overseas surgeons and the obligation of the Royal Colleges to those hundreds of men overseas who, having already passed the primary fellowship, will be required to train in Britain before proceeding to the final examination. Their prospects are bleak because of the hard core of men, unlikely ever to become senior registrars, who, by making a career at the registrar grade, are blocking the training posts.

Excellent as the training programme is, it is hard to see how "the scheme could . . . restore the lost confidence and waning enthusiasm about their career prospects . . . of young surgeons . . ." or of many already not so young. The Royal Commission has pin-pointed the need to control the element of maldistribution at its source, but neither the Commission nor your leader-writer has found a remedy for the "anguished doubt" of those already in the queue. "One obvious answer," that "to increase the number of consultant posts . . . would . . . increase the number of training posts" is (happily) welcomed in your article, which is a little confusing since it runs counter to the argument, developed earlier, in favour of "substantially fewer" such posts.

Between the lines of your leading article one may read how much there is to be done: in the implementation of a training programme; in the unblocking of training posts; in the control of maldistribution and, possibly, in the acceptance of a short-term relief programme for the many trained but unsatisfactorily employed surgeons (and physicians) who are a standing reproach to the mismanagement of their elders. Myself one of these, I am, etc.,

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Leukaemia and Reticuloses

SIR,—We have read with interest the letter from Dr. M. Tobias (19 October, p. 190) describing his findings suggesting a possible association between lymphosarcoma and leukaemia.

We should like to record our experience in the last 12 months, October 1967 to September 1968, during which time we have seen 10 new children with lymphosarcoma and 13 with leukaemia. Nine patients came from homes in Liverpool, two from Birkenhead, six from other local authority areas included in the Merseyside conurbation, and six from more distant parts of the Liverpool Region; total population approximately 2½ million.

During 1966, 44 deaths under 15 years of age were attributed to lymphosarcoma and 328 to leukaemia in England and Wales (population 48,075,300), and the corresponding mean annual rates over the preceding five years are 33.8 and 343.8 respectively.¹ From the Liverpool Regional Hospital Board area population we should therefore expect to see two to three new children with lymphosarcoma in any one year, and approximately 12–18 new cases of childhood leukaemia if all such cases should be referred to us. It is apparent therefore that the number of children with lymphosarcoma diagnosed in the last year is four to five times that expected, whereas the number with leukaemia is not excessive. The distribution of these cases in time is interesting in that all the lympho-

sarcomas became known to us during the earlier part of the 12-month period, and the majority of the leukaemias in the latter part.

Like Dr. Tobias our numbers are small, but we must determine whether statistical analysis of our observations would support a small epidemic of lymphosarcoma. Clustering in time and space is usually sought by techniques such as those developed by Knox² and we intend to continue analysing our cases along such lines as was done in the period 1955–64.³ We would value advice from statisticians as to whether other techniques might produce more conclusive results, and also be interested to learn whether other clinicians have noted any apparent excess of lymphosarcoma during the winter of 1967–8. If the occurrence of such an excess does not become apparent until mortality statistics have been compiled, the opportunity for useful field work to investigate common aetiological factors may be lost.

Histologically lymphosarcoma and lymphoblastic leukaemia are probably the same disease, and the clinical label is usually determined by whether haematological abnormalities are detected at onset or later in the course of the disease. It is not unreasonable therefore to suspect and to search for similar preceding factors in the life histories of patients with these diseases.—We are, etc.,

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REFERENCES

- ¹ *General Register Office, 1963–1967, Registrar General's Statistical Reviews of England and Wales, 1961 and yearly to 1966, Part I. Tables, Medical, Table 17. H.M.S.O. London.*
- ² Knox, G., *Brit. J. prev. soc. Med.*, 1963, 17, 121.
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Carcinoma, Smoking, and Rhodesian Africans

SIR,—I was very interested to read Dr. Gerard J. Burke's letter (21 September, p. 742), because his suggestions about the possible causes of carcinoma of the lung in the African in the Bulawayo area closely parallel the conclusions reached by me in a study made when I was district medical officer in Gwanda (a district of Matabeleland, south of Bulawayo).¹

I reported then that the incidence of cancer of the lung in the Gwanda district is only slightly less than that in European males in the U.S.A. in 1950; in the case of the smaller group of mining Africans the incidence is three times that in males in the U.S.A. Nineteen out of 22 lung cancer cases (in the remaining three histories were not available) had worked at some time in mines or were at present employed there, and the inference is that the cancer is an occupational disease associated with mining. The most likely specific cause is thought to be arsenic. Tobacco smoking possibly plays a subsidiary role as a summation factor.

In a subsequent series of 38 proved cases of cancer of the lung which were admitted to Gwanda District Hospital between January 1957 and November 1963 all except one were miners or ex-miners, and 13 of them had palmar hyperkeratoses. Of the 28 patients from whom smoking histories were taken there were eight non-smokers; these eight had mining histories varying from 13 to 33 years, with an average of 20.5 years. The one non-miner out of the group of 38

cancer patients had smoked 24 cigarettes a day for 14 years, which is a high consumption for an African labourer. Only two of the other 20 smokers exceeded eight per day.

This further study of cancer of the lung in south Matabeleland therefore supports the tentative conclusion reached in my earlier analysis that tobacco smoking and some agent connected with mining (probably arsenic) act together as summation factors to cause cancer of the lung in the African male population; although either mining alone (in eight cases in the present series) or tobacco alone (one case) can with sufficiently intense exposure also cause the disease. Professor M. Gelfand (24 August, p. 468) comments on the unlikelihood of atmospheric pollution being a factor in the causation of lung disease in Rhodesia, but the Gwanda cases suggest that the mining environment may substitute very effectively as a carcinogen for the contaminated air of more highly urbanized societies in Europe and America.—I am, etc.,

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REFERENCE

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Malabsorption and the Skin

SIR,—We were surprised to read the letter from Dr. Janet M. Marks and Professor S. Shuster (16 November, p. 455), concluding "with confidence" that the relation between dermatitis herpetiformis and the enteropathy with which it is associated is "indirect." We were equally surprised to read of their "astonishment" at the confusion that has arisen over the relation between these two diseases. Their own publications would appear to have contributed to the confusion; they omitted to include gluten sensitivity among the suggested causes of the enteropathy in dermatitis herpetiformis,¹ and they now dismiss the observation that the skin lesions in dermatitis herpetiformis may improve with a gluten-free diet as a misinterpretation or as due to increased absorption of dapsone. Our observation that in the majority of patients with dermatitis herpetiformis and an enteropathy the enteropathy improves when the patients are fed a gluten-free diet² has been confirmed by other workers⁴ and is now generally accepted.

The main point still leading to confusion is whether or not the skin lesions also improve on gluten withdrawal. We found some improvement in the skin condition in five of the seven patients after six months on a gluten-free diet,³ and there have also been reports of two other patients whose skin improved with this diet.^{5,6} We have now studied six of our patients for a year and one for six months on a gluten-free diet, and also studied all seven three months after re-introducing gluten into the diet. We should like to summarize the findings here, since they confirm that in a proportion of patients the skin disorder is closely influenced by the presence or absence of gluten in the diet.

While taking the diet three patients became free of skin lesions and required no dapsone, and a further three showed a substantial reduction in the amount of dapsone required for control of their skin disease. On reintroduction of gluten all three patients whose skin disease had