Letters to the Editor

Pseudohyponatremia and hyperviscosity

Serum from patients with hyponatremia have low sodium values when analysed by flame photometry. The reason for the spuriously low sodium is related to problems with sample aspiration and dilution by the instrument and also because of the decrease in plasma water due to high protein concentration. Methods that are available for measuring serum sodium include the traditional technique of flame photometry and more recently the use of an ion-selective electrode. When the serum sample is diluted before analysis by the electrode (indirect potentiometry), the serum sodium values would be expected to be low in the presence of hyperviscosity, proteininaemia and hyperviscosity. With direct potentiometry where no sample dilution takes place, no interference would be expected since the activity of sodium in the water phase only is being measured. The present study was undertaken to determine the magnitude of the decrease in serum sodium in viscous sera using flame photometric and indirect potentiometric measurements for sodium measurement.

Patients and methods

Samples were obtained from patients at the Vancouver General Hospital whose sera when analysed were noted to have increased viscosity at 37°C (Table). The range of serum viscosities was 3.0–17.8 centistokes (normal is <1.8). Serum sodium concentrations were measured in duplicate by flame photometry (FP) (Beckman KLiNa flame: Beckman Instruments Inc, Fullerton, California, indirect potentiometry (IP) Beckman Astra, Beckman Instruments Inc, Fullerton, California) and by direct potentiometry (DP), (Nova-1 Na/K analyser, Nova Biomedical Inc, Newton, Massachusetts). Serum viscosity relative to normal saline was measured using a Cannon Manning Viscometer. The differences in serum sodium obtained between measurements by DP and IP (\( \Delta Na \)) and DP and FP (\( \Delta Na \)) were correlated with measurements of serum viscosity.

Results

Figure 1 shows the correlation (r = 0.95, p < 0.001) between %Na and serum viscosity. Figure 2 shows the correlation (r = 0.71, p < 0.01) between %Na and serum viscosity. For serum sodium measurements performed by indirect potentiometry, a 2 mmol/l decrease is seen for every one centistoke increase in serum viscosity.

Discussion

Dangerous pseudohyponatremia can occur in patients with hyponatremia (Table). We wish to draw attention to the fact that the degree of pseudohyponatremia is dependent upon the technique that is used for
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References


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, 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-Arg9-bradykinin.

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, a normal range of 13 μmol/min/l (SD ± 4) liberated benzoylglycine was shown. The tripeptidylcarboxypeptidase activity could be completely inhibited by 1 μmol/l captopril. Sera of four patients with active sarcoidosis were also examined. Since different studies 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 μmol/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-Adhadh 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-Adhadh 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...