

an excess incidence among doctors and nurses.

The sharply conflicting data from the two studies suggests that one may be biased. The lack of any excess of MS among spouses of MS patients<sup>3</sup> indicates that MS is not (or is very rarely) a transmissible disease among adults. This observation, as well as our study, and the potential biases outlined above suggest that the incidence and mortality of MS among doctors and nurses is likely to be close to that in the general population.

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- 1 Dean G, Gray R. Do nurses or doctors have an increased risk of developing multiple sclerosis? *J Neurol Neurosurg Psychiatry* 1990;53:899-902.
- 2 Shepherd DJ, Downie AW. Prevalence of multiple sclerosis in north-east Scotland. *BMJ* 1978;2:314-16.
- 3 Editorial. Risk factors for multiple sclerosis: race or place? *J Neurol Neurosurg Psychiatry* 1990;53:821-3.

### Evaluation of vigabatrin in refractory epilepsy

We were interested to read the report by Sander *et al* of their experience of vigabatrin in 128 patients with severe medically refractory epilepsy, and in particular their comments on neurotropic side effects.<sup>1</sup> We began using vigabatrin at the beginning of 1990 and were struck by the high incidence of such side effects. We therefore recorded the effects of vigabatrin therapy in 30 sequential patients.

All the patients had localisation related seizures intractable to conventional medical therapy. The seizures were complex partial in 24, focal motor in five, and secondary generalised in one patient. Eleven patients had secondary generalised seizures in addition to focal seizures. The average age was 33.9 years with an average duration of seizure disorder of 17.8 years. The first 10 patients were started on a dose of 2000 mg/day, the rest on 1000 mg/day (see below). The maximum dose used was 4000 mg/day. The patients were started on vigabatrin between January and August 1990, and all are still being followed up. Side effects permitting, all had a minimum three month trial of therapy.

Of the 30 patients started on vigabatrin only seven remain on it. The drug was withdrawn in 10 patients because of lack of effect, in four patients who relapsed following an initially good response (all the relapses have occurred within three months of starting therapy), in two patients whose seizures appeared to become worse on vigabatrin, and in seven patients because of neurotropic side effects. This type of side effect was most common in the group as a whole, and included drowsiness (five), irritability (three), anxiety (one), depression (two), emotional lability (two), confusion (one) and psychosis (one). Other side effects included weight gain (two patients) and headache (one patient).

The seven (23%) patients remaining on vigabatrin therapy have all had either a useful reduction (> 30%) in seizure frequency, and/

or significant amelioration of seizure manifestations, but none is seizure free.

The starting dose of vigabatrin was reduced after the first 10 patients because seven of these patients suffered neurotropic side effects, in two cases severe. There were no severe neurotropic side effects in patients started on the lower dose of 1000 mg/day. All of our patients who showed a therapeutic response did so at a dose of 2000 mg/day or less, and there were no patients in whom increasing the dose beyond this produced any further response.

Our group of patients was different from that of Sander *et al*, in the type of epilepsy, and in being composed entirely of outpatients who may have less severe disease. We have found its therapeutic effect less good, but our experience of the neurotropic adverse effects associated with vigabatrin is similar, and we too have seen tolerance develop in a significant number of patients. A response rate of 23% in patients refractory to first line anticonvulsant agents is certainly worthwhile, but careful supervision is required in the early stages of therapy, and we agree with Sander *et al* that vigabatrin should be used with particular caution in those with a previous history of psychological problems.

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- 1 Sander JWAS, Trevisol-Bittencourt PC, Hart YM, Shorvon SD. Evaluation of vigabatrin as an add-on drug in the management of severe epilepsy. *J Neurol Neurosurg Psychiatry* 1990;53:1008-10.

## BOOK REVIEWS

**Handbook of Sleep Disorders.** (Neurological Disease and Therapy Series/6). Edited by M THORPY. (Pp 817 Illustrated; Price US & Canada \$165.00; All Others \$198.00.) New York, Marcel Dekker Inc, 1990. ISBN 0 8247 8295 X.

This is a new and attractive book about sleep disorders. The stated aim is to be a comprehensive summary of knowledge in nearly all aspects of human sleep. Under the able editorship of Michael Thorpy the book substantially but not completely fulfils this claim. About a third of the book covers the physiology and anatomy of sleep mechanisms and the pharmacology of sleeping and waking. The remainder is concerned with clinical sleep medicine. The code of practice of American sleep disorders centres is amply covered, and reflects the very high ratio of North American authors. There are two starting points for this approach. The first is the chapter by Lemmi on sleep disorders centres and polysomnographic evaluation, and the second is the recent American-inspired, international classification of sleep disorders. This system may result in a primary focus on the sleep laboratory rather than on the patient.

The coverage of primary and secondary sleep disorders as well as circadian rhythm problems is wide. Some might argue at the

use of the term "dyssomnia" to cover narcolepsy, obstructive sleep apnoea and other hypersomnias, but this classification is clinically relevant. Many of the sections on insomnia are particularly good, notably those by the Italian school, led by Lugaresi in his description of fatal familial insomnia. The discussion of parasomnias and secondary sleep disorders is thorough but I wonder if a disproportionate amount of the book is devoted to these topics. For example, the detailed focus on sleep disorders in many degenerative neurological disorders seems excessive. However the book is essential reading for European as well as American polysomnographers and contains outstanding sections on narcolepsy from Broughton and Honda, despite some apparent contradictions. Thus Broughton reports that monozygotic twins may be concordant for narcolepsy and cites three references in favour of this; in the next chapter the same references are cited by Honda to support the statement that no examples of complete concordance are known. The book is well produced, with good illustrations and figures and adequate references. It is however far too expensive.

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**Positron Emission Tomography in Dementia.** *Frontiers of Clinical Neuroscience Vol 10.* Edited by RANJAN DUARA. (Pp 186; Price: US\$ 89.95). New York: Wiley-Liss, 1990. ISBN 0 471 56804 X.

This is an elegantly bound volume in the Wiley-Liss series of *Frontiers of Clinical Neuroscience*. It reviews our current understanding of dementia using Positron Emission Tomography. At \$89, I picked up this book excited as if I had been invited to eat at an exclusive restaurant by a selection of famous transatlantic chefs.

The first course, which explained the methodology behind PET was excellent. Despite the risk of being a rather indigestible topic, it combined sufficient spices to make a most agreeable *hors d'oeuvre*. To the non expert this section was refreshingly easy to read and understand.

The main course in contrast, which tackled the metabolic deficit found in Alzheimer's disease, was rather disappointing. One of the reasons for this was the tone set by a prefacing chapter on cerebral atrophy. This was the wrong accompaniment for a review of the PET findings and the savoury topic of atrophy should have been reserved for after dinner. An opportunity was missed to provide an elegant meta-analysis of PET results. The presentation was not as good as I expected and the chef must have been disappointed with the quality of the plates. However the chapter on Huntington's chorea was excellent.

For dessert there was a wide range of topics, including, ligands and PET, activation paradigms, and SPECT. It was a little over-ambitious, since it failed to mention a number of important findings. For example the studies of 18F Dopa in Alzheimer's Disease, and some of the case reports of PET in the rarer dementing syndromes, were absent. Work from Mesulam and others from this side of the Atlantic on focal degeneration were omitted. The foreword expressed a