five patients. The EEG showed excess slow
wave activity, of varied degree, in every case
(table).
An abnormal EEG occurs frequently in
SC. Diffuse slowing of spontaneous
activity is the commonest reported change, with an
incidence of between 55 and 87 per cent.1 Its
occurrence in all five of the patients in this
series may reflect the fact that each exhibited
disturbed behaviour at the time of study.
Behavioural disorders occur frequently in
SC2 and furnish an important diagnostic clue. They may be subtle and overlooked and
distractibility or dysphoria attributed to
chorea per se.
The absence of pathological change on CT
brain scan has been a constant finding to date and
is consistent with the poverty of lesions seen at autopsy.
We are not aware of a published article
documented a series of cases of SC, SEP
recordings or analysis of CSF for oligoclonal
immunoglobulin (Ig) G bands. It is generally
accepted that value for CSF total protein and
white cell count in SC are normal,3 although the
evidence for this is not well documented. Our
findings substantiate this traditional doctrine.
The absence of oligoclonal bands is somewhat
consistent with the result of an apparent selective increase in CSF IgG
reported previously.4 In this case, however, the
evidence for local CSF IgG synthesis was based on an elevated IgG/total protein ratio
where spuriously high values may be found
when, as happened in that patient, there
exists an abnormally high serum IgG. The
detection of oligoclonal bands is an accurate
method for demonstrating intrathecal synth-
thesis of IgG. The negative result in all of our
patients tested argues against a primarily
antibody mediated pathologic immune
reaction within the central nervous system being
responsible.
What role cortical SEP results may have in
the differential diagnosis of chorea has yet to be
determined. Our findings differ from those in a recent report of a single case of SC
in which central sensory conduction time was
prolonged.6 However, this patient exhibited
psychomotor retardation, tonic gaze deviation,
facial weakness, hyperactive deep ten-
don reflexes and the plantar response was extensor, all of which are atypical signs for
SC.3 Normal results in SC stand in contrast to
the situation in Huntington's disease, where
the early cortical components are either
reduced in size or absent.7 This disparity may
be explained by differences in the pathologic
anatomy of the two diseases. Cortical SEPs
also have been reported to reveal abnor-
malities in patients with Wilson's disease,8
whereas results in benign hereditary chorea9
and chorea gravidarum (personal observa-
tions) have been normal.
The results of this study emphasise that
EEG abnormalities and associated behav-
ioral disturbance are frequent con-
comitants to chorea in patients with SC. While clinical features remain the cornerstone
in diagnosing SC, our experience sug-
gests that the stereotyped pattern of results
can allow standard neurodiagnostic tests to
be of some discriminatory value.
We thank Professor P Bartel for scientific
advice, Mr Y Chetty, Dr RA Dupont and
Mrs J Bester provided technical help.
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## Table Results of neurodiagnostic tests

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (y)</th>
<th>Duration of chorea (mo)</th>
<th>EEG</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7</td>
<td>Diffuse excess theta</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>11</td>
<td>Excess theta with posterior dominance</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>Excess theta with right dominance</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>5</td>
<td>Diffuse excess theta</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>12</td>
<td>Defuse delta and excess theta</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CSF</th>
<th>White cells (×10^6/L)</th>
<th>Total # protein (g/L)</th>
<th>Oligoclonal μ IgG bands</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.15</td>
<td>0.10</td>
<td>Neg</td>
</tr>
<tr>
<td>2</td>
<td>0.22</td>
<td>0.22</td>
<td>Neg</td>
</tr>
<tr>
<td>3</td>
<td>0.23</td>
<td>0.23</td>
<td>Neg</td>
</tr>
<tr>
<td>4</td>
<td>0.13</td>
<td>0.13</td>
<td>Neg</td>
</tr>
</tbody>
</table>

# 95 per cent reference range (black adults) 0.10 to 0.50 g/L.
*Microscopically less than 10 per cent.
$μ$ Two or more bands, not present in serum, identification by agar gel electrophoresis with silver stain.
Neg = Negative.

### MATTERS ARISING

**Outpatient referrals**

I was astonished to read that among 7836 successive new outpatient referrals analysed by G D Perkin,1 that "conversion hysteria" with 297 examples in ten years was number six in the top twenty of his diagnoses and constituted 3.8% of the total. Moreover, conversion hysteria was twice as common as either Parkinson's or post traumatic syn-
drome and almost three times as common as depression. In my experience, depression is a very common symptom and presents in many
guises often with somatic symptoms. Conv-
erion hysteria is, by contrast, a very rare
disorder and I do not think that I make this
diagnosis more often than once a year in
the whole of my clinical practice. Does Dr Perkin have special criteria for the diagnosis of
conversion hysteria or is his outpatient
practice biased heavily by referrals from
psychiatric colleagues?

I was also surprised that no diagnosis was made in 26.5% of patients—surely an
unusually high percentage. I should have
thought that 5%, would be nearer the mark.
I recognise that in an outpatient clinic one
may make an inaccurate diagnosis but I do
not think that one should make no diagnosis at all in more than a quarter of the
patients.

E A L NIEMAN,
St Mary’s Hospital, London,
United Kingdom

Dr Perkin replies:
The criteria that I use for the diagnosis of
conversion hysteria are the presence of
physical signs which by their nature can only
be explained on the basis of an elaborated
disability on the part of the patient. I would
disagree that the condition is rare. The cases
that I discussed were seldom, if ever, referred by the psychiatrists. They appear to share Dr
Nieman's view about the scarcity of the
problem. I have analysed some of this data in
greater detail elsewhere, where, I found that
approximately half the patients with features of conversion hysteria had the additional
characteristics of Briquet's syndrome.
That particular syndrome with or without evidence of conversion hysteria has been found
remarkably frequent in those patients who are frequent attenders of medical outpatient
clinics.

I find Dr Nieman's use of the word "should" in the last but one line of his
comment, a curious one. It almost suggests a
compulsive need on the part of the neurolog-
ist to make a diagnosis in his patients. Indeed
that compulsive need, I suspect, often
produces spurious diagnoses which may pos-
sibly help the doctor but do little to assist the
patient. It may be that all patients who
developed giddiness on turning their head have
vertebro-basilar insufficiency. I rather doubt
it and because of this doubt, this would be one
category of patients that I would leave
undiagnosed in many circumstances. If, on

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**References**

1. Gledhill RF. Selective increase in cerebrospinal fluid immunoglobulin G in a patient with
Do leucocytes have a role in the cerebral no-reflow phenomenon?

Aspey et al. failed to demonstrate any difference in either the incidence or severity of the no reflow phenomenon induced by severe cerebral ischaemia in gerbils rendered leucopenic by pretreatment with cyclophamide as compared to controls. The authors have suggested that the absolute number of leucocytes is of limited value in the no reflow phenomenon. We would like to make the following points: first, it may be invalid to compare data derived from humans suffering from focal ischaemia, and probably having coexistent atherosclerotic disease, to that obtained from an experimental model of bilateral middle cerebral arterial hemispheric ischaemia in gerbils with an intact cerebral circulation. Secondly the results do not take into consideration the qualitative role of leucocytes in ischaemia, that is, ischaemic related no reflow phenomenon induced by activation of blood cells and the role of the individual leucocyte subpopulations during ischaemia. Furthermore, comments on the subtype of blood cell responsible for plugging of capillaries are not valid given the method used.

Our own interest in leucocytes and cerebral ischaemia led us to study the rheological behaviour of blood cell subpopulations, that is, red blood cell, granulocyte and mononuclear leucocytes, in 20 males suffering from acute cerebral infarction compared to 20 age-matched healthy controls. The filterability of blood cells, separated using Ficoll-Hypaque density gradient after centrifugation, were determined following the technique of Lennie et al.1 This method allows investigation of blood cell subpopulations under similar conditions and above all, results are unaffected by the number of cells, platelet contamination, or plasma protein interactions. Filterability is expressed as a pressure ratio of cell suspension to buffer after six minutes filtration. Our results suggested that mononuclear leucocyte and granulocyte filterability was impaired in cerebral infarction [7.26 (SD) 2 and 5.75 (SD) 0.87 respectively] compared to controls [5.55 (SD) 1.23 and 4.19 (SD) 0.45], while no differences existed in red blood cells. Furthermore we have obtained similar results in a human model of treadmill-induced, controlled ischaemia in stage II peripheral arterial disease1 and coronary artery disease (unpublished material). These studies provide evidence that leucocytes are functionally altered under ischemic conditions.

The mechanism leading to the altered filterability of leucocytes during ischaemia is not fully understood. It has, however, been suggested that leucocyte activation may occur as a consequence of ischaemia2 and may contribute to the ischaemia by releasing vasoactive substances and mechanically obstructing capillaries.3 Furthermore, activation of the cells impairs their filterability.4

This hypothesis concurs with our results since the filtration procedure measures the ability of cells to pass through the pore-filter, whose diameter of 5 microns approximate the diameter of capillaries. We feel an important issue is the role played by leucocyte activation in the pathogenesis of the no reflow phenomenon. While Aspey et al have studied the role of the absolute leucocyte count, they have not considered leucocyte behaviour. It would be interesting to assess the filterability or the percentage of activated cells in the neuropenic compared to the control group. It may be that only a small number of activated leucocytes are required to contribute to the no reflow phenomenon.

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