Fig. 1 Correlation between serum viscosity and the decrease in serum sodium when measured by indirect potentiometry.

Fig. 2 Correlation between serum viscosity and the decrease in serum sodium when measured by flame photometry.

sodium measurement. When direct potentiometric methods are used, the presence of hyperviscosity does not significantly influence the serum sodium value. With measurements using flame photometry and indirect potentiometry, pseudohyponatremia occurs. With indirect potentiometric measurements, a correction factor which can be used is a 2 mmol/l decrease in serum sodium for every centistoke increase in serum viscosity. This is linear, at least in our study to a serum viscosity of approximately 20 centistokes. With flame photometric measurements of serum sodium, no such correction factor can be meaningfully applied. For example, one patient with a serum viscosity of five centistokes had almost the same decrease in serum sodium as the patient with a viscosity of 15 centistokes.

It is essential that every laboratory report indicate the technique used for measuring serum sodium. Moreover, the clinician should also be aware of the methodology in use especially when different techniques are in use in the same laboratory.

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Tripeptidylcarboxypeptidase activity of angiotensin I converting enzyme in human serum

Angiotensin I converting enzyme (ACE) is considered as a dipeptidylcarboxypeptidase (EC 3.4.15.1). Two of us,¹ however, were able to demonstrate that in hog lung and kidney, ACE also acts as a tripeptidylcarboxypeptidase. The possibility of enzymatic release of C-terminal peptides of substrates having a proline group in the penultimate position has never been shown in human serum but may induce new insights in the generation and deactivation of some vasoactive peptides—for example, des-Arg⁹bradykinin.

With benzoyl-glycyl-L-seryl-L-prolyl-L-phenylalanine as a substrate we were able to demonstrate that human serum also contains this tripeptidylcarboxypeptidase activity. When examining normal human sera ($n = 27$) with a high resolution chromatography-assisted technique,² a normal range of 13 $\mu\text{mol/min/l}$ ($\text{SD} \pm 4$) liberated benzoyl-glycine was shown. The tripeptidylcarboxypeptidase activity could be completely inhibited by 1 $\mu\text{mol/l}$ captopril. Sera of four patients with active sarcoidosis were also examined. Since different studies³ have confirmed that active sarcoidosis is reflected in a high dipeptidylcarboxypeptidase activity of ACE, we thought it would be interesting to measure the tripeptidylcarboxypeptidase activity in four cases of active sarcoidosis with increased dipeptidylcarboxypeptidase activity of ACE, there was also significant increase in tripeptidylcarboxypeptidase activity (mean 27 $\mu\text{mol/min/l}$). Although the increase of ACE activity in sarcoidosis and other pathological conditions could

still have been due to an isoenzyme, the results we obtained did not support this hypothesis.

This is the first observation in which the existence of a tripeptidylcarboxypeptidase activity of ACE in human serum is demonstrated under normal and pathological conditions. Therefore we would like to suggest the reconsideration of the trivial name of ACE.

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Assessing bone marrow cellularity

I was very interested in the recent report by Dr Al-Adhah and Dr Cavill¹ on the assessment of cellularity in bone marrow fragments. Some four years ago, I and my colleague, Mr W Slidders had shown the close correlation ($r = 0.98$) between our point-counting method and results from the Quantimet 720.² In this paper, we devised a method to overcome the non-random distribution of fragments because of their different rates of sedimentation during fixation and processing.

Al-Adhah and Cavill obtained a coefficient of variation (CV) between 8.2 and 34.7%. These authors felt that their result reflected the more likely level of reproducibility of the point-counting method than our CV of 2.6%. However, the crucial difference is that the method of