recurrent tension type headaches and epigastric pain. Because of the epigastric pain she underwent gastroscopy under lidocaine spray anaesthesia without further premedication. Thirty minutes later she suddenly became disoriented and started to ask the same questions repetitively and two hours later she was referred to our department. On arrival she continued to repeat the same questions. She was alert and her comprehension, verbal expression, and fluency were normal. She was oriented with reference to person but not to time and place. There was a complete anterograde amnesia. On informal questioning, she was unable to remember life events of the past two years. Twelve hours after onset of transient global amnesia, anterograde amnesia was still present but retrograde amnesia had recovered. Twenty hours after onset, the anterograde memory disturbance had also recovered but a memory gap for part of the previous day remained. Doppler and duplex sonography of extracranial and intracranial cerebral arteries, EEG, and MRI of the brain were normal. A follow up clinical examination two months after transient global amnesia was normal without recurrence of similar episodes.

The first SPECT study was performed during the ongoing amnesic episode four hours after onset, the second at three weeks, and the third at three months after transient

global amnesia. Acquisition was performed one hour after injection of 900 MBq ^{99m}Tc -ECD (NEUROLITETM, Du Pont Pharma) on a triple head gamma camera (Picker PRISM 3000). After visual interpretation, lesion specific rectangular regions of interest were placed over the focus of interest and over the corresponding contralateral region in the hippocampal slices with calculation of the right to left ratio (normal range 0.9–1.1). Visual appraisal of the first SPECT study showed a focus of increased activity in the posterior caudal region of the right temporal lobe (figure) with a right to left ratio of 1.27 on quantification of the lesion specific region of interest. The second study visually disclosed a slightly decreased activity uptake in the same region with a right to left ratio of 0.90. In addition, decreased activity uptake was detected in the left temporal lobe. In the third study, no asymmetry was detected visually or by quantification.

The patient fulfilled all proposed criteria of transient global amnesia.¹ During transient global amnesia, SPECT showed increased activity uptake in the posterior caudal region of the right medial temporal lobe. After the event, a reverse pattern was seen, with a slight relative hypoperfusion in this region which resolved completely after three months. We therefore conclude that the time course of the reversible hyperperfusion makes it most probable that this feature is associated with the pathophysiology of the reported transient global amnesia. By contrast, the impaired left temporal perfusion three weeks afterwards was documented long after the patient's amnesic symptoms had recovered and might therefore not be a direct phenomenon.

Few functional imaging studies performed during transient global amnesia have been reported. The findings include unilateral reduction of regional cerebral blood flow (rCBF) in the left medial temporal lobe² and bilaterally decreased rCBF in the postero-medial part of both temporal lobes³ or thalamus.⁴ An even more pronounced divergence could be found after the attack. Some patients displayed persistently decreased activity uptake in the temporal lobe, whereas a normal pattern was present in others, and one study reported transient hyperperfusion in the left medial temporal lobe.⁵

The pathogenesis of the functional changes leading to transient global amnesia remains controversial. All SPECT studies performed during it have shown hypoperfusion in memory relevant brain structures, suggesting a primary diminution of rCBF due to functional vascular changes or a secondary diminution due to decreased neuronal activity. Our present report, however, shows a transient global amnesia accompanied by increased perfusion of the right medial temporal lobe, suggesting increased neuronal activity in this region.

The discrepancy between the results of the functional imaging studies may be due to different timing of the SPECT examinations in respect to the chronology of each individual attack. On the other hand, transient global amnesia presumably represents a clinical entity with variable pathogenesis.¹ Dynamic changes in rCBF have been measured during migraine attacks, showing a significant increase during headache. An increased rCBF was also found in epileptic foci during complex partial seizures. Amnesia may be a symptom of complex partial seizures and up to 7% of patients with transient global amnesia develop epilepsy

within less than a year. Patients with transient global amnesia who go on to develop epilepsy, however, typically have recurrent attacks of less than one hour in duration.¹ Pure amnesic seizures, furthermore, are usually accompanied by additional types of seizures and features such as automatisms, altered consciousness, and behavioural abnormalities. In our patient no such features were found during an attack that lasted 20 hours, and she never had epileptic seizures. Due to this and as she reported a history of headache, we suggest that the hyperperfusion seen during this transient global amnesia attack might be due to dynamic phenomena similar to those seen in migraine attacks.

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Prevalence of serum antibodies to hepatitis C virus is not increased in patients with multiple sclerosis

In multiple sclerosis there is a significant association with other autoimmune diseases and autoantibodies.¹ Hepatitis C virus (HCV) has been associated with immunological disorders such as Hashimoto's thyroiditis, uveitis, type II cryoglobulinaemia, and membranoproliferative glomerulonephritis.²

We tested serum from 249 consecutive patients with definite multiple sclerosis seen at our clinic in 1992 and 1993 for HCV antibodies.

We used two third generation enzyme linked immunosorbent assays (ELISAs; Monolisa, anti-HCV Pasteur and anti-HCV, Abbott). Samples yielding positive results on one or both of the tests were retested with a second generation recombinant immunoblot assay (RIBA II, Orthodiagnostic systems, Chiron, USA). One patient whose result remained undetermined was positive on a third generation recombinant immunoblot assay and on polymerase chain reaction. As controls 107 members of the patients' families were tested.

Eight patients with multiple sclerosis (3.2%) and six controls (5.6%) had a positive result on ELISA. Two patients with secondary progressive multiple sclerosis (0.8%)

Hippocampal slices. (A) First SPECT study during the transient global amnesia attack, disclosing a focus of increased activity uptake in the posterior caudal region of the right medial temporal lobe (arrow). (B) Second SPECT study three weeks after transient global amnesia, disclosing slightly decreased activity uptake in the region of the previously demonstrated increased uptake (arrow) and, in addition, decreased activity uptake in the region of the left temporal lobe pole (arrowhead). (C) Third SPECT study three months after transient global amnesia, showing symmetric activity uptake.

and one control (0.9%) had a positive result on immunoblot assay (NS between groups). Positive controls and positive patients with multiple sclerosis were not related.

How our patients have been contaminated is uncertain. They had normal liver tests. They had not received blood transfusions, they were not intravenous drug users, or healthcare workers, and they did not have tattoos. Thus we could not determine if the suspected period of contamination by the virus followed or preceded the onset of multiple sclerosis.

In conclusion, the prevalence of anti HCV antibodies is not higher in our population of patients with multiple sclerosis than in our controls or in the general population in France³ (0.8% v 0.29% in blood donors and 0.87% in organ donors), and our results do not support a role for hepatitis C virus in the pathogenesis of multiple sclerosis.

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MATTERS ARISING

Epidemiology of schizophrenia

Cannon and Jones are to be congratulated for their thorough and scholarly review on the epidemiology of schizophrenia.¹ However, they do not do justice to the literature on immigrant groups and in particular that concerning "British Asians"—that is, migrants to Britain from the Indian subcontinent and their descendants.

They quote one study² in support of their assertion that an increased incidence of schizophrenia has been reported from this group. However, this study is concentrated in one particular district of London; British Asians have settled in many districts of London and in most provincial cities, now constitute about half of all ethnic minorities (1.5 million people in the 1991 census), and represent a multiplicity of cultures, languages, and countries of origin. The quoted study concerns only one small fraction of this population and involves only a very few Asian cases. It has been the subject of a considerable body of criticism in subsequent issues of the *BMJ*.

Given the heterogeneity of this population and the limitations of much of the research,

firm conclusions on the subject are some way off. But a review of schizophrenia in British Asians concludes that studies carried out soon after the migration showed an excess, whereas more recent surveys have indicated a rate close to or below that of the rest of the population. The early excess can probably be put down to faulty diagnosis, given the absence of effective transcultural training for British psychiatrists in the decade or so after the first migrations (personal observations). This situation should be viewed in the context of better mental health generally for this group.³

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NOTICE

Announcement from the British Neuropsychiatry Association

The Winter meeting of The British Neuropsychiatry Association will be held on 17 January 1997 at the London Zoo.

Topic: "memory"

Morning sessions: theoretical/experimental aspects of memory

Afternoon session: clinical controversies of memory

For further details of this meeting please contact: Suzanne Miller, 44 Roan Street, London SE10 9JT. Telephone: 0181 858 2699, Fax: 0181 853 4416, e-mail 100023.2012@compuserve.com

For details of membership of the BNPA which is open to medical practitioners in psychiatry, neurology and related clinical neurosciences, please contact: Dr Jonathan Bird, Secretary BNPA, Burden Neurological Hospital, Stoke Lane, Stapleton, Bristol, BS16 1QT. Telephone 01179 701212 ext 2925/2929.

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.

Giant Intracranial Aneurysms. Edited by ISSAM A AWAD and DANIEL L BARROW. (Pp 299; price \$95.00.) 1995. Published by The American Association of Neurological Surgeons, Illinois. ISBN 1-879284-22-7.

During the early period of my training in neurosurgery, it was the received view that giant aneurysms infrequently bled and that their problems, if any, were due to their mass compressive effects rather than to haemorrhagic or haemodynamic effects. That this view held sway for so long is perhaps because these lesions are rare and an appreciation of their epidemiology and natural history can only be determined by the aggregation of information from many different sources. However, it has gradually become clear that these lesions are much more sinister than our earlier complacent view had led us to believe. They have a significant risk of haemorrhage, have serious compressive neurological sequelae, and may also present with thromboembolic complications. Most patients do not survive for more than a few years, usually dying from either haemorrhage or brain ischaemia. Similarly because of their relative infrequency, it is often difficult for individual surgeons to develop the necessary expertise in dealing with these formidable lesions. The neurosurgeon with an "average" general neurosurgical practice may thus be presented with considerable problems in deciding the best management options. In the initial flurry of enthusiasm which greeted the beginnings of endovascular treatment it was felt that this may perhaps represent the best answer for these lesions, but the results have on the whole been disappointing and in many cases it has not proved possible to deal with them by indirect means. Surgical repair remains an important part of the armamentarium. This volume sets down in considerable detail the current "best practice" for the management of giant intracranial aneurysms. For British readers the "inspirational dedication" may be rather hard to take but the individual papers contained within the volume are useful and are usually clearly illustrated with angiograms, MRI, and diagrammatic representations. The book represents a useful addition to the volumes already produced by the American Association of Neurological Surgeons. The AANS publications committee are to be congratulated on the generally high standard of presentation, illustration, and content of their publications. The book is highly recommended to those who have an interest in intracranial vascular disease.

DAVID HARDY

Magnetic Resonance of Myelin, Myelination, and Myelin Disorders. 2nd Edition. Edited by MS VAN DER KNAAP and J VALK. (Pp 558; price DM398.00.) 1995. Published by Springer-Verlag, Heidelberg. ISBN 3-540-59277-6.

Magnetic Resonance of Myelin, Myelination, and Myelin Disorders is a really useful book. In this second edition, van der Knaap and Valk have collated information on all the central nervous system disorders which affect myelin; so, this is definitely not just a book on multiple sclerosis. The 64 main chapters (some no more than a couple of paragraphs) describe individual conditions, syndromes and groups of white matter disorders.