conduction studies revealed no abnormalities in the face, upper or lower extremities. Supramaximal stimulation of the tibial nerve elicited unusual late components in addition to F waves. Normal electrophysiological studies included blink reflex, median nerve somatosensory evoked potentials, brain stem auditory evoked potentials, visual evoked potentials and EEG.

The patient was given clonazepam, valproic acid and trimethylenidihydriodil with no relief of symptoms. Carbamazepine lessened the palatopharyngolaryngeal movement and also the muscle cramps in legs. Electromyographic and palatopharyngolaryngeal involuntary movements seen in our patient can be clinically classified into “palatal myoclonus,” although the movements were not exactly rhythmic and occurred at a slower rate than the usual palatal movements. Needle EMG showed myokymic discharges in the palatal and mylohyoid muscles in synchrony with the movement. These EMG findings differed from those reported in symptomatic “palatal myoclonus” showing rhythmic discharges at a faster rate, although the previous EMG studies on “palatal myoclonus” did not disclose firing patterns or wave forms in detail. Myokymic discharges and muscle cramps in the legs relieved by carbamazepine in the present case may indicate that they arise from neuromuscular hyperexcitability rather than a central nervous system disorder.

Ephaptic transmission resulting from demyelination can cause focal myokymia, but in our patient there was no evidence of organic lesion in the posterior fossa, nor did other diseases suggesting diffuse injury or hyperirritability of the peripheral nerves, for example, in association with toxins, thyrotoxicosis, Guillain-Barre syndrome or polyneuropathy. Facial myokymia, though commonly seen in patients with pontine glioma, multiple sclerosis or Guillain-Barre syndrome, rarely involves the muscles innervated by lower cranial nerves.

Myokymic discharges, also called grouped fasciculations, usually cause vermicular movements, but involvement of the dorsal interosseous muscles may cause tremor-like or flickering movements of the fingers. Myokymic discharges in the palatal muscles could therefore cause movements resembling “palatal myoclonus” or “tremor.” Needle EMG is important to differentiate palatopharyngolaryngeal myokymia from the essential “palatal myoclonus,” which has a slower rate of movement than symptomatic “palatal myoclonus.”

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Table

<table>
<thead>
<tr>
<th>Days on drug</th>
<th>Threshold dose mg/day</th>
<th>Max. dose mg/day</th>
<th>Best dose mg/day</th>
<th>Effect of myoclonus</th>
<th>Side effects</th>
<th>Seizure frequency</th>
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<tbody>
<tr>
<td>Patient Diagnosis</td>
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<td></td>
<td>Subjective Objective</td>
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<tr>
<td>Baltic</td>
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<td>15</td>
<td>60</td>
<td>35</td>
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<td>Sedation</td>
<td>1</td>
<td></td>
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<tr>
<td>Baltiic</td>
<td>18</td>
<td>25</td>
<td>30</td>
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<td>Mood swings</td>
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</tr>
<tr>
<td>Baltic</td>
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</tr>
<tr>
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<tr>
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</table>

Best dose/day may indicate merely least side effects.
Balint's syndrome in subacute HIV encephalitis

I was interested in the report of Dr Schneider et al on a 45 year old woman with Balint's syndrome complicating subacute HIV encephalitis. Authorizing 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. A predilection for the parieto-occipital region is typical and visual symptoms are a prominent manifestation in 35% of patients. 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 recovery and improvement following the use of zidovudine 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 historical nature of the lesions in our patient who presented with Balint's syndrome as the first neurological manifestation of AIDS. Unlike some patients with Balint's syndrome due to stroke in whom visual movement perception is impaired, she perceived movement particularly well. We ascribed this variant to a subcortical lesion site, as shown by MRI, that spares corresponding 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. In the absence of a biopsy or necropsy the possibility 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, 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 the protein-A method.

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 demonstrated that the precipitating agent was actually a rat muscle AChR as the antigen. Later, we modified the system using human amputa- tion muscles AChR which increased the detection to 88%, still using protein-A as the precipitation agent. These results agree with most reported series, 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 antiserum 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 encourage the authors to consult with "EuroEQAS for AChR antibodies".

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1 Clarke CE, Shepherd DI, Yuill GM, Snaite MG, Wilson PB. Deficiencies in anti-acetylcholine
Buspirone in progressive myoclonus epilepsy.

M R Pranzatelli, D Franz, E Tate and J M Martens

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