be of whole blood so as to traverse the same pathway as the unknown samples. We have found a commercial whole blood preparation (Hyland Laboratories) to be satisfactory for this purpose.

In practice such a control procedure is sometimes insensitive in detecting small aberrations. We have, therefore, introduced the cumulative delta sum technique which is applicable to large batches. Theoretically the daily average should be constant. Deviations from the norm are rapidly detected when the average result each day is subtracted from a standard reference figure and these differences cumulatively added. The acquisition of the necessary information for the Q-sum is simplified by the recent introduction into the laboratory of a punch card data processing system.

This cyanmethaemoglobin method of haemoglobin analysis on the AutoAnalyzer has been in routine use for two years and has proved itself to be entirely satisfactory.

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Letters to the Editor

RENAL MASTOCYTOSIS IN A CASE OF
CONN'S SYNDROME

Sir,

Evidence has accumulated to suggest a role for heparin in the regulation of aldosterone secretion (Vallent, Fachet, Palkovits, and Dévényi, 1964; Abbot, Gornall, Sutherland, Steifel, and Laidlaw, 1966; Conn, Rovner, Cohen, and Anderson, 1966) and, therefore, we think it is worthwhile to make known an observation on renal mastocytosis in a case of Conn's syndrome.

The mast-cell count was estimated according to Mills, Strickland, and Paterson (1958). The results were expressed as the average number of mast cells per square centimetre of tissue.

According to Staemmler (1921) the normal kidney does not contain any mast cells, but these appear accompanying any kind of sclerotic process. In the renal cortex of a case of Conn's syndrome, when examining several tissue blocks, we found that the average number of mast cells was 784/sq. cm. (between 611 and 937). The cortices of 14 nephrosclerotic kidneys, used as controls, had an average mast-cell count of 135/sq. cm. (between 0 and 264).

On the basis of the aldosterone-inhibiting action of heparin, it may well be that the renal mastocytosis was a compensatory effort as a consequence of aldosterone hypersecretion. We should like to call attention to this phenomenon which, naturally, has yet to be confirmed in other cases. We consider it is necessary to re-examine the kidneys in cases of advanced primary aldosteronism from the point of view of their mast-cell content.

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EFFECT OF DILUENTS ON BLOOD CLOT LYSIS

Sir,

I was interested to read Mr. M. J. Gallimore's paper 'Effect of diluents on blood clot lysis' (May, 1967). For the record, in 1958 Dr. Ferguson and I, in experiments very similar to those of Mr. Gallimore, showed that sodium chloride is inhibitory to fibrinolysis compared with phosphate buffer, and we concluded: 'normal saline is found to be inhibitory to fibrinolysis, which in part explains the very different degrees of fibrinolytic activity found in normal blood by various workers.'

G. R. FEARNLEY
Painswick, Glos.

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