Correspondence

Firstly I should like to clarify that all of the 23 patients more than fulfilled the diagnostic criteria for Reye’s syndrome, as used by both the Boston workers and the British Paediatric Association–Public Health Laboratory Service surveillance study. These criteria are more stringent than those used by Corey and colleagues during the 1974 epidemic of Reye’s syndrome in the United States. The one patient who did not have appreciably elevated transaminases did, however, have an increased blood ammonia concentration, a very prolonged prothrombin time, and a compatible clinical history, and the necropsy findings also supported the diagnosis.

Secondly, there is a degree of variation within the diagnostic criteria in clinical presentation, degree of biochemical abnormality, complications, and outcome. Vomiting, for example, is a variable symptom which may be changed by a mother’s persistence in offering feeds or the sick child’s ability to drink. In my experience the consistency of the liver is a more telling clinical sign than hepatomegaly.

Thirdly, only 11 of 23 patients had seizures—two were judged to be in Lovejoy stage II and the rest in stage III or higher. There are two possible reasons for seizures occurring earlier than stage IV or V:

1. In 22 of our 23 patients hypoglycaemia was found and this seems to increase the likelihood of seizures. 
2. Paediatric staff in peripheral hospitals in Northern Ireland are, in general, very familiar with the symptoms of Reye’s syndrome, which makes early diagnosis and, therefore, treatment possible at a stage when recovery is more likely. Patients who have seizures, however, tend to do less well than those who do not have them.

I would dispute, therefore, the suggestion that for some of our 23 patients the diagnostic criteria do not apply. It seems less important, however, that cases of Reye’s syndrome conform strictly to the stages described by Lovejoy and colleagues. Our current practice is to emphasise four key aspects of neurological function which we find to be more useful clinically than the composite staging that can be derived from them. (Table)

Aiyathurai and colleagues have previously described the Singapore syndrome in which children were admitted to hospital with pyrexia, seizures, and features of encephalopathy suggestive of Reye’s syndrome. These children had a much better outcome, however, considering that their treatment was mostly conservative. It is difficult to make a fuller comparison between our experience of the two syndromes as technical constraints have limited the pub-

Clinical features and prognosis of Reye’s syndrome

Sir,

The paper by Glasgow raises questions as to the homogeneity of the 23 children reported as having Reye’s syndrome as the prodromal illness was not biphasic in all; persistent vomiting was not a feature in some; seizures occurred in stages II and III in 14; the liver was not pathologically large in 12; and some did not have appreciable increases in ammonia or transaminases, or both. Thus, it is likely that neither the diagnostic criteria nor the Lovejoy classification (where seizures occur in stage V and less often in stage IV) could have been applicable to some of these children.

Among the 23 children there may be a phenotype we have described as Singapore syndrome, that is a Reye’s syndrome-like illness with early seizures. In our report of 20 children, there was no mortality, and none required ‘prophylactic’ hyperventilation, intracranial pressure monitoring, mannitol, or barbiturate coma. Advocating invasive management of children in stage II Singapore syndrome is therefore questionable.

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Dr Glasgow comments:

I am very grateful for Dr Aiyathurai’s interest in and comments on my recent paper which detailed four years’ experience of Reye’s syndrome in Northern Ireland.

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Table: Central nervous system modified Lovejoy staging

<table>
<thead>
<tr>
<th>Stage</th>
<th>Level of consciousness</th>
<th>Respiration</th>
<th>Response to pain</th>
<th>Pupils</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Drowsy</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>II</td>
<td>Restless/agitad</td>
<td>Rapid</td>
<td>Appropriate</td>
<td>Normal</td>
</tr>
<tr>
<td>III</td>
<td>Light coma</td>
<td>Rapid</td>
<td>Decerebrate</td>
<td>Fixed</td>
</tr>
<tr>
<td>IV</td>
<td>Deeper coma</td>
<td>Variable</td>
<td>Decerebrate</td>
<td>Fixed</td>
</tr>
<tr>
<td>V</td>
<td>Deeper coma</td>
<td>Apnoic</td>
<td>Flaccid</td>
<td>Dilated</td>
</tr>
</tbody>
</table>

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The dual purpose of our paper was to describe and discuss the feasibility of a possible model for use in a district health authority. Our research model is now being adopted as a service procedure in the Exeter District Health Authority. The resource implications are one consultant session per week (in practice this session is being shared by two consultants—half session per week each—who act as alternate chairman for each confidential inquiry), the appointment of a half time health visitor to carry out the home visits, and part time medical secretarial help provided without additional cost by the existing secretariat of the District Medical Officer’s staff. This expenditure is not large for the purposes which can be achieved.