diagnostic groups are heterogenous, with only six of 23 patients (combined group, with amyotrophic lateral sclerosis, lower motor neuron disease, and Guillain-Barré syndrome) showing raised titres against GM1 and only four of 23 (one of four patients with amyotrophic lateral sclerosis) against AGM1 and GM1. As in our sample, this may reflect heterogeneity of disease processes in motor neuron disease. Also, these findings argue against a specific pathogenetic role of anti-ganglioside antibodies. As was pointed out in our study, there was no association of AM1 antibodies with amyotrophic lateral sclerosis and antibodies to GM1. These data support our earlier findings, that GM1 may not be specific for motor neuron disease or amyotrophic lateral sclerosis (in our sample only four of 35 patients with amyotrophic lateral sclerosis had serum IgM anti-GM1 serum antibodies). Thus to establish a possible pathogenic or diagnostic role for these antibodies, studies with large sample size and tests for reactivity to multiple antigens are needed.  


Three decades of normal pressure hydrocephalus: are we wiser now?  

In his excellent editorial on this subject, Vanneste refers to the place of CSF removal and pressure monitoring tests in the diagnosis of normal pressure hydrocephalus, and their possible predictive role for the success of shunt procedures. With regard to lumbar CSF infusion methods, he regards their invasiveness and the need for technical expertise, as making them unsuitable for widespread clinical use. In the early days of recognition of the syndrome, and well before the advent of modern imaging techniques, we described a simple bedside CSF manometric procedure. This involved CSF pressure readings after withdrawal of fluid into a syringe and its reinsertion after the pressure returned to its previous level. The immediate resulting pressure rise lasted for only a few minutes in cases where normal pressure hydrocephalus was not present, but for a long period in those patients in whom the diagnosis of normal pressure hydrocephalus was considered acceptable. The assumption, in view of the close coincidence between the physiopathological basis of normal pressure hydrocephalus prevalent at the time, was that the test reflected reduced fluid reabsorbance capacity responsible for the syndrome. It would seem now that according to Vanneste’s reference to modern views, the results would have been explained somehow in terms of obstructions, either subarachnoid or intraventricular. The essential predictive value for benefits resulting from shunt diversion was not followed up at that time.  

Incidentally I think that we owe McHugh* credit for the first report describing the clinical normal pressure hydrocephalus syndrome.  

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Post-traumatic syringomyelia  

The excellent study of Squier and Leht* reports that post-traumatic syringes based on clinical examination and spinal CT occur with an incidence of between 1 and 4-5%; based on MRI studies with an incidence of 12-22%. Their own pathological findings in 20 spinal cords examined between two days and 43 years after severe spinal injury yielded four cases (20%) with cysts extending at least two segments from the site of original trauma. The table summarises the positive results.  

<table>
<thead>
<tr>
<th>Time after injury</th>
<th>Level of initial cord lesion</th>
<th>Extent of pathological syrinx</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>6 weeks</td>
<td>T5</td>
<td>T5-T8</td>
<td></td>
</tr>
<tr>
<td>12 weeks</td>
<td>C7</td>
<td>C4-C7</td>
<td></td>
</tr>
<tr>
<td>22 weeks</td>
<td>L2</td>
<td>L1-T12</td>
<td></td>
</tr>
<tr>
<td>34 years</td>
<td>L3</td>
<td>C8-L3</td>
<td></td>
</tr>
</tbody>
</table>

Before we accept the important inferences suggested by these data, we should examine the defining criteria. The authors do not provide clinical data to establish to what extent, if any, their four patients had clinical signs of a syrinx. A mixture of myelomalacia, fibrillary gliosis, and often cystic change is found at the level of injury in all cases after the first few months, and extends locally, rostrally, and/or caudally in relation to the severity and extent of the softening, infarction, and atrophic damage. Any persisting bone fragments or malalignment may increase the size of the cord damage and cyst formation.  

The criterion of a cystic cavity extending for at least two segments is, I suggest, arbitrary. For if we took the defining limit as no longer than three segments, the incidence would be one in 20 (5%), a figure roughly that previously accepted for clinical post-traumatic syringomyelia. This single case report for three years of an injury at the level of C5. It may be that many patients with an extensive syrinx shown at MRI have no clinical signs, and we do not know how long it takes for the MR image of a syrinx to become symptomatic; indeed in some such injuries in middle aged and elderly people it is likely that the syrinx may never cause appreciable symptoms or deterioration of neurological function. The medicolegal implications are self-evident.  

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Verbal fluency in cortical and subcortical dementia  

In a recent issue Rosser and Hodges* have presented some interesting data relating to measures of verbal fluency in patients with Alzheimer type dementia, Huntington’s disease, and progressive supranuclear palsy. Their main finding is unfortunately dubious in view of an inappropriate statistical analysis. 

The effect being sought by Rosser and Hodges* and which they claim to have found, is a differential pattern of performance against measures across different measures of fluency between the different patient groups. To confirm what they were seeking in statistical other ways, this form of analysis does not demonstration of a significant interaction between the patient groups and type of measure. In their investigation they have employed four separate groups (including a control group, as well as the three patient groups already mentioned) with patients in each of these being tested on three separate fluency measures. This corresponds to what is commonly described as a split plot analysis of variance model having both withing subject and between subject effects. Data obtained in accordance with this model and subjected to an appropriate analysis do enable the computation of a groups x measures interaction. 

By contrast what Rosser and Hodges* report for their statistical analyses are three one way analyses of variance carried out on each of the three measures with these followed by post hoc comparisons of particular pairs of groups. Apart from possibly being unsuitable in other ways, this form of analysis does not permit the computation of interaction terms. It is also possible for the effects described as emerging from these analyses to appear and yet to find that the more appropriate analysis fails to achieve the
Jarisch-Herxheimer reaction in a patient with neurosyphilis: non-convulsive status epilepticus?

In their lesson of the month Zifko et al. described a patient with neurosyphilis who developed fluctuating consciousness, disorientation, restlessness, and fever 10 hours after starting intravenous penicillin G. He had tachycardia and hyperhydrosis. An EEG showed generalised 3/5 rhythmic activity (periodic lateralised epileptiform discharges). These findings were attributed to a Jarisch-Herxheimer reaction.

We suggest that non-convulsive status epilepticus is an alternative explanation. Altered consciousness and prolonged confusion are the central clinical findings of non-convulsive status, which may occur without preceding or accompanying generalised tonic clonic seizures, may resolve spontaneously, and may be associated with the autonomic symptoms of fever and tachycardia. Periodic lateralised epileptiform discharges are one of the EEG correlates of non-convulsive status. As stated by the authors, patients with neurosyphilis are susceptible to epileptic seizures, both due to the condition itself and due to a Jarisch-Herxheimer reaction. In addition, penicillin itself may precipitate seizures. In their patient, non-convulsive status may have been evoked by one of these mechanisms. It would be of value to know whether patients such as this respond to antiepileptic medication.

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Risperidone in Parkinson’s disease

A recent, excellent review of the management of Parkinson’s disease called attention to the atypical neuroleptic drug clozapine (an antagonist of dopamine D2 and serotonin 5HT2 receptors) for amelioration of psychotic symptoms derived from dopaminergic treatments. When temporary withdrawal of antiparkinsonian drugs fails, small doses of the atypical neuroleptic drug risperidone (0.25–1.25 mg/day) can also be used for ameliorating hallucinations induced by levodopa without worsening motor symptoms in Parkinson’s disease. Risperidone has a strong affinity for 5HT2 receptors and only moderate affinity for D2 receptors. For parkinsonian patients in whom the 1–2% risk of agranulocytosis with clozapine is unacceptable, risperidone is another option.

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CORRECTION


During production, the figure was incorrectly printed. The correct version is given here.

T1 weighted axial (A) and sagittal (B) MRI of the patient showing the hypointense cystic lesion in the right thalamus and basal ganglia. Compression of the upper midbrain by the mass is evident in the sagittal scan.