women (59·8%), mean age 74·9 (range 65–101) years.[5] Prevalent cases of Parkinson's disease were ascertained in this population during 1988 (time 0) with a two phase design (home interview then neurological evaluation), as described elsewhere.[6] The diagnosis of Parkinson's disease was reached on the basis of the presence of at least two cardinal signs—typical history and drug response—and exclusion of other conditions such as drug induced parkinsonism, parkinsonism associated with cerebrovascular disease, and parkinsonism associated with other neurodegenerative diseases.[6] The population was then studied again, five years later during 1993 (time 5), with the same procedure. The proportion of prevalent cases of Parkinson's disease deceased at time 5 was compared with that of subjects without Parkinson's disease (χ^2 test).

To take into account the different age structure between the Parkinson's disease group and the group without the disease, we calculated the standardised mortality ratio (SMR). The SMR compares the observed number of deaths in the Parkinson's disease cohort with an expected number obtained by applying the age specific SMRs to the Parkinson's disease cohort age structure. In addition, we calculated the annual mortality for age groups 65–74, 75–84, and 85 and over, in prevalent cases of Parkinson's disease and subjects without Parkinson's disease, by dividing the number of deaths by the number of person-years in each age group.

The number of prevalent cases of Parkinson's disease at time 0 was 24 (2768 for the subjects without the disease).[6] The mean age of cases of prevalent Parkinson's disease was 78·54 years (SD 6·73) and 74·92 (SD 6·98) for the non-Parkinson's disease population. At time 5, 16 cases of Parkinson's disease (67%) and 605 subjects without the disease (22%) were deceased. The mean age at death of patients with Parkinson's disease was 82·1 years (SD 7·63) and 82·3 (SD 7·44) for the population without the disease. The difference in number of deaths was highly significant (χ^2 = 27, P < 0·0001) and the SMR was 3·43 (95% confidence interval (95% CI) 1·96–5·58). An excess of deaths in cases of Parkinson's disease occurred in each age group (table).

These results provide further evidence of increased mortality due to Parkinson's disease compared with the general population by studying an unselected population based cohort. The risk of mortality due to Parkinson's disease was about three times that of the population without the disease and was not explained by an effect of age. Our results are similar to those of Ebmeier *et al*[3] and Ben Shlomo and Marmot,[4] and provide further evidence of a greater than twofold increased risk of mortality for patients with Parkinson's disease. However, our results concern subjects aged 65 and over and living at home, thus omitting younger subjects and those living in institutions. A follow up study of subjects living in institutions is being undertaken.

Annual mortality (%) calculated for each age group for prevalent cases of Parkinson's disease and other subjects of the cohort without Parkinson's disease (controls)

	Age group (y)		
	65–74	75–84	> 85
Parkinson's disease (n = 24)	25	12·5	29·4
Controls (n = 2768)	2·5	3·6	10·7

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Severe tick borne encephalomyelitis after tick bite and passive immunisation

Tick borne encephalitis is a flavivirus infection which is transmitted by ticks and only rarely results in severe neurological deficits in patients in central or northern Europe.[1] The necessity of passive immunisation after a single tick bite remains controversial as a protective effect can only be achieved in 60% of patients.[2] We report a 32 year old patient who developed serious tick borne encephalitis after a single tick bite, despite passive immunisation.

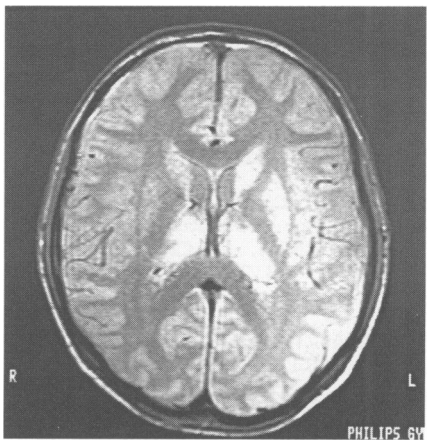
A previously healthy 32 year old man was bitten by a tick during a vacation trip in southern Germany. He was passively immunised with 9 ml (0·9–1·53 g) of tick borne encephalitis immunoglobulin given intramuscularly 24 hours later. Twelve days later, he complained of high fever and severe headache. On admission to hospital, he presented moderate meningeal rigidity and moderate flaccid paresis of both arms. One week later he became comatose for six weeks. Complete flaccid tetraparesis with diminished tendon reflexes and a slight facial palsy were noted. In addition, he had severe facial myoclonic fits with pronounced hypersalivation. During a 15 month follow up period the initial locked in-like state ameliorated moderately. Finally, he was able to speak single words. Comprehension seemed to be only slightly affected. Tetraplegia persisted but there were minimal movements in the left arm. Pronounced generalised atrophy developed, and later on, spasticity on the right side. The sensory pathway seemed to be undamaged.

Examination of the CSF initially disclosed a pleocytosis of 380 lymphocytes, which became normal within eight weeks. Later on, several oligoclonal bands and a temporary increase in protein to a maximum of 8 g/l were seen. The diagnosis of tick borne encephalitis was verified with titre ratios of tick borne encephalitis specific IgM/IgG in serum and CSF. Infections with *Borrelia burgdorferi* were excluded. An EEG showed severe and generalised slowing, which recovered over time. Electromyography showed severe generalised spontaneous activity.

Magnetic resonance imaging disclosed bilateral hyperintense signals with accentuation in the thalamic area on T2 and proton weighted analysis 10 days after onset of the illness (figure). Further disseminated lesions were present in the left striatum, insular cortex, tegmental mesencephalic area, raphe nuclei of the pons, and left inferior olive. Slight haemorrhagic infarction of the thalamus and striatum and discrete gadolinium enhancement were seen on MRI after 12 weeks. Three months later, MRI showed clear regression of the lesions, and a pronounced cerebellar and moderate cerebral atrophy, especially in the frontotemporal lobes.

Tick borne encephalitis is a rare disease which comes about after flaviviruses are transmitted via tick bites. Around 60–70% of tick borne encephalitis infections go unnoticed. Influenza-like symptoms appear in 20–30% of patients after a latency phase of several days. Only 10% of patients have neurological symptoms. Residual deficits are found in up to 10% of the victims and about 1% die. In a few necropsy reports on fatal tick borne encephalitis, polioencephalomyelitis with a prominent thalamic involvement has been recorded.[3 4] Other affected sites have been found in the brain stem (especially the tegmentum, red nucleus, substantia nigra, and inferior olive), the cerebellum, the basal ganglia, and the anterior horns of the cervical and thoracic spinal cord. The polioencephalitic lesions, detected on MRI in our patient, corresponded well with the known pathological accounts. Similar MRI abnormaliites have been seen in the flavivirus induced Japanese encephalitis,[5] but have not been reported in tick borne encephalitis as far as we know. The mostly flaccid tetraparesis seems to result from anterior horn affliction, as was suggested by electrophysiological examination.

There is no known specific treatment. For prophylactic purposes an inactivated vaccine can be used for active immunisation, which provides adequate protection in 97% of patients. Adverse side effects, such as myeloencephalitis and focal neuritis, have been described after active immunisation.[6] Passive immunisation with a tick borne



Proton weighted MRI with hyperintense signals in both thalami and in the striatum five weeks after the onset of the disease.

encephalitis immunoglobulin is given either before or within four days after the tick bite.[2] Immunoglobulin given after more than four days has been shown to delay the antibody response by means of feedback inhibition and worsens the clinical course, especially in children.[7] We cannot offer a definite explanation for the severe course of disease in our patient. It may be the unfortunate combination of a failure to react to the immunoglobulins and the development of a severe course of disease after a single exposure. It may also be possible that the patient was unknowingly exposed to additional tick bites days before the passive immunisation, thereby making it useless.

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NOTICE

Announcement from the British Neuropsychiatry Association: 1996 summer meeting

The 1996 Summer meeting will be held on 14–16 July at Robinson College, Cambridge. It will include topics on neurodevelopment, language, and the presentation of short scientific papers and single case videos by members. The Association's AGM will be held on 16 July.

For further details of these meetings please contact: Sue Garratt, Administrative Assistant, BNPA, 17 Clocktower Mews, London N1 7BB. Telephone/Fax: 0171 226 5949.

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 or Sue Garratt at the address given above.

CORRECTION

Migraine *J Neurol Neurosurg Psychiatry* 1996;**60**:338;1996;**60**:448. These two listed publications were inadvertently not attributed. They were written by Dr E M R Critchley, affiliation as given in the third in the series, this volume (1996;**60**:584).

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.

Motor Neuron Disease. Biology and Management. Edited by PN LEIGH and M SWASH. (Pp 468). Published by Springer-Verlag, London 1995. ISBN 3-540-19685-4/0-387-19685-4.

"Inevitably the disease progresses, but one must never give in too quickly, neither the sufferer nor the helper, who must always be quick with encouragement since success breeds success" (p454). So writes the wife of a patient with motor neuron disease (MND) at the end of this book dedicated to his disease. A moving account that not only relays what the disease means at the personal level to both the patient and family but also serves as an encouragement to those involved in the research and management of this most feared of neurological diseases. This fear for many years reflected our ignorance, but MND is now currently yielding some of its secrets with the advent of modern molecular genetics and families of neurotrophic factors. This book therefore appears at an appropriate time.

This book primarily concentrates on the pathology, pathogenesis and treatment of MND, and covers the ground well, if somewhat repetitively at times—for example, inclusion body pathology is discussed in chapters 4, 5 and 7 at least! However, in a field that is currently moving at speed, the book can clearly be seen to be dated, irrespective of the obvious comments in the text (for example, p230 "Since this chapter was first submitted for publication in 1989, . . ."). It is therefore not surprising that some topics are already in need of revision including: discussion of the SMA gene; the role of glial cell-line derived neurotrophic factor (GDNF) in motor neuron survival; the results of clinical trials using neurotrophic factors in MND; the significance of anti-GM1 antibodies in MND and motor neuropathies and the future of riluzole therapy, to name but some recent developments.

The updating of chapters with some of this new information has been tackled by some authors, by the tagging on of relevant information. This sadly fails to work in the majority of cases as the overall discussion of the chapter does not necessarily fit naturally with the new points raised by recent research. A better approach to try and tackle this delay in conception of the book to publication may have been to include an epilogue detailing recent developments as well as providing an introduction outlining the developmental history of motor neurons and their organisation into central motor pathways. This latter topic is taken up in chapters 4 and 13, but an account earlier on would have put discussion in later chapters of the book into a clearer context. Furthermore chapter 13 on the somatic motor neurons and descending motor pathways (a 72 page chapter), seemed out of place in its discussion and attention to neuroanatomical detail in a book that has as its main topic a diffuse neurodegenerative process. Indeed, the individual biases of authors is always difficult to accommodate in multi author books, so, for example, in the chapter on theories of causation, Appel *et al* emphasise the evidence for an auto immune basis to MND. This in itself is not a bad thing but does rather detract from other possible pathogenic processes, and may unduly distort the field to the reader who simply reads this chapter in isolation.

Overall the book represents an impressive body of work relating to MND, but chapters on the cognitive deficits in this condition and the role of anti-GM1 antibodies in distinguishing MND and multifocal motor neuropathy with conduction block would have been welcome. However, the chapters are extremely well referenced, and issues are dealt with that are often skipped over by books of this type—for example, the chapters on the management of MND and the concluding chapter from the spouse of a patient with MND being notable examples. It is therefore a book which serves to summarise a complex and evolving field, and although that summary is somewhat dated it is not without relevance and importance to the neurologist's current management of this disease.

ROGER BARKER

Clinical Neurology. Third Edition. Edited by MICHAEL J AMINOFF, DAVID A GREENBERG and ROGER P SIMON. (Pp 344; $34.95). Published by Appleton & Lange, Connecticut 1995. ISBN 0-8385-1383-2.

This text book has 344 pages of fairly small print, which is well laid out and beautifully illustrated. It also contains a large amount of information in tabulated form. The material is set out in 12 chapters covering the common neurological syndromes and investigations.

This book is comprehensive in its approach. This may appeal to some of its intended readers . . . medical students, house officers and non-neurologist practitioners. For others, its detailed, all-embracing comprehensiveness may impede comprehension. In general this group of readers requires more help in the identification of common, important, day-to-day neurology, from the large mass of rarer conditions which are the responsibility of the neurological specialist. The textbook may attract young neurologists in the early stages of their training.

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