ronds are also present. It also implies that the 5-HT terminal is functional enough to mediate decreased 5-HT tone. This interpretation is supported by the finding of reduced CSF 5-HT-3 IA in some patients.

The second observation was that buspirone did not exacerbate seizures unrelated to myoclonus in progressive myoclonic epilepsy. Decreased exacerbation on myoclonus may precipitate myoclonus-associated seizures, perhaps as in our case 2. This supports the clinical observation that myoclonus and epilepsy respond differently to drugs in progressive myoclonic epilepsy and the hypothesis that they have different regulatory mechanisms. The 5-HT₅A agonist 8-OH-DPAT has not been found to be an anticonvulsant in standard experimental models of epilepsy. Anticonvulsants could have masked a proconvulsant effect of buspirone, however, 8-OH-DPAT has a proconvulsant effect in mice and extremely high doses induce seizures in rats which may be unrelated to 5-HT neurotransmission.

It would be premature to conclude that drugs acting at 5-HT₅A receptors are ineffective in progressive myoclonic epilepsy. A post-synaptically acting 5-HT₅A agonist might have a benefit on myoclonus from buspirone. Different partial 5-HT₅A agonists, such as gepirone or ipsapirone without buspirone’s weak D₂ dopamine receptor antagonism, may also have a different effect on myoclonus. Continuous treatment with buspirone has effects on other neurotransmitter receptors such as 5-HT, receptors which could have influenced myoclonus. Pre- or post-synaptic 5-HT₅A antagonists are other therapeutic possibilities.

Further studies on the role of 5-HT receptor subtype involvement in myoclonus are indicated, particularly in Lance-Adams syndrome (post-hypoxic myoclonus) for which evidence is best for involvement of 5-HT systems.

This work was supported in part by FDA Orphan Products and Development grant F1-C00-0074-01-1 and the Children’s Research Institute.

M R PRANZATELLI
Departments of Neurology, Paediatrics, Pharmacology, The George Washington University, Washington, DC

D PRANZ
Departments of Neurology and Paediatrics, Wright State University School of Medicine, Dayton, OH

E TATE
J M MARTENS
Department of Neurology, Children’s National Medical Center, Washington DC, USA

Correspondence to: Dr Pranzatelli, Neurology Department, Children’s National Medical Center, 111 Michigan Avenue NW, Washington DC, 20010, USA


———

MATTERS ARISING

Balint’s syndrome in subacute HIV encephalitis

I was interested to read the report of Dr Schneider et al on a 45 year old woman with Balint’s syndrome complicating subacute HIV encephalitis.1 Attribution her cognitive disorder to subacute HIV encephalitis in the absence of biopsy confirmation is presumptuous and likely to be incorrect. Focal neuro- logical findings are distinctively unusual with this disorder. It is far more likely that progressive multifocal leukoencephalopathy (PML) was responsible. PML affects approximately 4% of all AIDS patients.2 A predilec- tion for the parieto-occipital region is typical and visual field defects are a prominent manifesta- tion in 35% of patients.3 The radiographic characteristics of the white matter lesions in PML4 mirror those observed in their patient. Furthermore, the improvement with zidovudine hardly dispels the diagnosis of PML. Spontaneous recovery5 and improvement following the use of zidovu- dine6 have both been reported with HIV- associated PML.

JOSPEH R BERGER
THOMAS E WHIGHAM
University of Miami, School of Medicine, Department of Neurology (DD-5), PO Box 01860, Miami, Florida 33101, USA


Schneider et al reply:

We appreciate the comments by Dr Berger regarding the histological nature of the lesions in our patient who presented with Balint’s syndrome as the first neurological manifestation of AIDS.1 Unlike some patients with Balint’s syndrome due to stroke in whom visual movement perception is impaired,2 she perceived movement particu- larly well. We ascribed this variant to a subcortical lesion site, as shown by MRI, that spares cortico-cortical connections between primary visual cortex and visual association areas. Both subacute HIV encephalitis and progressive multifocal leukoencephalopathy (PML) primarily involve subcortical white matter and would explain the findings in our patient. The differential diagnosis was not elaborated in our article as it was not the primary objective. We favoured the former diagnosis because the MRI showed bilateral, extended, confluent lesions on T2-weighted images appeared more typical of subacute encephalitis and because of the response to zidovudine treatment.3 In the absence of a biopsy or necropsy, the nature of a patient’s lesions remains conjectural and we agree with Dr Berger that PML is a serious consideration in this patient.

M. SCHNEIDER
THEODOR LANDIS
MARIANNE REGARD
Department of Neurology, University Hospital, CH-8091 Zurich, Switzerland
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 allow for additional 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.


This large, and at first sight intimidating, book will be a source of fascination and pleasure to anyone interested in the localisation of function within the cerebral hemispheres. Two thirds of the cases reported in this volume were laid by the early work of Penfield and Jasper and their colleagues in Montreal. The main theme is to examine those correlations between the site of origin of epileptic seizures and the pathways of spread that result in the complex symptomatology of human partial seizures of frontal origin.

The clinical importance of frontal lobe seizures cannot be underestimated. They are common and frequently resistant to treatment. However, frontal lobe seizures tend not to remain confined to their sites of origin, as is the case with temporal lobe seizures, but to spread rapidly. Indeed, their symptomatology may be more determined by the pathways of spread than by their site of origin. This volume makes it clear that we must abandon the classical idea of frontal lobe seizures resulting in the classical Jacksonian march, or tonic aversion and little else. The speed with which generalisation can occur from a frontal lobe focus means that generalised tonic-clonic seizures or "pseudo absences" are not infrequently seen. Frontal seizures are often associated with immediate loss of consciousness associated with versive posturing or with contraversive head and eye turning without loss of consciousness. Typical complex partial seizures may arise from frontal lobe structures and these can be suspected clinically. They are very frequent, often occurring in clusters, relatively brief and associated with rapid recovery of consciousness without post-ictal confusion. They are often associated with bilateral automatisms at the onset of the seizures. However, more typical complex partial seizures can arise from frontal sites with auras which are more typically associated with temporal lobe seizures.

There is considerable debate as to whether particular electro-clinical seizure types are associated with different sites of origin within the frontal lobe. This reviewer is more persuaded by the difficulties in identifying precise localisational patterns of seizures.

The book runs to over 700 pages and contains no less than 46 chapters. Whilst much of the volume is taken up with frontal lobe epilepsy, there are contributions on the cytoarchitecture and neurophysiology of the frontal lobes in both man and primates. There are discussions into aspects of neurochemistry, like frontal lobe projections from the basal ganglia and discussions of the pharmacological management of the partial epilepsies. One can criticise the repetitive nature of many of the discussions. It will certainly serve as a state of the art review for anyone with anything more than a passing interest in epilepsy.

DAVID CHADWICK


"Pain is one of the prime movers of life" declared François Magendie. Doctors have advanced from this merciless standpoint, but not enough to satisfy the authors of this decidedly mixed volume.

On the one hand, here are definitive accounts of pain-generating mechanisms (though contentious areas such as the role of the cerebral cortex, are avoided) and commonsensical, compassionate descriptions of pain management. But alongside such sound contributions there are ill-conceived and hastily written chapters. Even allowing for constitutional difficulties with minding ones mus, deltas and kappas, getting through a section on opioid pharmacology felt like walking through quick-setting cement, not least because of spectacular typos, e.g. "delirious" (page 699).

Neurological purists will also balk at suggestions that trigeminal neuralgia may be caused by intracerebral (sic) tumours (page 650), that anti-serotonin agents may be classified as adrenergic blockers (page 772), and that diphenylhydantoin and phenytoin are somehow different (page 771).

The most irritating feature of the book is its inclination to accuse the medical profession en bloc of not advancing from the Magendie line. "Doctors fail because of ignorance, inexperience ..." (page 567), "doctors often become frustrated or even angry ..." (page 763). Such pejorative and condescending remarks mirror the very opinions these doctors are supposed to have towards their patients. They are unsubstantiated and, even if true, two wrongs do not make a right. The presence of such comments, along with a tendency to sloganising and attempts at fundraising, strike a note of desperation (in a purportedly scientific test) which must ultimately be counterproductive.

These flaws make it hard to recommend the book to neurologists who will already have access to classic textbooks on pain. Similarly, trainees may find the price a little steep for under 300 pages of plainly produced text with few illustrations.

L GINSBERG
Anti-acetylcholine receptor antibody measurement in myasthenia gravis.

T Brenner, I Wirguin and O Abramsky

J Neurol Neurosurg Psychiatry 1993 56: 115-116
doi: 10.1136/jnnp.56.1.115-b

Updated information and services can be found at:
http://jnnp.bmj.com/content/56/1/115.3.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/