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 antiganglioside antibodies. As was pointed out in our study, there were no characteristic antibody patterns (in 75% of our sample of 35 patients with amyotrophic lateral sclerosis); however, we warn against considering them specific.

There is a similar debate about the role of serum antibodies in motor neuron disease and especially of antibodies to GM1: raised titres of anti-GM1 antibodies are not specific for motor neuron disease, but when there are motor symptoms, they can be lower motor neuron disease type.2-3 Whereas the first studies claimed an association of GM1 antibodies and amyotrophic lateral sclerosis, Willison et al found in their 1993 case-control study no association of 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 pathogenetic 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 patho-physiological basis of normal pressure hydrocephalus prevalent at the time, was that the test reflected reduced fluid reabsorptive 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.

J BRAHAM
Department of Neurology, Chaim Sheba Medical Centre, Tel Hashomer, Tel Aviv University, Israel

Post-traumatic syringomyelia

The excellent study of Squier and Lehtm1 reports that post-traumatic syringes based on clinical examination and spinal CT occur with an incidence of between 1-1 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>
</tr>
</thead>
<tbody>
<tr>
<td>6 weeks</td>
<td>T5</td>
<td>T5-T8</td>
</tr>
<tr>
<td>12 weeks</td>
<td>C7</td>
<td>C4-C7</td>
</tr>
<tr>
<td>26 months</td>
<td>W2</td>
<td>C3-T12</td>
</tr>
<tr>
<td>34 years</td>
<td>L3</td>
<td>C8-L3</td>
</tr>
</tbody>
</table>

Verbal fluency in cortical and subcortical dementia

In a recent issue Rosser and Hodges1 have presented some potentially 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 Hodges1 and which they claim to have found, is a differential pattern of impairment between different measures of fluency between the different patient groups. To confirm what they were seeking in statistical analysis, we ran a range of analyses with the aim of demonstrating 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 within 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 × measures interaction.

By contrast what Rosser and Hodges1 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