MATTERS ARISING

Balint's syndrome in subacute HIV encephalitis

I was interested to read the report by Dr Schröder 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 presumably 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 prominent manifestation in 35% of patients.3 The radiographic characteristics of the white matter lesions in PML mirror those observed in their patient. Furthermore, the improvement with zidovudine hardly dispels the diagnosis of PML. Spontaneous recovery4 and improvement following the use of zidovudine5 have both been reported with HIV-associated PML.


Anti-acetylcholine receptor antibody measurement in myasthenia gravis

In a recent study, 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 "elaborate, technical protein-A for immunoprecipitation rather than anti-human IgG antisera. To support their claim, the authors cite our early report of 36% detection in an assay employing protein-A." In that study, however, we treated rat muscle AChR as the antigen. Later, we modified the system using human amputa- tion muscles AChR which increased the detection rate to 88%, still for the same IgG precipitating agent.1,2 These results agree with most reported series,3 which stress the notion that the assay efficiency (sensitivity and antibody titres) depends primarily on the quality of the antigen and the precipitating agent. Thus protein-A is similar to human IgG anti-sera for immunoprecipitation in the anti-AChR antibody assay and we feel that the authors should look for other technical 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 welcome the authors to consult with "EuroEQAS for AChR antibodies".

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1 Clarke CE, Shepherd DJ, Yuli GM, Snaige J, Wilson PB. Deficiencies in anti-acetylcholine...
 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 staphylococcal protein A for immunoprecipitation rather than anti-human IgG antisera. 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 staphylococcal dried cells (Sigma S0504) to a more homogeneous protein A cell suspension (Sigma P7153). 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.

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.


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 bulk of the work reported in this volume was 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 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 extra-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 localisation patterns of seizures.


"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 those of the cerebral cortex, are avoided) and common-sensical, 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 kappa, getting through a section on opioid pharmacology felt like walking through quick-setting cement, not least because of spectacular typos, e.g. "dele-ritous" (page 699).

Neurological purists will likely 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 text) 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.

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