Ophthalmological examination was unremarkable and radiological investigation revealed no anomaly. A diagnosis of acute onset esotropia was given. Neuro-toxicity associated with the epileptic medication was postulated as a possible aetiology. The patient was advised to cease fasting and medication dosage should be reassessed.

By April of the following year, following a reduction in epilepsy medication, the patient was found to be asymptomatic and controlling to an esophoria with no evidence of nystagmus. The patient demonstrated evidence of binocular single vision. Lees screen assessment confirmed a reduced angle convergent deviation (Fig 2b) correlating with orthoptic clinical findings.

The patient presented again in October 2005 with identical symptoms following fasting for three days. Clinically the patient showed very similar findings to that of the previous episode (Fig 2c).

The patient remains under review in the neuro-ophthalmology clinic.

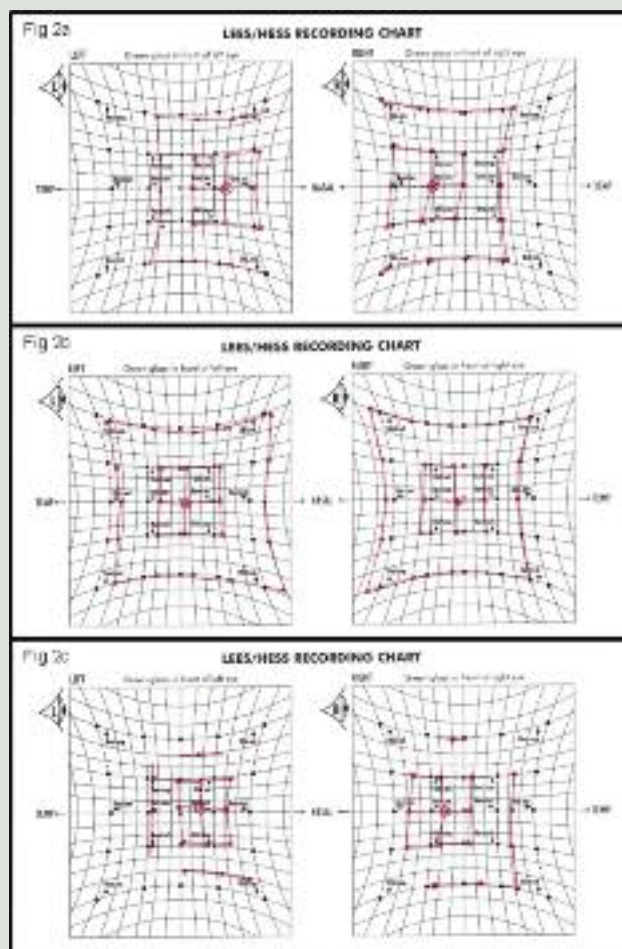
## Comment

Acute onset esotropia is thought to arise as a result of lesions within the posterior fossa and in particular, involving the cerebellar vermis.<sup>1</sup> The translational component of the vestibuloocular reflex (VOR) has been postulated as an integral constituent of the physiology of brainstem esotropia. The VOR is 'a mechanism to maintain clear vision during rotation of the head and stimulated by the angular acceleration sensors of the labyrinthine semicircular canals.'<sup>2</sup> The VOR consists of both rotational (angular) and translational (linear) components in order to respond to movements of the head whilst maintaining clear fixation of a target.

Translational VOR occurs as the centre of rotation of the eyes are displaced from the centre of rotation of the head.<sup>3</sup> Therefore, when the head is rotated purely horizontally the orbits are effectively rotated and translated linearly. Rotational VOR accounts for the rotation of the orbits whilst the translational component accounts for the lateral translation of the orbits,



Figure 1.



resulting in an amalgamated rotation of the eyes.

In acute onset esotropia disturbance to the otolithic inputs elicits an ocular reaction to counteract the apparent movement of the head, clinically demonstrable as a concomitant esotropia.

Lamotrigine is known to have dose-related side-effects, including diplopia and nystagmus.<sup>4</sup> The occurrence of two separate episodes of identical clinical features coinciding with Ramadan suggests that the high dosage of epilepsy medication compounded by fasting, resulted in a more rapid absorption and therefore increased levels of medication within the blood. The resultant toxicity was postulated as the most probable aetiology.

A previous case of acute onset esotropia was reported in a young boy after increasing the dosage of another anticonvulsant, carbamazepine. Similar symptoms of horizontal diplopia and nystagmus were present. Symptoms resolved following a reduction in medication.<sup>5</sup>

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## References

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