change, or the specific recommendations that the panel "took into consideration" any of this research.

Clearly, policy considerations are becoming central to the debate on diet and disease. The moment of promised government action is almost upon us. The 1984 COMA panel was therefore correct to consider the practical implications of its recommendations. If it failed to do so adequately that is no cause for a retreat back into purely scientific committee work. On the contrary, it is cause for a serious effort to investigate and explicate the policy options for achieving desirable dietary change. Apart from the Bradford project, our research effort on this front is meagre. When the 1984 COMA panel wanted to review the evidence on diet and cardiovascular disease it had over 600 published scientific papers available to it. There is no equivalent body of research on the policy side, and the panel's recommendations suffered from the lack. If we begin now with a national programme of research into nutrition policy we might just be ready to help by the time the next COMA panel prepares its report.

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Lesson of the Week

A narrow escape from splenectomy

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Splenectomy may be performed for diagnostic purposes in lymphoma. Visceral leishmaniasis is well recognised in eastern Mediterranean countries but would rarely be linked with a holiday-maker returning from an area outside the Mediterranean such as the Algarve. We report here on a patient with visceral leishmaniasis who narrowly escaped splenectomy.

Case report

A 53 year old white housewife, resident in Brentwood, Essex, presented in June 1984 with a three month history of progressive malaise, lethargy, and fevers and a weight loss of 9.5 kg (21 lb). In February 1984 she had visited the Algarve for a two week holiday, after which she had suffered a 10 day episode of non-bloody diarrhoea. Her bowel habit had returned to normal, but she had continued to feel unwell.

On examination she was pale and had a swinging fever. There was no lymphadenopathy. She had an ill defined mass arising from the left hypo-

Tropical infections such as visceral leishmaniasis should be suspected in travellers to a wider range of European destinations than Mediterranean countries alone

chondrium, which was thought to be an enlarged spleen. Investigations showed: haemoglobin concentration 9.3 g/dl, mean corpuscular volume 77 fl, white cell count $3.0 \times 10^9/l$ with 64% neutrophils, 26% lymphocytes, and 8% monocytes, platelet count $107 \times 10^9/l$, reticulocytes 20×10^{-3} , erythrocyte sedimentation rate 75 mm in the first hour, direct Coombs' test weakly positive for C3d; her biochemical profile was normal; immunoglobulin electrophoresis showed a mild diffuse increase in IgG and IgM; culture and analysis of midstream urine yielded negative results; repeated blood cultures were sterile; no parasites, cysts, or ova were seen in her stools; and stool culture was negative. Serological tests for brucellosis, infectious mononucleosis, and amoebiasis were negative, and paired sera for antibodies to viruses, including cytomegalovirus, showed no rise in titres. Malarial parasites were not detected. Rheumatoid factor was weakly positive at a titre of 1/40 (RA Latex). Chest radiography showed an unfolded aorta consistent with hypertension. Computed tomography of the abdomen confirmed an enlarged spleen extending to the level of L5 with a normal liver. An initial sternal bone marrow aspirate showed mild iron deficiency alone, and routine cultures yielded no growth of bacteria.

Lymphoma was diagnosed clinically, with hypersplenism accounting for the peripheral blood picture. A second bone marrow aspirate and trephine were taken and laparotomy for diagnostic splenectomy planned. The day before her operation re-examination of the marrow film unexpectedly

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showed Leishman-Donovan inclusion bodies within the macrophages. Culture of the marrow in Evans's medium 6 VI¹ was positive for leishmania promastigotes within three days (fig 1). Laparotomy was therefore cancelled. A course of intravenous sodium stibogluconate (Pentostam) 0.6 g daily for 21 days was started. Her temperature began to settle within 48 hours and had returned completely to normal after seven days' treatment.

On review eight weeks after discharge she was feeling much better and had gained weight. The spleen was enlarged by only 2 cm below the costal margin. Her peripheral blood picture was normal, and bone marrow aspirates were negative for leishmania on both direct microscopy and culture.

FIG 1—Culture of bone marrow at ambient temperature showing promastigotes produced within three days. Giemsa $\times 1000$ (original magnification).

Comment

Kala-azar (black sickness in Hindi) refers to the grey skin colour seen in many cases of visceral leishmaniasis, a disease caused by a protozoon. Fever, weakness, dizziness, headache, loss of weight, and abdominal swelling due to hepatosplenomegaly are the most common symptoms.²

The flagellated promastigotes are transmitted to the vertebrate host by the bite of an infected sandfly. Within the cells of the reticuloendothelial system they develop into rounded amastigotes, or Leishman-Donovan bodies, which can be shown at this stage in the macrophages of the bone marrow, spleen, or lymph nodes. Slight increases in IgG and IgM concentrations are to be expected, and rheumatoid factor was found in 93% of a series of patients with visceral leishmaniasis in Brazil.³ Local distribution is related to the presence of the sandfly vector, *Phlebotomus perniciosus*, and there is an endemic focus in Portugal in the area south of Lisbon. The animal reservoir has been confirmed in dogs and suspected in foxes: isolation of leishmania from one fox has been described.⁴ During 1960-76 as many as 1134 cases of visceral leishmaniasis were reported from Portugal, mainly from the south.²

In our patient isoenzyme characterisation of the promastigotes cultured in vitro (courtesy of Dr D Evans, London School of Hygiene and Tropical Medicine) identified the organisms as *Leishmania-Donovani infantum*, the species causing the visceral disease in the Mediterranean region (fig 2).² Portugal is not, however, a Mediterranean country, its coastline being entirely in the Atlantic.

Splenectomy is not an operation to be undertaken lightly as a recent review confirmed.⁵ Our patient was delighted at her narrow escape, which resulted from careful re-examination of the bone marrow. If, however, treatment fails after adequate courses of chemotherapy splenectomy may be necessary as an adjunct⁶; our patient may have won only a temporary reprieve. Most untreated patients with this disease die within two years.

Leishmaniasis was an unexpected diagnosis. This case emphasises the importance of a travel history and highlights the incidence of tropical infections in travellers to a broader range of European holiday destinations than the confines of the Mediterranean.

FIG 2—Isoenzyme identification of organisms isolated from our patient (P) as *Leishmania-Donovani infantum* (London profile 49). Tracks D, C, and I refer to variations in the *Leishmania-Donovani infantum* complex isoenzyme patterns. Tracks P and I (Eastathion *Leishmania-Donovani infantum*) were identical for all enzymes tested. Shown here are (left) malate dehydrogenase and (right) 6 phosphogluconate dehydrogenase.

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Does ague occur in Britain and if so what insect is the vector for it?

Ague was a term originally used for a chill or fever. In more modern times it has become restricted to the initial fever or relapses of malaria. Ague is now rarely used in medical publications, although it persists colloquially. Malaria was widely prevalent in the British Isles up to about 1850, especially in marshy areas such as the Fen country. After that time the disease regressed in Britain and Europe owing to several factors, including increased availability of quinine and drainage of wet land habitats suitable for the vector species. All human malaria is transmitted by mosquitoes of the genus *Anopheles*, except for the special circumstances of infections acquired transplacentally or through blood transfusions. In Britain *Anopheles atroparvus* was the main vector species, although it is no longer regarded as an active vector.¹ Nevertheless, locally contracted malaria (*Plasmodium vivax*) in Essex and Kent during 1917-9 was transmitted by *A. atroparvus*.² Bruce-Chwatt summarised the World Health Organisation data for cases of malaria imported into Britain.³ The number of such cases peaked in 1979 (2053) but declined by about 20% in 1980, and this trend continued in 1981. Imported cases of malaria might give rise to local foci of transmission in the UK; the 1917-9 outbreaks from introduced malaria noted above serve to demonstrate this possibility. —J C CHUBB, reader in zoology, Liverpool.

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