poorly. The only new criterion he proposes is that fat contents less than 10% (or possibly 14%) of dried stool can be eliminated from consideration of steatorrhoea. In his series he states that the procedure would have eliminated 92/231 (40%) of the 24-hour fat analyses.

Now, in the first place, he did not, in fact, do random stool specimens for his screening test, but determined per cent. fat excretions on an aliquot of the complete 24-hour specimen from a prepared patient. We have no way of knowing that his results would have been comparable had he actually used random specimens on out-patients. Secondly, other investigators do not seem to find such a relatively large number of patients with percentage fat below 10% or 14%, and this finding may be due to a local peculiarity. Thirdly, the proposed procedure, far from simplifying the work of the laboratory, increases it greatly. This is because 24-hour stool fats can be determined on wet stool by the methods of Van de Kamer *et al.*[1] with about one-third the effort (and about one-tenth the unpleasantness) required for determination of percentage fat in dried stool. In order to eliminate 92 simple wet stool analyses, therefore, the author has undertaken 231 tedious dry stool analyses—hardly a saving in labour.

Finally, I would recommend to your readers a screening procedure which is much simpler than either of the above: the serum carotene. A serum carotene above 70 μg./100 ml. effectively rules out steatorrhoea. Perhaps some 70% of patients would be ruled out by this simple test, leaving 30% who could be diagnosed by a 24-hour fat excretion (on wet stool). The one point that Dr. Hendry has shown clearly (as have others[2]) is that relatively simple preparation of the patient gives an entirely reliable 24-hour fat excretion.—I am, etc.,

Royal Columbian Hospital, New Westminster, B.C., Canada. RALPH SPITZER.

REFERENCES

[1] Van de Kamer, J. H., in *Standard Methods of Clinical Chemistry*, Vol. 2, 1958, ed. D. Seligson. Academic Press, New York.
[2] Cooke, W. T., *Brit. med. J.*, 1958, **2**, 261.

Smallpox in Nyasaland

SIR,—In the letter from the British Medical Association in Nyasaland (December 24, p. 1882) it is written that, in a statement to the press in London, a member of the Association denied the existence of an outbreak of smallpox in Nyasaland. The letter also states that the Nyasaland B.M.A. believes that the figures issued by the Director of Medical Services (Nyasaland) are accurate.

It should be pointed out that the member referred to did not deny the existence of an outbreak, but denied the existence of an epidemic. I do not wish to begin a correspondence on the difference between an outbreak and an epidemic, but it should be pointed out also that the D.M.S. has not used the word epidemic, but refers to a "serious outbreak." There is thus no incompatibility between the statement of the said member and the Director of Medical Services.

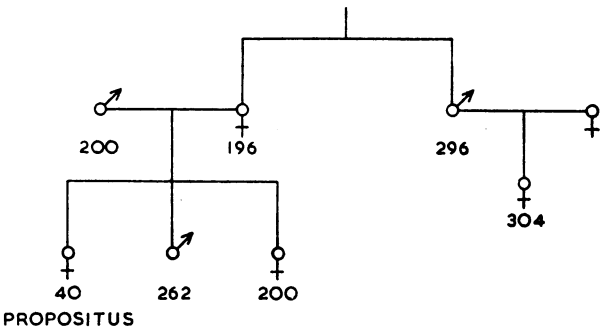
It is extremely important that any statement made by the B.M.A. in Nyasaland should be accurate, especially at the present time, as such statements are being used by the press as political propaganda, and we do not wish to discredit our profession.—I am, etc.,

Mlanje Mission, Nyasaland. KATHERINE M. ROBERTSON.

Suxamethonium Apnoea in Infancy

SIR,—Writing under the above heading (August 13, p. 535), Drs. L. Kaufman and H. Lehmann, and Mrs. Elsie Silk produce a family tree showing low pseudocholinesterase levels in the propositus and two siblings. It is therefore presumably the intention of these authors to suggest some genetic scheme of inheritance of this characteristic, though they produce no evidence for such an assertion.

I recently encountered a case of suxamethonium apnoea with low pseudocholinesterase, and, in the light



of the statements of Kaufman *et al.*, I decided to investigate the family tree. The result was as follows, being expressed in units=mols/ml./hour=mols of acetylcholine destroyed by 1 ml. serum in one hour. (Normal limits: 130–320.)

These results lend no support to the suggestion of any genetic behaviour on the part of pseudocholinesterase deficiency.

I am indebted to Mr. E. B. Love, B.Sc., F.R.I.C., and Mr. V. Rich, F.I.M.L.T., for the biochemical work involved.
—I am, etc.,

Swansea General Hospital. F. D. ADRIANVALA.

Uterine Rupture after Oxytocin

SIR,—To those who still advocate and practise medical induction I would respectfully suggest that they read Donald's *Practical Obstetric Problems*, second edition, Chapter XV.[1] "It is a common practice to prescribe castor oil, hot bath, and enema as a test of maturity in the mistaken belief that it is totally harmless. If the patient, as often happens after a hectic few hours of excretory activity, fails to go into labour, the psychological effects may be profound." Disappointment, fear for her baby, fear for herself, etc. In my opinion, it is on account of the usual failure to "bring on" labour that medical induction is so dangerous, because it stimulates some of the more enthusiastic and ill-advised "interferers" to give repeated intramuscular injections of oxytocin—a practice completely condemned by Wrigley at the Congress meeting at Cardiff in July, 1959, and one which can cause such a tragic result, as in the case reported by Dr. P. J. Dwyer (November 26, p. 1570).

This unfortunate young woman had had two previous normal confinements—and presumably the only reason for the "full" medical induction was because she was supposed to be one week overdue. Many obstetricians are undecided as to the importance of post-maturity and the need for induction, but I doubt if any obstetrician is able to decide that any pregnant woman is one week overdue. After nearly 40 years in the practice of