rons 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-HIAA in some patients.

The second observation was that buspirone did not exacerbate seizures unrelated to myoclonus in progressive myoclonus epilepsy. Drug-induced exacerbation of 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 myoclonus epilepsy and the hypothesis that they have different regulatory mechanisms. The 5-HT_{1A} agonist 8-OH-DPAT has not been found to be an anticonvulsant in standard experimental models of epilepsy.[5] Anticonvulsants could have masked a proconvulsant effect of buspirone since anticonvulsants are better able to block seizures than myoclonus in these disorders. There are no data to support a proconvulsant effect of buspirone, however, 8-OH-DPAT has a proconvulsant effect in mice[5] and extremely high doses induce seizures in rats[2] which may be unrelated to 5-HT neurotransmission.

It would be premature to conclude that drugs acting at 5-HT_{1A} receptors are ineffective in progressive myoclonus epilepsy. A post-synaptically acting 5-HT_{1A} agonist might have a different effect on myoclonus from buspirone. Different partial 5-HT_{1A} agonists, such as gepirone or ipsapirone without buspirone's weak D_2 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_2 receptors which could have influenced myoclonus. Pre- or post-synaptic 5-HT_{1A} 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.

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MATTERS ARISING

Balint's syndrome in subacute HIV encephalitis

I was interested to read the report of Dr Schnider *et al* on a 45 year old woman with Balint's syndrome complicating subacute HIV encephalitis.[1] Attributing her cognitive disorder to subacute HIV encephalitis in the absence of biopsy confirmation is presumptuous and likely to be incorrect. Focal neurological findings are distinctly 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 predilection for the parieto-occipital region is typical and visual symptoms are a presented manifestation in 35% of patients.[3] The radiographic characteristics of the white matter lesions in PML[4] mirror those observed in their patient. Furthermore, the improvement with zidovudine hardly dispels the diagnosis of PML. Spontaneous recovery[5] and improvement following the use of zidovudine[6] have both been reported with HIV-associated PML.

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Schnider 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 particularly 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 leucoencephalopathy (PML) primarily involve subcortical white matter[3] 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 appearance with bilateral, extended, confluent lesions on T2-weighted images appeared more typical of subacute encephalitis[4] and because of the response to zidovudine treatment.[5] In the absence of a biopsy or necropsy, the histology of our patient's lesions remains conjectural and we agree with Dr Berger that PML is a serious consideration in this patient.

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Anti-acetylcholine receptor antibody measurement in myasthenia gravis

In a recent study,[1] Clarke *et al* reported a "deficiency of anti-acetylcholine receptor (AChR) antibodies measurement in myasthenia gravis (MG)". In their retrospective study, antibodies were detected in only 38% of 86 patients with MG, compared with 66–93% in other reports. The unusually low antibody detection rate is attributed by the authors to the use of staphylococcal protein-A for immunoprecipitation rather than anti-human IgG antiserum. To support their claim, the authors cite our early report of 36% detection in an assay employing protein-A.[2] In that study, however, we used denervated rat muscle AChR as the antigen. Later, we modified the system using *human amputation muscles AChR* which increased the detection rate to 88%, still using protein-A as the precipitating agent.[3,4] These results agree with most reported series,[5,6] which stress the notion that the assay efficiency (sensitivity and antibody titres) depends primarily on the quality of the antigen preparation. Thus protein-A is similar to anti-human IgG antisera for immunoprecipitation in the anti-AChR antibody assay and we feel that the authors should look for other methodological flaws to account for the low sensitivity of their assay. Finally, we completely agree with the authors that all laboratories engaged in routine antibody assays should be subject to a quality control audit, and we refer the authors to consult with "EuroEQAS for AChR antibodies".

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Clarke et al reply:

We thank Dr Brenner *et al* for their comments on our paper on the lower sensitivity of our anti-acetylcholine receptor antibody assay. We do not claim that this can be solely attributed to the use of staphylocccocal protein A for immunoprecipitation rather than anti-human IgG antiserum. Indeed, we state that the use of receptor preparations from single individuals rather than pooled material may be partly responsible.

Regarding the problem of quality assurance for such antibody assays, we participated in the first EURO EQAS anti-acetylcholine receptor workshop held as part of the Euro-myasthenia III meeting, 1991. The results which our laboratory reported for the circulated samples agreed with the other participating laboratories, which does not suggest current methodological flaws. The assay used remained unchanged from the one described in our paper, with one modification. Owing to problems of availability, the form of protein A had been changed from staphyloccocal dried cells (Sigma S0504) to a more homogeneous protein A cell suspension (Sigma P7155). We are unable to comment if this alone could significantly alter the sensitivity of our assay.

It was apparent from the meeting that standardisation of human muscle antigen preparation is perceived as a problem. One useful suggestion concerned the possibility of utilising tissue culture derived acetylcholine receptor as a reference material for calibration purposes.

Finally, our laboratory has registered to participate in the EuroEQAS for AChR antibodies when this scheme starts on a regular basis.

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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, expiratory date, and your full name.

Frontal Lobe Seizures and Epilepsies. (Advances in Neurology, Vol. 57). Edited by P CHAUVEL, A V DELGADO-ESCUETA, E HALGREN and J BANCAUD (Pp 750; Price: $119.00.) 1992. New York, Raven Press. ISBN 0-88167-827-9.

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. The foundations of the work 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 their 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 understated. 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 also interesting contributions on the cytoarchitecture and neurophysiology of the frontal lobes in both man and primates. There are discursions into aspects of neurochemistry relevant to frontal lobe projections from the basal ganglia and discussions of the pharmacological management of the partial epilepsies. One can criticise the repetitious 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.

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PAIN Mechanisms and Management. (British Medical Bulletin Vol 47, No 3, July 1991). Edited by J C N WELLS and C WOOLF (Pp 791; Price: £33.00). 1992. Edinburgh, Churchill Livingstone. ISBN 0-443-04491-0.

"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, like 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. "deleritous" (page 699).

Neurological purists likewise will 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 sloganizing 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