House late at night. While accident mortality in Great Britain is lower than in Northern Ireland, such a measure would still result in the saving of very many deaths and serious injuries. At a time when the National Health Service is crippled from lack of finance it is very difficult to see any other measure which could so quickly release many resources in hospitals. Mr Rodgers, the Minister for Transport, is well aware of the advantages and is keen to introduce an official Government Bill. If Mr Ennals does not recognise the implications for the Department of Health and Social Security they should be explained to him.

In Northern Ireland surgeons are now playing their part in mobilising understanding among the community at large and among MPs in particular. In Great Britain the public may well be deprived of the benefits of similar legislation for two or three years unless doctors and surgeons give a similar lead. Until we acted we were unaware how widespread and how strong medical opinion was. One would not question the ability of surgeons in Great Britain to persuade the community. The question is whether the will is there.

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Investigation of preclinical iron overload

SIR,—Your recent leading article (12 November, p 1242) drew attention to the inadequacy of present methods for the detection of preclinical iron overload, a condition which should be sought in the relatives of a symptomatic patient with haemochromatosis if affected individuals are to be offered prophylactic treatment. Although they may be abnormal in some cases, the lability of plasma iron and transferrin saturation and the delayed increase in serum ferritin reduce the value of these non-invasive techniques as screening tests.¹⁻⁴

We have recently described a 74-year-old Englishman (case I in the accompanying table) who had thalassaemia minor, developed iron overload in the absence of exogenous iron therapy, and died of a hepatoma.⁵ We have investigated the iron metabolism of three of his children, all of whom have raised liver iron concentrations. One of them (IIC) has normal plasma iron, transferrin saturation, and stainable iron, but his liver iron concentration is high. Another (IID) has a raised transferrin saturation and high liver iron concentration, but normal ferritin and hepatic stainable iron.

These findings suggest that the systematic evaluation of adult family members of patients with iron overload should, as suggested by Feller *et al*,⁴ include a percutaneous liver biopsy with quantitative measurement of liver

iron content. A recent conference on iron metabolism and thalassaemia has accepted the necessity for liver biopsy to evaluate body iron load.⁶

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¹ Wands, J R, *et al*, *New England Journal of Medicine*, 1976, **294**, 302.

² Crosby, W H, *New England Journal of Medicine*, 1976, **294**, 333.

³ Edwards, C Q, *et al*, *New England Journal of Medicine*, 1977, **297**, 7.

⁴ Feller, E R, *New England Journal of Medicine*, 1977, **296**, 1422.

⁵ Parfrey, P S, and Squier, M, *British Medical Journal*. In press.

⁶ *Iron Metabolism and Thalassaemia*, ed D Bergsma, *et al*, p xv. New York, Liss, 1976.

Tourist hepatitis

SIR,—Although a little late, we wish to comment on your leading article on this subject (22 January, p 189) and the subsequent correspondence (30 April, p 1158; 11 June, p 1534). We have evaluated 221 cases of traveller's hepatitis occurring within the Zürich area,¹ of which 80% were in tourists, 7% in businessmen, and the remaining 13% in foreign aid volunteers, other staff abroad, airline personnel, travel agency employees, and alien workers entering Switzerland. HBsAg was found in 53% of the cases presumably contracted in other European countries, the corresponding proportions being 6% for Africa, 12% for the Americas, and 34% for Asia.

The probable places of origin of the hepatitis were compared with the numbers of Swiss immigrants there, as published by the World Tourism Organisation, the Organisation for Economic Corporation and Development, and other sources. This allowed us to extrapolate approximate minimum incidences per entry as follows:

1/50 for overland trekkers to the Middle East;
1/350 for West Africa and Mexico;
1/750 for Algeria, India/Nepal, Indonesia, North-east Africa, Near East (excluding Israel), and South America;
1/850 for Middle East (all travellers);
1/1200 for Morocco and Tunisia;
1/1500 for East Africa, Sri Lanka, Turkey, and Thailand and the rest of the Far East;
1/3500 for Israel and South Africa;
1/9000 for Southern Europe; and
1/150 000 for Northern Europe, Canada, and the USA.

Our rate for Southern Europe is only one third of that reported by Iwarson and Stenqvist.² This may be partly explained by statistical uncertainties within both studies;

possibly differences in the prevalence of hepatitis between Göteborg and Zürich and different national behavioural patterns abroad may have played an additional role.

On the basis of the collected hard data and after consideration of the economic and ethical positions, we decided to recommend that standard immune globulin should be given—if necessary repeatedly—to all trekkers, foreign aid volunteers, and other persons with an adventurous travelling style going to any developing country and furthermore to anybody visiting West Africa or Mexico (for exceptions see below). For all other travellers to areas with an incidence exceeding 1/1500 the duration, style and frequency of travel, eating patterns (oysters, etc), personal risk factors, and indispensability in family and profession ought to be taken into consideration in making the decision. It is our belief that at least for our population such immunoprophylaxis is not indicated for trips within Europe. Nor is it necessary for persons going to areas of the Third World for a very brief duration at an exceptionally high living standard, as is the case for diplomats, airline crews, etc. Prophylaxis with the expensive hepatitis-B hyperimmune globulin must be reserved for special situations.

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¹ Steffen, R, Regli, P, and Grob, P J, *Schweizerische medizinische Wochenschrift*, 1977, **107**, 1300.

² Iwarson, S, and Stenqvist, K, *Scandinavian Journal of Infectious Diseases*, 1976, **8**, 143.

Effects of levodopa on "frontal" signs in Parkinsonism

SIR,—The fortuitous observation in a 74-year-old non-demented woman with Parkinson's disease of symptoms attributable to frontal lesions led us to investigate systematically a further series of 37 patients for the following signs: (1) bilateral grasping reflex; (2) inability to reproduce a series of three gestures; and (3) inability to react to an audiovisual stimulus by a defined opposite gesture (it was previously established that the patients understood and remembered the code). None of these signs could be explained by akinesia, apraxia, or a defect of memory, attention, or watchfulness.

Among the 38 patients, 10 presented one or more of these signs. All the patients were treated with a combination of levodopa with a peripheral decarboxylase inhibitor in usual doses. Under treatment nine patients improved. In some of them there was a parallel improvement in the Parkinsonism and the "frontal" signs. In most patients only one or two of the three signs disappeared—grasping reflex in 7 out of 8, failure of gesture reproduction in 3 out of 7, and failure to produce a "coded answer" in 3 out of 5. Since the patients were examined only at four-week intervals it appears likely that the improvement was due to the drug itself rather than to learning through repeated examinations.

This improvement is in contrast with a lack of effect of levodopa found in several patients with frontal lesions (infarcts, tumours) and similar symptomatology in the absence of Parkinsonism. The explanation is not clear. It is possible that these features can be diagnosed and treated with success only during a limited phase of Parkinson's disease, perhaps in relation to the extension of the neuronal loss. On the other hand in some Parkinsonian

Iron metabolism in family with thalassaemia minor and iron overload

Case	Age	Sex	Plasma iron (μmol/l)	Total iron binding capacity (μmol/l)	Transferritin saturation (%)	Plasma ferritin (μg/l)	Liver iron disposition	Liver iron concentration (μmol/100 mg dry tissue)
I	74	M	37	40	93	3593	4	9.8
IIB	46	F	18	55	33	66	0	2.5
IIC	44	M	20	41	49	125	0	10.2
IID	42	M	39	50	78	100	0	7.9

Mean normal liver iron concentration = 1.4 μmol/100 mg dry tissue; normal plasma ferritin = 15–250 μg/l.

Conversion: SI to traditional units—Iron: 1 μmol/l ≈ 5.6 μg/100 ml; 1 μmol/100 mg ≈ 56 μmol/100 mg.