tick bites and health problems over the preceding five years. Blood samples were tested for *B. burgdorferi* antibody by indirect ELISA at the microbiology department, Charing Cross Hospital, London. 32 of the 33 workers were men and the age range was 22–63. Of the 33 Nature Conservancy workers 27 recorded tick bites, while all 10 Red Deer Commission workers had been frequently bitten, several over 18–28 years. Symptoms previously described in Lyme disease were reported by 28 workers (85%), the most common being joint pains (21) and rashes (5). 7 (16%) sera were antibody positive, 5 being "weak positive" (20–30 ELISA units). No rashes were recalled in any of the seropositive cases (table)—indeed serological results, symptoms recalled, tick bites, and length of exposure did not seem to be related.

It seems that a significant proportion of workers in occupations carrying a risk of tick bite transmission of Lyme disease in Scotland have antibodies to *B. burgdorferi* but the relation between serology and clinical Lyme disease is unclear. Baseline studies and more detailed epidemiological surveys in high-risk groups are needed to establish an accurate picture, a task that would be enhanced by the more uniform and consistent reporting of cases. Making Lyme disease a non-statutory reportable infection in Scotland is a step in the right direction.4

Clinical Department of Infectious Diseases, N. Hamlet
Ruchill Hospital, Glasgow G32 9SW
Broomgrove Hospital, Inverness
Commonwealth Diseases (Scotland) Unit, D. Nathwani
Ruchill Hospital
E. Walker


LONG-TERM FOLLOW-UP OF IMMUNOMODULATION IN TREATMENT OF HTLV-I-ASSOCIATED MYELOPATHY

Sir,—Last year we reported that plasmapheresis was useful, at least temporarily, in 11 of 18 patients with HTLV-I-associated myelopathy (HAM). During the 1-8-2-5 years after plasmapheresis, 15 of these 18 patients have been treated with other immunomodulatory methods. We report here the long-term follow-up of these patients.

All patients except 1 who had tuberculosis were treated with prednisolone at an initial dose of 30–60 mg daily or on alternate days. Prednisolone was continued for at least 4 weeks at the initial dose and then tapered off. 8 patients had another series of plasmapheresis (four to six sessions, with AP-05H plasma separator or IM-1 350 immunosorbent column).1,4 4 received oral cyclophosphamide 25 mg daily. 2 were treated with a daily intramuscular injection of interferon-alpha (IFN-α) for 4 weeks. We used human lymphoblastoid interferon (Sumitomo Pharma, Osaka). The dose was increased stepwise from 10^4 to 9 x 10^8 units per day. Patient 1 had six sessions of lymphoblastoid interferon (Asahi Medical, Tokyo). Clinical assessment was by disability grade (0–10) and the mean follow-up period from initial diagnosis has been 26 months (range 22–33).

Patients 1–5, who had improved on plasmapheresis by two grades or more tended to respond to other immunomodulatory therapies in the same way (figure). 2 patients treated with IFN-α improved in motor function by two grades. In these patients spontaneous proliferation of peripheral blood lymphocytes, which increase in patients with HAM,2 decreased from 18 731 (3202) to 635 (116) counts per min and from 35 716 (3690) to 10 086 (2037) counts per min, respectively. Even in the 5 responsive patients, however, 4 progressed and the other returned to the pre-treatment level by the end of follow-up. The other 10 patients responded less to these treatments than they had to the initial plasmapheresis. Prednisolone was useful to prevent symptoms from recurring. However, more than 30 mg per day was necessary in all 14 patients.

Cyclophosphamide was beneficial in 1 of the 4 patients but not in the others. Lymphocytapheresis also seemed to be effective, though this was a short-term observation in only 1 patient. Each immunomodulatory therapy resulted in some short-term improvement. In the long-term follow-up, however, no-on improved and 6 were one grade worse off by the end of the study. No severe adverse effect or leukemic change (adult T-cell leukaemia) occurred after treatment.

Hidenori Matsuo
First Department of Internal Medicine, Nagasaki University, Nagasaki
School of Medicine, Nagasaki, 852, Japan
and School of Allied Medical Sciences, Nagasaki University


DIETARY FAT GUIDELINES FOR MEN AND WOMEN

Sir,—Dr Crouse (Feb 11, p 318) argues that the association between low-density lipoprotein (LDL) cholesterol and cardiovascular risk is more evident and consistent in men than in women. Furthermore decreasing fat intake was suggested to lower HDL-cholesterol (a risk factor for both sexes)—to a greater extent in women than in men. He therefore questioned the applicability of dietary guidelines to women. We have found that, relative to a diet high in monounsaturates, a carbohydrate-rich diet lowered HDL-cholesterol more in men (25%) than in women (13%) (p < 0.005). The increase in serum triglycerides on the cholesterol-rich diet was also more pronounced for men (40%) than for women (14%) (p < 0.1). The effects observed were opposite to those suggested by
Sarcosporidiosis Revealed in Sputum

Sir,—In August, 1988, a 31-year-old man, a plumber, born in France and living in a small town north of Paris, had a severe fever with profuse sweating, chest pain, and shortness of breath. He coughed up abundant mucopurulent sputum which, in a Zielh-Neelsen smear, was found to contain many bright-red banana-shaped protozoans (8–10 μm). A cluster, 60–100 μm in size and consisting of a hundred parasites, was observed (figure). The likely diagnosis was infection with schizonts or trophozoites of Sarcocystis sp. The patient refused a chest X-ray and was treated with iodosycin for 5 days.

3 weeks later the infection had gone and the sputum was negative for Sarcocystis, but positive for Candida albicans. These yeasts were also present in the faeces, with cysts of Giardia intestinalis. Haematological indices were normal (erythrocytes 4.8 × 10^12/l, leucocytes 9000/μl [65.5% neutrophils, 1% basophils, 0.5% eosinophils, 27% lymphocytes, 6% monocytes], platelets 258 000/μl) and the 1 h sedimentation rate was 3 mm.

Serodiagnostic tests for toxoplasmosis showed IgG 7.5 IU/ml and no IgM (Renshaw and ISAGA tests) and antibody and/or antigen tests were negative for Aspergillus fumigatus, A flavus, C albicans, Cryptococcus neoformans, and HIV.

PLASMA D-DIMER AND PULMONARY EMBOLISM

Sir,—Bounaumeau et al have suggested that the plasma concentration of D-dimer might be of diagnostic value in suspected pulmonary embolism. However, when perfusion scans were inconclusive, a normal value of D-dimer (< 500 ng/ml) could not be related to the absence of pulmonary thromboembolism. Moreover, because phlebography was not done thromboembolic disease could not be ruled out in 7 patients with high D-dimer levels.

Using the same ELISA method (Bago, Auzière) we have prospectively assayed plasma D-dimer in 29 patients in whom embolism was sought by pulmonary angiography.

At day 0 (date of diagnosis), all the patients had high levels of D-dimer ranging from 910 to 26 000 ng/ml; there was no significant relation between the D-dimer level and the degree of obstruction (Miller’s index). In sharp contrast the D-dimer level was 194 ng/ml in 1 patient in whom pulmonary embolism was excluded after blind examination of the angigrams; this value fell into the normal range defined by the results of 20 healthy volunteers of 130 (5) ng/ml; mean (SD).

During heparin therapy there were no recurrences of pulmonary embolism or extension of deep venous thrombosis; D-dimer levels fell from day 1 to day 8 (table). However, 2 of the 29 patients exhibited a secondary moderate increase (less 1800 ng/ml), coincident in both cases with a voluminous haematoma. In 1 patient who relapsed more than a month after the initial pulmonary embolism despite well-adjusted oral anticoagulant therapy, the D-dimer level was 15 000 ng/ml.

Our study, with angiographic evidence, supports the suggestion of Bounaumeau et al that the D-dimer level might be of diagnostic value.

PROGRESS OF D-DIMER LEVELS

<table>
<thead>
<tr>
<th>Day</th>
<th>Plasma D-Dimer (ng/ml)</th>
</tr>
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<tbody>
<tr>
<td>0</td>
<td>2800 (910–26 000)</td>
</tr>
<tr>
<td>2</td>
<td>1700 (500–7200)</td>
</tr>
<tr>
<td>8</td>
<td>1000 (235–4960)</td>
</tr>
</tbody>
</table>

Normal (n = 20) 140 (55–305)

Results given as median, with ranges in parentheses.