

**VIGABATRIN** (Comment)**Hazards associated with the use of vigabatrin**

The main adverse effect associated with sustained vigabatrin use is progressive, concentric, peripheral visual field damage usually affecting both eyes almost equally. This problem can be hard to assess in a very young child, but there is increasingly robust evidence that the problem is uncommon in the first year of life. In addition, it is hardly ever seen until the drug has been in use for at least six months and, more usually, several years. The findings of a recent follow up study of the drug's use in older children certainly support such a view (Wild *et al.*, 2009). Nevertheless, although central vision is only very rarely affected, peripheral visual field loss can eventually become quite disabling, so all children on sustained treatment need to be screened at six-monthly intervals to see if a problem of this sort is starting to develop. Luckily, testing for this, in skilled hands, seems to be more reliable, even in young and disabled children, than many would have thought possible (Agrawal *et al.*, 2009). Although these concerns have led to some questioning the continued use of this drug (Harinder, 2009; Cohen *et al.*, 2009), most would still support short term use, and longer term use as well with appropriate monitoring where this seems the best treatment option (Willmore, *et al.*, 2009).

There have also, very recently, been three independent reports, all suggesting that abnormalities may develop in the appearance of the brain on MRI scanning in children – particularly in those given a high dose of vigabatrin (125 mg/kg a day or more) for some months – abnormalities that are usually asymptomatic and transient and usually resolve once treatment is stopped. The thalami and the basal ganglia usually seem to be the areas most noticeably affected (Milh *et al.*, 2009; Pearl *et al.*, 2009; Wheless *et al.*, 2009).

Additional, as yet unpublished, data have suggested that the treatment of infantile spasms with vigabatrin can lead to the development of a movement disorder including dystonia and dyskinesia in a few children. The precise significance of this 'movement disorder' is not yet clear. Neither is it clear whether these infants also have a detectable MRI abnormality. Several of the affected children have had a known cause for their infantile spasms, and it is possible that these findings are merely a feature seen in children whose infantile spasms are one aspect of an underlying metabolic disorder such as a mitochondrial cytopathy. Abnormal involuntary movements are a relatively common feature in many such children and can be seen in children who have never taken vigabatrin.

The 'take home' message has to be that vigabatrin should only be prescribed by those with access to the facilities needed to assess the true underlying cause for the seizure activity, and also in a position to organise competent and well coordinated follow-up and support for the children and their families.

**References**

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