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 theophylline without any relief of symptoms. Carbamazepine lessened the palatopharyngolaryngeal movement and also the muscle cramps in legs. Electrophysiological recordings when 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 myoclonus. 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 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 problem.

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

**Buspirone in progressive myoclonic epilepsy**

Progressive myoclonus epilepsy is a clinical syndrome with obligate features of myoclonus and epilepsy and variable or inconstant features of dementia and ataxia. The most common is Unverricht-Lundborg disease (Baltic or Mediterranean myoclonus), but other types include Lafora disease and mitochondrial myopathy.

A serotoninergic disturbance is suggested by reduced CSF- HIAA in Baltic myoclonus and the antomyoclonic effect of L-tryptophan plus a monoamine oxidase inhibitor or 5-hydroxy-L-tryptophan in some patients. The problem is that these observations do not point to a specific locus of abnormality in the 5-HT system.

Serotonin (5-HT) receptor pharmacology has advanced rapidly, identifying multiple 5-HT receptors types. Only a few have been studied experimentally. Myokymic discharges and movements in the full 5-HT₃ receptor agonist 8-OH-DPAT induces myoclonus but partial 5-HT₃ receptor agonists such as buspirone do not. This pilot study was intended as a preliminary step in the evaluation of possible role for 5-HT₃ receptor abnormalities in progressive myoclonic epilepsy. The 5-HT₃ receptor is key to the 5-HT-containing raphe neurons on which it is located, because its stimulation decreases cell firing. The activity of these neurons may be especially important in brainstem-mediated myoclonus, but the raphe nuclei also project widely to forebrain and spinal cord. Buspirone (Buspar) is the first clinically used 5-HT₃ agonist of its class, widely prescribed as an anxiolytic. Since anxiety increases myoclonus in our patient population, we also hypothesised that they may benefit from an anxiolytic. Much evidence suggests buspirone exerts its clinical effect by stimulating pre-synaptic 5-HT₃A receptors.

This uncontrolled observational study in a small number of patients suggests that buspirone does not help and may exacerbate myoclonus in progressive myoclonic epilepsy. Worsening of myoclonus was not explained by decreased anticonvulsant levels, and there are no experimental data to support an interaction between buspirone and anticonvulsants. Although a fluctuating baseline of patients with progressive myoclonic epilepsy could give the false impression of drug-induced exacerbation, the patients improved when buspirone was discontinued.

The data should be viewed as positive findings for several reasons. Any response to buspirone suggests that the 5-HT₃ somatodendritic autoreceptors are involved and by inference, that 5-HT₃-containing raphe neurons

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**Table: Effects of buspirone on myoclonus and seizures**

<table>
<thead>
<tr>
<th>Patient Drug use</th>
<th>Diagnosis</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>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Subjective</td>
<td>Seizure frequency</td>
</tr>
<tr>
<td>Baltic</td>
<td>60</td>
<td>60</td>
<td>35</td>
<td>worse</td>
<td>No change</td>
<td>Sedation 1</td>
</tr>
<tr>
<td>Baltics</td>
<td>18</td>
<td>30</td>
<td>20</td>
<td>worse</td>
<td>worse</td>
<td>Sedation 1</td>
</tr>
<tr>
<td>Balsic</td>
<td>3</td>
<td>15</td>
<td>10</td>
<td>worse</td>
<td>worse</td>
<td>Sedation 1</td>
</tr>
<tr>
<td>Best dose/day</td>
<td>60</td>
<td>35</td>
<td></td>
<td>worse</td>
<td>worse</td>
<td>Sedation 1</td>
</tr>
</tbody>
</table>

Best dose/day may indicate merely least side effects.
Matters Arising

Balint's syndrome in subacute HIV encephalitis

I was interested to read the report of Dr. Schneider 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 presuppos- tuous 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 loss is a predominant mani- festation 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 zidovu- dine5 have both been reported with HIV-associated PML.

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Antiacetylcholine receptor antibody measurement in myasthenia gravis

In a recent study, Clarke et al. reported a "deficiency of anti-acetylcholine receptor (AChR) antibodies measurement in myas- thenia gravis (MG)."1 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 non-specific 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.2 In that study, however, we used a pre- pared rat muscle AChR as the antigen. Later, we modified the system using human amputa- tion muscle AChR which increased the detection rate to 88%, still insufficient for the precipitating agent.3,4 These results agree with most reported series,5 which stress the notion that the assay efficiency (sensitivity and antibody titres) depends primarily on the quality of the antigen. The precipitating protein-A is similar to human IgG antisera for immunoprecipitation in the anti- AChR antibody assay and we feel that the authors should look for further technical flaws to account for the low sensitivity of their assay. Finally, we completely agree with the authors that all laboratories engaged in rou- tine antibody assays should be subject to a quality control audit, and suggest to the authors to consult with "EuroEQAS for AChR antibodies".6

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1 Clarke CE, Shepherd DI, Yull GM, Snaile JC, Wilson PB. Deficiencies in anti-acetylcholine...