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-HT-NA in some patients. The second observation was that buspirone did not exacerbate seizures unrelated to myoclonus in progressive myoclonic epilepsy. Decreased excitability 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. A post-synaptically acting myoclonus may have a different effect on motor cortex. Different partial 5-HT agonists, such as gepirone or ipsapirone, have been shown to reduce myoclonus in animal and human studies, but they differ in their effects on motor cortex. 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 problems are a prominent manifestation in 35% of patients.3 The radiographic characteristics of the white matter lesions in PML7 mirror those observed in their patient. Furthermore, the improvement with zidovudine hardly dispels the diagnosis of PML. Spontaneous recovery6 and improvement following the use of zidovudine7 have both been reported with HIV-associated PML.

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Antidepressant receptor antibody measurement in myasthenia gravis

In a recent study, Clarke et al reported a "deficiency of antidepressant receptor (AChR) antibodies measurement in myasthenia gravis (MG)."5 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 the biochemical protein A for immunoprecipitation rather than anti-human IgG antisera. To support their claim, the authors cite our early report of 36% detection in a small employing protein A.6 In that study, however, we investigated rat muscle AChR as the antigen. Later, we modified the system using human anti-myasthenia gravis (AChR) which increased the detection rate to 88%, still using protein A as the precipitating agent.7,8 These results agree with most reported series,9 which stress the notion that the assay efficiency (sensitivity and antibody titre) depends primarily on the quality of the antigen. 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 methodical 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 encourage the authors to consult with "EuroEQAS for AChR antibodies".

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