Fig. 1 Amylases of saliva and pancreatic extract (diluted to yield activities of approximately 1,000 Somogyi units), separated on Beckman microzone¹ cellulose acetate membrane. The pancreatic isoenzyme has a greater mobility towards the cathode.

Fig. 2 Amylases of normal urine, U (activity 320 Somogyi units) and sera, S1 and S2 from children with mumps parotitis (activities 980 and 205 Somogyi units respectively) separated on Beckman microzone cellulose acetate membrane. Two isoamylases are visible in the urine sample. The sera each show a single isoamylase of mobility identical to that of simultaneously separated diluted saliva.

For these reasons, and because of its simplicity, the staining method described above is preferred.

¹The gift of Dade Reagents, USA

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Letters to the Editor

Modification of Plasma Fibrinogen Method

The article by Burmester, Aulton, and Horsfield (*J. clin. Path.*, 23, 43-46) published in your recent issue describes a modification of the plasma fibrinogen method originally published by Miss A. Stransky and myself. I wish to point out an error in their article which seems to have been missed by them.

They recommend that venous blood is anticoagulated with one tenth its volume of 3.8% sodium citrate. In their eventual calculation of the fibrinogen result they include a correction factor of 10/9 for anticoagulant dilution. This of course is wrong because, first, the plasma dilution by anticoagulant is much more, and secondly their calculation ignores variations in haematocrit. The significance of these criticisms may be illustrated by the following examples:

Let (a) 4.5 ml blood be anticoagulated with 0.5 ml 3.8% sodium citrate and, (b) the plasma (+citrate) fibrinogen as measured by the method be 250 mg%. Using their factor 10/9 this would give a corrected fibrinogen concentration of 278 mg%.

The following calculations show the results in three examples where the packed cell volume is 40%, 50%, and 60% respectively, assuming, of course, that citrate dilutes the plasma only.

1 PCV 40%

Volume of plasma in 4.5 ml blood = 2.70 ml
Citrate dilution = 0.5 in 3.20 = 1 in 6.4

Plasma fibrinogen = $\frac{250 \times 6.4}{5.4} = 296 \text{ mg\%}$

Their corrected result is too low by 10 mg%, ie, an error of 6%.

2 PCV 50%

Volume of plasma in 4.5 ml blood = 2.25 ml
Citrate dilution = 0.5 in 2.75 = 1 in 5.5

Plasma fibrinogen = $\frac{250 \times 5.5}{4.5} = 305 \text{ mg\%}$

Their corrected result is too low by 20 mg%, ie, an error of 9%.

3 PCV 60%

Volume of plasma in 4.5 ml blood = 1.80 ml
Citrate dilution = 0.5 in 2.30 = 1 in 4.6

$$\text{Plasma fibrinogen} = \frac{250 \times 4.6}{3.6} = 325 \text{ mg\%}$$

Their corrected result is too low by 47 mg%, ie, an error of 14%.

In our article we recommended 0.05 ml 38.0% sodium citrate plus 4.0 ml venous blood. In the three examples cited above the dilution would be 1 in 49 at 40% PCV; 1 in 41 at 50% PCV; and 1 in 33 at 60% PCV. The error in the fibrinogen result would be 2%, 2½%, and 3% respectively, and the dilution effect using these proportions we ignored.

For these reasons I recommend that this aspect of their modification of our method is unacceptable.

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False Positive Curry's Test

The letter by A. W. Matthews and P. G. L. Stovin¹ published in the November, 1969, issue of the *Journal* prompts me to describe an unexpected cause of false positives.

Curry's test has been successfully used in this laboratory for a considerable time, but with the more orthodox use of separating funnels for phase separation, rather than Curry's technique which, in our hands, has often resulted in considerable loss of chloroform extract.

An unexpected positive result occurred which was traced to the unauthorized use of Whatman's no. 1 P.S. phase-separating filter paper to effect final separation of the phases prior to adding dithizone. If pure chloroform is filtered through this paper and dithizone added, a strong positive colour reaction occurs.

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¹ *J. clin. Path.*, **22**, 738.

Book reviews

Enzymopenic Anaemias, Lysosomes, and other Papers Proceedings of the Society for the Study of Inborn Errors of Metabolism, Symposium No. 6. Edited by J. D. Allan, K. S. Holt, J. T. Ireland, and R. J. Pollitt. (Pp. x + 213; illustrated. 60s.). Edinburgh: E. and S. Livingstone.

The publication in book form of the papers read at symposia is becoming increasingly common practice, although the need and usefulness of such records is often questionable. Invited papers from eminent workers tend to be reviews of work which will have been published previously elsewhere; as the main value of a symposium lies in the liveliness of the discussion and in the selection of appropriate topics to ensure a relevant and comprehensive survey of the subject, the merits of publication of a symposium should be judged on the skill of the editing which should translate the spontaneity of the meeting into print. On these grounds the book under review can be considered successful.

The Society for the Study of Inborn Errors of Metabolism is a small and active group whose previous symposia have also been published. This was their sixth meeting which was held in Zurich in 1968. There were 33 participants and the subject matter of the main topics was spread equally between clinical chemistry and haematology—abnormalities of red-cell enzymes (under the inelegant title of 'Enzymopenic anaemias'), lysosomes and urea cycle disorders—while there was also a miscellany of individual papers, 14 in number, which covered a wide range of metabolic disorders. The session on enzymopenic anaemias consisted of four papers, including general reviews by T. A. Prankerd and A. J. Grimes, a paper, on neonatal and paediatric aspects by Werner Schröter, and one technical paper on assay of G6PD and 6PGD by Tan and Whitehead. The properties and functions of lysosomes and their role in chronic granulomatous disease, the Chediak-Higashi syndrome, and other unusual but interesting conditions were dealt with by A. C. Allison, H. G. Hers, and D. G. Nathan. Several aspects of ammonia intoxication were dealt with in the next session, including one paper on congenital hyperammonaemia in which Alex Russell postulates

a common biochemical basis for adult migraine and childhood cyclical vomiting as a result of an enzymatic defect of the Krebs-Hersleit urea cycle.

Some of the papers were rather more pedestrian but all are well presented in a clear manner and they have been enhanced by the discussion which has been included in full. The editors have carried out their task with success, and it seems likely that the series has become established as an annual publication.

S. M. LEWIS

Blood Replacement By U. F. Gruber. Translated by L. Oxtoby and R. F. Armstrong. (Pp. xii + 284; illustrated. DM24.00.) Berlin, Heidelberg, and New York: Springer Verlag. 1969.

This book deals with the pathogenesis and therapy of haemorrhagic shock. The author's choice of a simple forthright title is characteristic of his presentation throughout. While every statement is supported by copious use of references, the reader is left in no doubt where Gruber stands and the summary of each chapter further clarifies his recommendations.

The overall message of the book is to deplore the extravagant use of donor blood for the treatment of moderate haemorrhage (up to 1,500 ml) in the otherwise healthy patient. The relative merits of the important plasma substitutes are weighed against the dangers and complications of blood and blood products. Highest in the order of dangers of blood is the risk of transmitting serum hepatitis, the incidence of which is put at 14% to 30% depending on the number of units of blood transfused. These high percentages take account of icteric and anicteric forms of hepatitis. No figures are quoted for the United Kingdom, but it is noted that the frequency tends to be highest in those countries using paid donors. Up-to-date reference is made to the use of plasma protein solution (PPS), a pasteurized form of fibrinogen-free plasma now undergoing clinical trial in this country, but the book has been published just too soon for reference to the exciting new work on the Australia (Au) antigen which may be the causative virus of serum hepatitis.

Haematologists with responsibilities for blood banking will welcome this book as it spells out the dangers of blood transfusion and, if read by anaesthetists and others engaged in resuscitation, it may encourage an awareness of the value of other forms of treatment.

The translators must be congratulated for giving us such a palatable version of this valuable German book.

W. J. JENKINS