CORRESPONDENCE

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Screening for Hyperlipidaemia in Childhood

SIR,—We wish to comment on two aspects of your leading article (16 November, p. 367). Firstly, you state that familial hyperlipidaemia in heterozygotes is readily corrected and imply that the outstanding problem is the age at which treatment should start. In our experience of treating children with the heterozygous form of familial hyperbetalipoproteinaemia (which is the only commonly encountered form of familial hyperlipidaemia in childhood) the abnormality is not always easily corrected, even in the short term, and long-term evaluation shows even poorer results. We have found that dietary measures alone achieve a mean reduction of serum cholesterol of about 20% of the pretreatment value,¹ and the addition of clofibrate to the diet achieves a further 10% reduction; cholestyramine resin (with or without dietary restriction) achieves a reduction of 36%.² The use of these treatments has not invariably been successful in lowering serum cholesterol to the generally accepted "normal" range even in the short term. Long-term evaluation of the three regimens³ has shown that two years after starting therapy the serum cholesterol level remains satisfactory in only 20% of children treated with diet alone and in none of a small group treated with the combination of diet and clofibrate. With cholestyramine, after three years adequate control was maintained in 57% of children. In our experience failure of long-term treatment is not due to loss of effectiveness of the agents prescribed but results from the inability of the children to follow the diet strictly over long periods or to take cholestyramine in sufficient amounts. Nearly all the children treated by us are from families in which premature coronary heart disease has already become manifest so that motivation towards treatment is likely to be high.

Secondly, you conclude by justifying extensive evaluation of cord blood lipoprotein assays for routine screening of babies born

to hyperlipidaemic parents and to families with a history of early-onset heart disease. While affected children may be identifiable by estimation of betalipoprotein cholesterol in cord blood serum,⁴ unless such diagnosis is to lead to the immediate institution of therapy it is probably better deferred. The need to treat such children in the first year is not proved; most paediatricians would be reluctant to use drug treatment at this age and evidence that infant formulae designed to lower serum cholesterol are always effective is lacking. Our own experience with two affected infants of heterozygous mothers has been disappointing; in neither case was adequate control of serum cholesterol achieved during the first year of life by dietary measures.

In view of the disappointing long-term results of available treatments, in our opinion screening entire populations of children for familial hyperbetalipoproteinaemia is not at present justified. We agree that screening of families in which early coronary heart disease has occurred should probably be undertaken but suggest that such screening be deferred until after the age of one year. Once treatment has been started it is important to supervise progress closely in order to assess adherence and to detect untoward side effects of the treatment(s).—We are, etc.,

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¹ Segall, M. M., et al., *Lancet*, 1970, 1, 641.

² West, R. J., and Lloyd, J. K., *Archives of Disease in Childhood*, 1973, 48, 370.

³ West, R. J., Fosbrooke, A. S., and Lloyd, J. K., *Postgraduate Medical Journal*, 1974, In press.

⁴ Kwiterovich, P. O., Levy, R. I., and Fredrickson, D. S., *Lancet*, 1973, 1, 118.

⁵ West, R. J., and Lloyd, J. K., *Gut*, 1974, In press.

Medical Graduates in Clinical Chemistry

SIR,—I have spent three years as registrar and then lecturer in clinical chemistry. After considerable speculation I have decided to give up the study of the specialty and return to a career in clinical medicine. On the eve of my departure it may be worth while expressing a few of my thoughts on the present and likely future role of the medical graduate in clinical chemistry.

A perusal of publications in journals devoted to clinical chemistry and attendance at Association of Clinical Biochemistry and other meetings convince me that at present the accent is on the word "chemistry" rather than on the word "clinical." Indeed, the word "clinical" might almost be dropped and replaced by some more accurate description such as "medical." The present trends of laboratory centralization and increasing subspecialization are likely to remove the medical graduate further and further away from patient management and more towards an administrative and scientific role. The medically-qualified clinical chemist in these circumstances has very little more to offer the patient than a non-medical biochemist. Indeed, our clinical colleagues are now becoming so well informed on scientific matters that the advisory role the chemical pathologist once enjoyed is tending to disappear.

Dr. F. L. Mitchell states¹ that a possible solution to present-day problems of recruitment of medical graduates would be to suggest a laboratory career from the outset of medical training rather than on completion. Surely a more logical solution would be fundamentally to alter the role of the medical graduate in clinical chemistry, taking him more out of the laboratory and into the wards where he should have definite clinical responsibility. There are many areas such as endocrinology, intensive care, etc., in clinical medicine where an able clinical chemist would be of immense value. The other cogent reason for getting the medical clinical