MATTERS ARISING

MRI lesions in younger healthy adults

We have read with interest the article about CNS lesions in chronic inflammatory demyelinating polyneuropathy (CIDP).[1] The author's findings were based on clinical, electrophysiological, and MRI investigations. They found MRI lesions in nine of 21 patients under the age of 50. This age dependent analysis was introduced by the authors as it is well known that MRI lesions are quite common in older healthy subjects. Even in younger normal subjects, however, such lesions may be more frequent than previously thought. We have investigated 32 healthy subjects (aged 20-50 years, mean age 31 years, 15 females, 17 males) for a comparison with a population of classic migraine patients.[2] Seven of our control subjects had hyperintense lesions on MRI in T2-weighted images (figure). Four subjects had lesions and three more than 3 lesions. The largest lesion was smaller than 3 mm in four subjects and larger than 3 mm in the remaining.

When our results are compared with those of Ormerod *et al*,[1] the incidence of MRI lesions did not differ significantly (table). Fisher's exact test for 2 x 2 tables: p = 0·097). Thus the authors may still be correct in claiming that MRI lesions are common in CIDP. There is certainly a trend but there is no proof for this as yet. Small hyperintense lesions should be interpreted with caution so far as demyelinating disease is concerned even in younger adults.

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1 Ormerod IEC, Waddy HM, Kermode AG, Murray NMF, Thomas PK. Involvement of the central nervous system in chronic inflammatory demyelinating polyneuropathy: a clinical, electrophysiological and magnetic resonance imaging study. *J Neurol Neurosurg Psychiatry* 1990;53:789-3.
2 Ferbert A, Busse D, Thron A. Microinfarction in classic migraine? A study with magnetic resonance imaging findings. *Stroke* 1991 (in press).

Table Incidence of hyperintense lesions on T2-weighted MRI in normal controls[2] and CIDP.[1] The figures indicate number of patients

	Lesions	No Lesions
Normal controls[2]	7	25
CIDP[1]	9	12

Ormerod replies:

I was interested to hear of the surprisingly high prevalence of MRI-detected cerebral white matter abnormalities in the control subject studied by Ferbert and Thron. In 32 neurologically normal subjects aged under 50 years, they found hyper-intense lesions on T2-weighted images in seven subjects. Our experience of normal subjects in this age group has shown cerebral abnormalities of this nature in less than 5% of cases. Our studies were all performed on a 0·5 Tesla imaging system and it is possible that with advances in MRI imaging technology, an increased number of abnormalities will be detected in a variety of conditions and also in normal control subjects.

The finding of hyperintense cerebral white matter lesions onT2-weighted sequences is a very non specific observation. We would still suggest that these changes in patients with chronic inflammatory demyelinating polyneuropathy (CIDP) are more frequent than would be expected and that they might indicate cerebral pathology, possibly related to the peripheral disorder. Nevertheless, as we concluded, it is clear that clinically significant CNS changes in CIDP are an uncommon event.

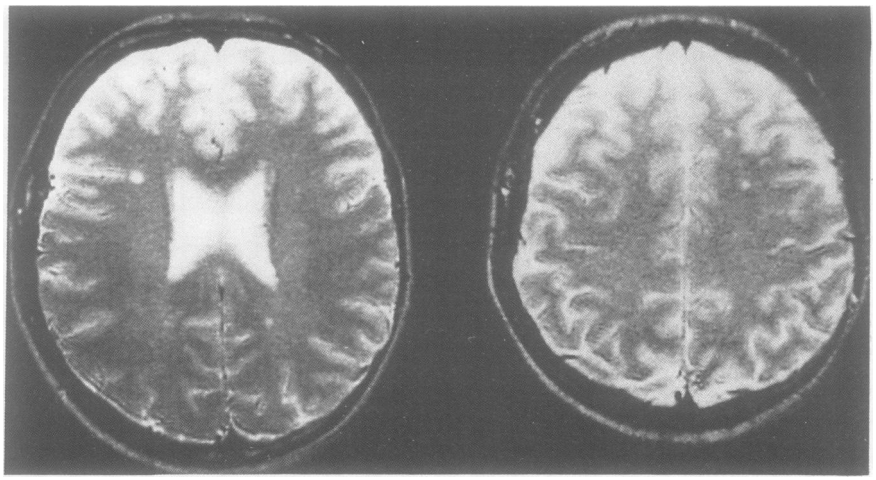

Figure 1 Hyperintense lesions on T2-weighted MRI in a 25 year old female physician without history or signs of a neurological disorder who was investigated as a control subject.

BOOK REVIEW

Current Neurology Vol. 11 (A Mosby Year Book). By STANLEY H APPEL. (Pp 321; Price £69.95.) 1991. London, Wolfe Publishing. ISBN 0-8151-0234-8.

An investment in this annual review would be justified by the single chapter on multiple sclerosis by Donald W Paty. Other sections cover Charcot-Marie-Tooth syndrome, myotonic dystrophy, neurofibromatosis, spinocerebellar degenerations, mitochondrial disorders, tremors, Alzheimer's and Parkinson's diseases and amyotrophic lateral sclerosis.

We learn about genetic mapping and linkage studies; of the elucidation of specific defective proteins, followed by cloning the complementary DNA (cDNA) and later its gene: the classic method (forward genetics) of study of hereditary disease. Reverse genetics uses data on the chromosome location of the faulty gene to isolate its genomic sequence which is then used to obtain the corresponding cDNA. Comparable molecular genetics now permeates every alleyway of research in mitochondrial and neurodegenerative disease, promising fundamental and therapeutic benefits undreamed of until the last decade.

The contributions are all of high quality, all show the industrious enthusiasm of real experts at the forefront of research, and collectively they are both an invaluable course of instruction and a substantial source of reference for neurologist and research worker alike. A first class compilation.

JMS PEARCE

NOTICE

The Upjohn prize for neurosurgical research.

This prize of $3,000 is offered by the Upjohn Company and awarded annually by the EANS. Those eligible for the prize should be neurosurgeons under the age of 40 at the time of submission, who are either fully trained or still in the course of their training. Applicants should be either a member of one of the national societies of the EANS or should be supported by such a member. The basis of the manuscripts submitted should be previously unpublished research work, either clinical or experimental or both, of relevance in the field of neuroscience. There are no specific regulations for the format or type of manuscript. Thirteen copies of the submitted manuscript together with a brief curriculum vitae should be sent to the chairman of the EANS Research Committee before 1 April 1992. The prize will be presented normally during the EANS training course of 1992 and the winner will be invited to attend that meeting and to present their work. The chairman of the EANS Research Committee is: Professor R Fahlbusch, Neurochirugische Klinik der Universitat, Erlangen-Nurnberg, Schwabachenlage 6, 8520 Erlangen, Germany.