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

D FRANZ
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.

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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.

JOSEPH R BERGER
THOMAS E WHIGHAM
University of Miami, School of Medicine, Department of Neurology (D4–5), PO Box 016960, Miami, Florida 33101, USA

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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 encephalits[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.

ARMIN SCHNIDER
THEODOR LANDIS
MARIANNE REGARD
Department of Neurology, University Hospital, CH-8091 Zürich, Switzerland

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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".

TALMA BRENNER
ITZHAK WIRGUIN
ODED ABRAMSKY
Neuroimmunology Laboratory, Department of Neurology, Hadassah Medical Center, POB 12000, Jerusalem 91120, Israel.

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