Matters arising

including hyperammonaemia and hyponatraemia, but also in iatrogenic encephalopathies such as those produced by lithium and/or levodopa. Although some tentative hypothesis can be proposed, the pathogenesis of periodic complexes still remains unknown.

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Smith and Kocen reply:
We thank Drs Brousselle and Chazot for drawing to our attention their case of a Creutzfeldt-Jakob like syndrome due to lithium and levodopa toxicity, first reported in 1972. The case is very similar to those we described, although the clinical features appeared rather sooner after the first administration of lithium than in our two patients, who developed clinical features after 1 and 2 months. The dose of lithium used by Broussolle et al however was much higher.

A further case of this syndrome has recently been identified (personal communication, Dr K Chiappa).

Book reviews


This is an excellent book. Iain Wilkinson has a high reputation for undergraduate teaching at Cambridge and this is reflected in his book. His priorities are clear as he has set out in the Preface. I also liked the way the book itself is set out. It is a pleasure to see illustrations that are related to text on the same page and are of good quality. I particularly liked the line illustrations and the diagrams are clear. It is good to see that common conditions are given appropriate prominence and detail whereas uncommon conditions such as myasthenia gravis are indicated as such. I have a number of quibbles, for example I cannot find ptosis listed in the main index but the Shy-Drager syndrome is. It would be better if eponyms were used less. I think it is a pity that the coma scale is introduced without the relevant numbers because the student cannot then grasp the significance of, say, a coma scale of 6 as opposed to a coma scale of 12. I have not come across the mnemonic A, E, I, O, U for coma before but its simplicity is obvious. It is a pity that the diagrams related to Parkinson’s disease did not make it clear that the striatum relays to the motor cortex and also that cerebellar damage results in changed inputs to the primary motor pathways.

However, overall this is an excellent book with a very good all round approach to an understanding of neurology at student level. I particularly liked his integration of counselling and genetic advice and I think this book will go a long way to dispel the myth that neurology is unduly complicated, a myth which tends to put off so many undergraduates.

Not least of all its attributes is the fact that its price is modest.

JE REES


This book is based on two seminars: one at Queens Square in December 1986, the other at Winston-Salem, N. Carolina in May 1987. Both were organised by the WFN Research Group on Cerebrovascular Disease. The topics under consideration are not the most promising areas for fruitful discussion nor areas for therapeutic euphoria; yet, the excellence of parts of this book will show otherwise. When you open it, your enthusiasm will not be enhanced by the cascade of dysphonious acronyms which assault the senses. I will not reproduce them, but if they are to be permitted at all, the editors might at least have adhered to one for the topic of the conference: not SDAT, DAT and AD in different sections.

The 26 chapters include a thorough coverage of clinical features, CT, PET and MRI scanning, electrophysiological tests as well as appraisals of sonography, haemorheology, biochemistry and a number of clinical drug trials. Some of the more general chapters present authoritative overviews, but they are too brief to be informative, and, though good introductions to leave the heavy science at a symposium, might usefully have been omitted from the published work.

Progress may be hampered until fundamental issues of definition have been resolved, and it is clear they have not. No laboratory test or combination of clinical, psychometric and radiological criteria have been put forward to define dementia. Some feel dementia is not an all or none phenomenon, but a gradually developing entity; this approach beggars definition and if accepted would negate epidemiological studies and therapeutic trials. We are told that vascular lesions may not cause dementia; if they do, the cognitive impairments produced may differ qualitatively from those resulting from the parenchymal atrophy of Alzheimer’s disease. Some would argue for this qualitative separation and emerging dementia should not be uncritically applied to both. Ischaemic scores are of no use in excluding Alzheimer’s disease, nor are they helpful in differentiating so called mixed cases from vascular dementia. There is a 30% incidence of normal CTs in mixed or vascular dementias, and low densities in the periventricular white matter, characteristic of Binswanger’s disease, are also present in 10% of non-demented 55 to 70 year olds and 33% of patients with Alzheimer’s disease. Incidentally, if you like trendy new names as well as acronyms try “Leuko-araiosis” for the rarified white matter lesions.

The overall impression is that experts still apply disparate criteria for classification, study populations of different ages and with different aetiologies and employ different psychometric tests. Thus a unified picture fails to emerge. I particularly enjoyed reading Mirsen and Hachinski on epidemiology and classification, Philip Wolf and colleagues on epidemiology and prevention, Ross Russell on microvascular and macrovascular occlusions and Nichols’ splendid review of Binswanger’s disease. Despite the rather forbidding subject matter, this is a useful compilation.

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References