Thalassaemia in the British

SIR,—The recent article by Dr. H. H. M. Knox-Macaulay and others (21 July, p. 150) and the subsequent letter on this topic from Dr. J. J. Taylor (11 August, p. 353) have prompted me to record my experience on this subject in Cardiff.

Over the last eight years my laboratory has conducted a search for abnormal haemoglobin variants and for thalassaemias mainly in the provinces of Alberta, Saskatchewan, and Manitoba. In the last two provinces for the laboratory diagnosis of these conditions. The diagnosis of β-thalassaemia trait was made in 132 individuals in 105 families in central and southern Africa. The diagnosis of α-thalassaemia in 16 of the 80 found in Maniobola were British. In all of them the HbA2 levels were raised (mean 5.4%, S.D. ± 1.3% in the former; mean 5.1%, S.D. ± 0.7% in the latter; mean 2.2%, S.D. ± 0.4% in a control group), while HbF levels were normal in about half of these subjects and did not exceed 8% in the rest. These values are in agreement with those of the Finnish paper which we all now know.

The term "thalassaemia" is probably involved in both series. The proposition in nine of the 25 British families with α-thalassaemia were of the two-drug variety. No instances of homozygous β-thalassaemia or of a-α-thalassaemia were found in British subjects.

These results suggest that at least 20% of all β-thalassaemias in central Canada are of immigrant British stock and that in nearly half of them the condition is not suspected clinically. I agree with Dr. Taylor that one of the commonest diagnoses is "thalassaemia" or "iron deficiency anemia" and I would like to bring to your notice the case of the patient with a diagnosis of "HbA2 trait" who was not known to have a family history of the same. This case is in convincing some colleagues of the diagnosis.—I am, etc.,

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Hyperamylasaemia in Diabetic Ketosis

SIR,—We were interested to note that Dr. A. H. Knight and his colleagues (21 July, p. 128) have confirmed our original observation that the serum amylase is often considerably elevated in diabetic ketosis and that this phenomenon is not obviously due to pancreatitis.

We did in fact take the matter a little further by studying serial amylase levels during the course of oral glucose tolerance tests in 60 newly diagnosed and untreated diabetics and 52 controls. The curves in each group were summated and mean curves constructed. The results indicated that the amylase level rose in normals but tended to fall in diabetics, and the difference was statistically significant. The rise of the amylase level following an oral glucose load in normal subjects could be explained in several ways. It could be that the pancreas is stimulated, leading to an overflow of amylase into the circulation, or that oral glucose is initially taken into the liver and the glucose appearing in the blood could be due to breakdown of liver glycogen with further liver amylase into the circulation. It is not apparent why the amylase should behave differently in diabetics. The very high amylase levels in diabetic ketosis could lead to a massive breakdown of liver glycogen, and it is even possible that the hyperamylasaemia is a secondary phenomenon.

In this connexion we have often wondered why the blood sugar should be so high in diabetic ketosis when the patient may have been ill for some dayspossibly with anorexia and vomiting, which should lower the blood sugar, particularly if the patient has continued to take insulin.

Several workers have doubted whether the amylase was actually raised or not and have speculated that it may have a primary physiological function. The evidence would now seem to be strong enough to conclude that there are definite abnormalities in amylase metabolism under these conditions and elucidation of the mechanisms involved may throw some further light on the pathogenesis of diabetes.—We are, etc.,

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Significance of Pseudomonas aeruginosa in Sputum

SIR,—Dr. M. W. Burns's article (18 August, p. 382) draws attention to a problem which is increasing in frequency not only in Australia but also in Britain. I would like to make a few comments.

Sputum, stained with methylene blue, sometimes shows a small number of pseudomonads. This may lead to the erroneous conclusion that the patient is suffering from, or is at risk of suffering from, pseudomonal infection. The clinical significance of isolated pseudomonads, however, is uncertain, and it is important to recognize that Pseudomonas aeruginosa is no exception. The present method most commonly used in clinical bacteriology laboratories (in Britain) is an examination of the Gram film in conjunction with the findings on culture. In the absence of pus cells in the film, the isolation of pseudomonads from sputum cannot be regarded as a clear indication for antibiotic therapy. The report from our laboratory states whether or not pus cells are present in the specimen.) The 34 cases described without clinical infection, in which the organism was considered to be of minor importance, would most likely be in the category of non-purulent specimen and the patient would not automatically be put on antibiotics. Dr. Burns does not seem to take the purulence of the sputum into consideration in assessing the importance or otherwise of the isolated pseudomonads.

Another point is that no mention is made of the in vitro sensitivities of any of the pseudomonads isolated. This would have been