than the Spanish case. The EEG, normal for background activity, did not differ from an EEG of primary idiopathic generalised myoclonic epilepsy and the patient had had only one isolated myoclonic seizure of the upper arms.

Rubio *et al*¹ have confirmed our reported data and it is interesting that the less aggressive skin and muscle biopsies are diagnostic in the early stages of the disease.

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Devic's neuromyelitis optica and Schilder's myelinoclastic diffuse sclerosis

I read with interest the article by Hainfellner *et al*¹ reporting the occurrence of Devic's neuromyelitis optica and Schilder's myelinoclastic diffuse sclerosis. I was surprised to find no information about anti-DNA antibodies or complement levels. Furthermore it was stated that the erythrocyte sedimentation rate was 27 mm in the first hour and that necropsy revealed perivascular lymphoid cell infiltrates of varying prominence. These findings, including the observation of cerebrospinal fluid immunoglobulin abnormalities, can be seen in systemic lupus erythematosus (SLE).² It is known that SLE may induce Devic's syndrome and may lead to a false diagnosis of demyelinating disease, especially when systemic manifestations of the disease are not yet present.^{3,4} I followed a patient with SLE and Devic's syndrome who was reported elsewhere.⁵ What are the arguments against SLE being the cause of the disease observed in the patient reported by Hainfellner *et al*?

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NOTICES

Riyadh Armed Forces Hospital Fourth International Course on Magnetic Resonance Imaging

The Departments of Radiology, Medical Physics and Medical Studies of the Riyadh Armed Forces Hospital are sponsoring the Fourth Course on Magnetic Resonance Imaging (MRI) on 2-5 October 1994. The course will provide an overview of MRI technology, and basic principles, current, and future applications of MRI in the whole body. Current and potential applications of MRI spectroscopy will also be discussed. Small group workshops on basic physics, neuro-applications, musculoskeletal and genitourinary systems will be provided for all participants. **Fees:** 1300 Saudi Riyals (1\$ = 3.75 Riyals); 650 Saudi Riyals for medical staff-in-training; 325 Saudi Riyals for radiographers. **For further information contact:** Department of Medical Studies, Armed Forces Hospital, PO Box 7897, Riyadh 11159, Saudi Arabia. **Tel:** ++966 1 477 7714 (Ext. 2289/2269); **Fax:** ++966 1 477 7194/477 9168.

Third International Congress of Movement Disorders

The congress will be held on 8-12 November 1994 in Lake Buena Vista (Orlando), Florida, USA. The deadline for abstract submission is 1 April 1994. For further information contact: Central Headquarters Office, The Movement Disorder Society, PO Box 6, Clarastrasse 57, CH-4005 Basel, Switzerland. **Tel:** ++41 61 691 51 11; **Fax:** ++41 61 691 81 89.

BOOK REVIEWS

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the United Kingdom and for members of the British Forces Overseas, but overseas customers should add £2 per item for postage and packing. Payment can be made by cheque in sterling drawn on a United Kingdom bank, or by credit card (Mastercard, Visa or American Express) stating card number, expiry date, and your full name.

Neuropsychology of the Amnesic Syndrome (Brain Damage, Behaviour and Cognition Series). By A J PARKIN and N R C LENG. (Pp 202 Illustrated; Price: £24.95). 1993. Hove (UK) Lawrence Erlbaum Assoc. Publishers. ISBN 0-86377-200-5.

For the purposes of this book the "amnesic syndrome" is defined in terms of permanent, non-progressive forms of amnesia linked to cerebral pathology. In practice the disorders considered as meeting this definition are amnesic phenomena associated with such things as the Wernicke-Korsakoff

syndrome, temporal lobe damage, diencephalic and thalamic lesions, herpes simplex encephalitis and anterior communicating aneurysms. After introductory chapters discussing models of memory and the assessment of memory disorders, neuropsychological research into amnesia resulting from each of the above causes is described. The book concludes with a discussion of some underlying theoretical issues and a brief chapter on the remediation of memory disorder.

Because the neuropsychology of amnesia is a heavily researched area with a large volume of potentially relevant published work, this book attempts to encompass a great deal in little more than 150 pages. This is inevitably selective and occasionally skates a little thinly over some details that might suggest reservations about the picture that is being offered. Nevertheless, the selection is generally judicious and the discussion is almost always clear and easy to follow as well as being up-to-date.

Overall this book can certainly be recommended as a useful and clear introduction to the psychology of amnesic syndromes. As such it fills a definite gap and deserves to be widely read by members of the various disciplines concerned with the problem of amnesia and not just by psychologists.

E MILLER

Down Syndrome & Alzheimer Disease (Progress in Clinical and Biological Research Vol. 379). EDITED by LYNN NADEL and CHARLES JEPSTEIN. (Pp 318; Price \$96.00). 1992. New York, Wiley-Liss Inc. ISBN 0-471-58841-5.

This book primarily serves as a permanent record of the proceedings of the 8th Science Symposium of the National Down Syndrome Society of the US, held in New York in January 1992. Its contents are 17 unedited, rapid reproduction manuscripts covering clinical aspects of dementia in Down's syndrome, chromosome 21, and animal models. Thus, there is considerable variation in the balance between review, and reporting of personal research work. A good proportion of the review material is also repeated from chapter to chapter.

The contents explore a number of associations. First, the development of features of Alzheimer's disease in Down's syndrome or trisomy 21, which pointed towards the second main observation of mutations of the amyloid precursor protein gene in some families with Alzheimer's disease. There is also an apparent increase in Down's syndrome in relatives of those with Alzheimer's disease. The observation that amyloid may be deposited in the brain in Down's syndrome and normal people as long as 30 years before the onset of dementia also appears to support amyloid as the prerequisite for neuronal loss and tangles. However the link between the amyloid gene mutations in Alzheimer's disease, and the pathogenesis of Alzheimer pathology in Down's and Alzheimer's remains obscure. Since preparation of the book, a cloud has descended over the validity of early claims that transgenic models with alterations of normal amyloid precursor protein expression develop neuronal loss and tangles.

This book will have a fairly limited appeal, mainly to budding research scien-