Value of Multiple Screening

SIR,—Professor Richard Scott and Dr. P. D. Robertson (15 June, p. 643) describe the organization and methodology of a multiple screening programme in general practice. They make the valid point that within the context of our health care organization the logical place for screening is in, or in conjunction with, general practice. They pose the question as to what is the short-term or long-term effect of screening on the health of the patient. It must be regretted, however, that this question is not given more prominence, nor will the design of their study allow any answer to be obtained.

Numerous examples of screening programmes have been reviewed by Wilson and Jungner.¹ All of these, as well as Scott and Robertson, show that they can find a substantial amount of morbidity. There is no evidence, however, in any of these publications that in terms of subsequent morbidity and mortality the individual shown to have one or more of the conditions screened for has benefited. For many of the conditions for which screening is performed there is still little evidence that methods of treatment used improve prognosis.

At this stage of our knowledge it is of great importance to assess the value of screening examinations by properly controlled trials. Any general practitioner who wishes to embark on a screening programme must be urged to plan his study in such a way that he will be able to evaluate his findings rather than simply present a list of conditions discovered.—We are, etc.,

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REFERENCE

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Prognosis of Lung Cancer

SIR,—Dr. F. A. Nash and others in discussion of their paper on the South London cancer study (22 June, p. 715) mentioned the desirability of having comparative rates of lung cancer survival from a population not radiographed at six-monthly intervals. We have, in fact, published the results from a group which provides a fair comparison.^{1,2} Further support for their thesis is given by the fact that, of all the cases occurring in our series, almost exactly half were diagnosed by the routine annual x-ray and the other half by the occurrence of symptoms, or because of referral in the interim. This supports, in some measure at any rate, the proposed six-monthly interval.

The overall five-year survival rate in our population was 10%, but the five-year survival rate, for the cases diagnosed first at routine radiography, was a little over 17%. The five-year survival rate of those in this group who did not report symptoms was of the order of 24%. While "with symptoms" and "without symptoms" groups are always open to some doubt, the fact that our sur-

vival rate in the cases diagnosed first at routine studies is of the same order as Dr. Nash and others appears to support their findings and their deductions. The case for six-monthly radiography of smokers over the age of 40 seems a strong one indeed. It is true that the prognosis is still not very good, and that much better results could be obtained by a big reduction in cigarette smoking. It remains a fact that this pragmatic approach is worth further evaluation and development alongside health education and other techniques.—I am, etc.,

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A Puzzle

SIR,—We thank Drs. A. Lyell and H. M. Sinclair (29 June, p. 826) for their efforts to try and solve the puzzle posed by the case of adult coeliac disease (15 June, p. 678).

The patient was examined by our dermatologist, Dr. Stephen Gold, who suggested that a skin biopsy might be helpful. The sections made for histological examination showed mild non-specific changes and there was no vascular tissue in the specimen. The vasculitis was not discovered until later when a different biopsy specimen was being examined by histochemical techniques. We were sorry that Dr. Gold could not be present at the Clinicopathological Conference to discuss these findings.

In his letter Dr. H. M. Sinclair (29 June, p. 826) suggests that pyridoxine deficiency might have caused many of the signs and symptoms of our patient. Subclinical deficiency of pyridoxine has been demonstrated in some patients with idiopathic steatorrhoea,¹ and this may be due to impairment of absorption of the vitamin.² Nevertheless, symptoms due to pyridoxine deficiency seem to be rare in adults,³ and we know of only one patient with malabsorption in whom pyridoxine deficiency may have been responsible for clinical changes.⁴ This patient had an anaemia with sideroblasts in the bone marrow which disappeared after treatment. The bone marrow in our patient was always normoblastic and there were never any abnormal sideroblasts.

In man, experimental pyridoxine deficiency may give rise to an irritant seborrhoeic dermatitis with glossitis and stomatitis.⁵ It is difficult to correlate these changes with those observed in our patient. He had no oral lesions, and the rash over his forearms was psoriasiform in appearance, consisting of slightly raised erythematous patches. The skin biopsy, reviewed at the end of the necropsy, showed an acute necrotizing arteritis (as illustrated in Fig. 9, Special Plate, 15 June) which seems difficult to ascribe to pyridoxine deficiency.

Neurological symptoms responding to treatment with pyridoxine have been described in patients taking isoniazid. After an oral dose of 1-tryptophan such patients have been shown to excrete an excess of xanthurenic acid in the urine, apparently due to an interference with the metabolism of pyridoxine.⁶ The neuropathy, however, is different from that seen in our patient. The symptoms and

signs are predominantly sensory and seldom affect the upper limbs.⁷ It is difficult to imagine extensive denervation atrophy of the deltoids, for instance (as illustrated in Fig. 8), arising on the basis of pyridoxine deficiency.

Thus we are unable to relate the signs and symptoms of our patient either to the known effects of pyridoxine deficiency in man or to those described in rats⁸ by Dr. Sinclair. Be that as it may, the "large doses of parenteral vitamins given throughout the patient's illness" included Parentovite, which ensured an intake of 100–200 mg. pyridoxine a week in divided doses. For adults the normal daily requirement of this vitamin appears to be 1–2 mg.² Therefore we cannot accept Dr. Sinclair's ingenious explanation of the unusual course of our patient's illness, and remain puzzled.—We are, etc.,

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SIR,—In the case of adult coeliac disease fully yet inconclusively investigated at the Clinicopathological Conference (15 June, pp. 640 and 678) you quote Dr. F. E. S. Hatfield, the family practitioner, as commenting: "Perhaps we should be using more relevant concepts." Dr. Hatfield then related the stages in the widespread manifestations of a fatal disease—the somatic aspects of which included idiopathic steatorrhoea, progressive neuromyopathy, and septicæmia—to painful incidents in the patient's life, and suggested that "he died because all that he had lived for had somehow come to nothing."

Is it now possible to widen the concept of sensitivity beyond but to include the genetically determined defects of which Professor C. Booth was thinking in his rebuttal of the psychogenic approach to this case? It was postulated (Dr. A. P. Douglas) that a causal relationship (in this case gluten sensitivity) might have created "damage so severe that it was self-perpetuating." May we not postulate a genetically determined emotional sensitivity upon which the environment of the patient's life played to determine such self-perpetuating injuries? The psycho-analytically orientated associate syndromes of intestinal malabsorption with death wishes. If that is justified it must sometimes be possible to exorcize such wishes by somatic therapy. Sometimes, hitherto, not. Our narrow dualistic concepts of today can leave us quite at a loss when the death wish wins.

Here then is the hypothesis: the emotional hypersensitivity is genetically determined, usually along with specific physical sensitivities that alone are presently recognizable as specific. After all, the well-recognized