pain but little change in head control. After subsequent injections of 260 mg given over two days, he noted considerable improvement in the ability to drive and carry out other manual tasks in his work. He graded improvement in head control and pain as 50% and 80%, respectively, above baseline, compared with 50% and 50% after the previous BTX injections. He had transient mild tenderness in the injected muscles. After the first month he experienced a slight decline (10%) in function and pain control that remained constant for the next four months and then gradually declined again. With subsequent injections of phenol at about six monthly intervals, he has maintained his maximal level of improvement.

These two patients had moderately severe spasmodic torticollis that had improved only partially after previous treatment. In the first patient, BTX initially provided relief but became ineffective. In the second patient, BTX provided improvement but the side effect of dysphagia was nearly intolerable. Within 18 hours after phenol injections into cervical muscles, there was definite reduction of involuntary movements and pain, with functional improvement. Improvement was greater than after all previous treatments and persisted for six and five months respectively, after the initial series of phenol injections. The only side effect was transitory—namely, mild tenderness in the injected muscles.

In patients who become resistant to repeated injections of BTX, presumably due to formation of antibody to the toxin, it would be of great benefit to have an alternative treatment. Phenol may be of benefit in this situation and has the additional advantage of being inexpensive. If the promising response in these two patients is confirmed in a larger series of patients I am currently studying, EMG guided intramuscular phenol injections may prove to be an effective treatment for some patients with spasmodic torticollis.

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Tuberculous myelopathy: a serial MRI study

In two definitive publications in 1969 Wadia and Dastur delineated spinal meningitis with associated radiculomyelopathy with particular reference to tuberculosis.1 The advent of MRI has meant that the nature of the intramedullary lesions can for the first time be defined during life. Furthermore, the natural history of spinal cord involvement in tuberculosis can be studied with serial MRI. The current study involves a single case followed up with a series of MRI examinations over an eight month period. It shows a previously unpublished pattern of cord involvement.

A 26 year old Samalian male refugee who had been in the United Kingdom for one year presented with a three month history of nausea and vomiting associated with increasingly severe headaches. He had no previous history of or exposure to tuberculosis. On admission to hospital he had neck stiffness only. Initial CSF examination...
three shown been Without the isolated spinal function: patient's MRI showed and produced. That showed clinically delayed myelopathy after chronic meningitis and syringomyelia. More recently MRI has confirmed this link. The myelopathy in these cases is usually delayed by many years and gadolinium enhancement is not seen in the wall of these syrinxes.

Correspondence to: Dr F Schon.

Bickerstaff's brainstem encephalitis associated with cytomegalovirus infection

Cytomegalovirus infection rarely induces encephalitis in a non-immunocompromised host. Bickerstaff's brainstem encephalitis was characterised by the acute onset of external ophthalmoplegia and cerebellar ataxia with CNS signs and symptoms. We describe here the first case of Bickerstaff's brainstem encephalitis associated with cytomegalovirus infection. A previously healthy 17 year old girl developed fever, headache, and general fatigue. Ten days later, drowsiness, diplopia, and walking difficulty developed. She had low grade fever, drowsiness, and neck stiffness. The right eye showed esotropia. External ophthalmoplegia was severe in both lateral directions and moderate in the right upward direction. Tics and reflexes were absent and biceps reflexes were reduced. Finger to nose and heel to knee tests showed dysmetria and decomposition. Romberg's sign was negative. She had a broad based gait and tandem gait was impossible. Muscle strength and sensory function were normal. Blood count, liver function, and cold agglutinins were normal. Serum cytomegalovirus-IgM antibodies were negative and HIV 1 antibody test was absent. Serum IgG anti-GQ1b antibody tests were positive. A lumbar puncture showed a high pressure (300 mm H2O), and high value for cell count, proteins, and IgG, IgA, and anti-cytomegalovirus antibodies (table). Cytomegalovirus DNA was detected in the CSF by the polymerase chain reaction. Brain CT and MRI were normal. Electroencephalography showed irregular slow waves with an enhanced and prolonged response to hyperventilation. Electromyography and sensory conduction studies were normal. The patient was treated with acyclovir (1500 mg/day) for seven days. Clinical symptoms disappeared as follows: low grade fever disappeared on the 17th day of illness, drowsiness and neck stiffness on the 22nd day, ataxia on the 24th day, and the eye movement disorder on the 30th day; deep reflexes returned to normal on the 36th day. On the 30th day of illness, euphoria with pathological laughing appeared; she burst out laughing when our eyes met or we began to talk and this lasted for seven days. Reduction of anticytomegalovirus antibodies and cytomegalovirus DNA by the polymerase chain reaction in the CSF paralleled improvement. The serum anti-GQ1b antibody titre was also decreased. The EEG abnormalities disappeared. Titres of antibodies against herpes simplex virus, varicella-zoster virus, and Epstein-Barr virus in both serum and CSF showed no significant changes.

Although brain MRI and CT showed no abnormalities, the rapidly developed ataxia, drowsiness, ophthalmoplegia without downgaze disturbance, and abnormal EEG findings suggested the presence of a CNS lesion. We therefore diagnosed the patient as having Bickerstaff's brainstem encephalitis. Reduction of the increased CSF cytomegalovirus antibodies and positive cytomegalovirus DNA paralleled the

Clinical data

<table>
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<tr>
<th>Day of illness</th>
<th>21</th>
<th>28</th>
<th>42</th>
<th>56</th>
<th>76</th>
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<tbody>
<tr>
<td>CSF</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cells (×10³/mm³)</td>
<td>63</td>
<td>67</td>
<td>29</td>
<td>24</td>
<td>13</td>
</tr>
<tr>
<td>monocytes (×10³/mm³)</td>
<td>(99)</td>
<td>(68)</td>
<td>(27)</td>
<td>(22)</td>
<td>(11)</td>
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<tr>
<td>protein (mg/dl)</td>
<td>99</td>
<td>76</td>
<td>52</td>
<td>35</td>
<td>27</td>
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<tr>
<td>IgG (mg/dl)</td>
<td>96</td>
<td>80</td>
<td>49</td>
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<td>28</td>
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<tr>
<td>CMV-IgG-FA (normal &lt; 1 &lt; )</td>
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<td></td>
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<tr>
<td>CMV-IgG-ELISA (normal &lt;200)</td>
<td>596</td>
<td>485</td>
<td>304</td>
<td>&lt;200</td>
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<tr>
<td>CMV-DNA-PCR</td>
<td>+</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
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<tr>
<td>Serum</td>
<td></td>
<td></td>
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<tr>
<td>IgG anti-GQ1b antibody (normal &lt;400)</td>
<td>6400</td>
<td>ND</td>
<td>3200</td>
<td>ND</td>
<td>1600</td>
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<tr>
<td>CMV-IgG-ELISA</td>
<td>3870</td>
<td>2700</td>
<td>3320</td>
<td>2270</td>
<td>3370</td>
</tr>
</tbody>
</table>

FA = indirect fluorescent antibody technique; ELISA = enzyme linked immunosorbent assay; ND = not done; PCR = polymerase chain reaction; CMV = cytomegalovirus.

Letters to the Editor
Tuberculous myelopathy: a serial MRI study.

F Schon, G Brown and J Britton

*J Neurol Neurosurg Psychiatry* 1995 58: 259-260
doi: 10.1136/jnnp.58.2.259

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