the decrease in ACE may have been due to a direct effect of the steroid on ACE. More recently, decreases in ACE activity were reported in samples from patients with asthma or sarcoidosis, or asthma or sarcoidosis, with prednisolone in vitro (2). We have made a more comprehensive study of the effects of prednisolone and methylprednisolone in vitro upon ACE activity in samples from four distinct patient groups: those presenting with small-cell bronchogenic carcinoma, pulmonary sarcoidosis, or asthma, and a control group presenting with no history or evidence of lung disease.

We assessed the following variables: two concentrations of each steroid, pharmacological (3.3 μmol/L) and suprapharmacological (1 mmol/L); incubation of steroid with plasma, from 0 to 96 h; and incubation time of 4 °C or 37 °C. Incubation of plasma from control and diseased groups of patients with pharmacological concentrations of either steroid resulted in no significant decrease in ACE activity under any conditions. At suprapharmacological concentrations, incubation at either 4 °C or 37 °C for up to 48 h produced no significant inhibition. However, when samples were incubated for 96 h, there was significant inhibition of ACE activation at both temperatures in samples from all groups except the lung-cancer group.

Qualitatively, these data support earlier findings (1, 2) suggesting a direct role for corticosteroids on ACE activity; they also indicate a similar action for methylprednisolone. Quantitatively, we conclude that the direct inhibition of ACE by prednisolone is less likely to interfere with the clinical usefulness of ACE monitoring of prednisolone therapy of sarcoidosis than was earlier thought. The lack of inhibitory response seen in the samples from patients with bronchogenic carcinoma warrants further study with samples from patients with different lung tumor histopathologies.

References

Magnesium, Surfactants, and Centrifugal Analyzers, J. Croker and M. J. Peake (Dept. of Biochemistry and Chemical Pathology, Flinders Medical Centre, Bedford Park, South Australia, 5042, Australia)

We have recently identified a problem that may cause sporadic errors in the measurement of magnesium by a colorimetric method (Clin Chem 1983;29:2120–2121) in the Cobas Bio centrifugal analyzer (Roche Analytical Instruments, Nutley, NJ 07110).

When using a bioMérieux calmagite reagent (cat. no. 61411) as per their recommendations for the Cobas Bio, we have found that the 30-s time interval after the final mix of sample with calmagite and base reagents may be insufficient for stable final absorbance measurements, and that in some instances inappropriately high absorbances can persist for as long as 2 min after mixing. Duplicate magnesium estimations have varied within-run by more than 0.2 mmol/L in this and in another Cobas Bio in our department.

We suggest as the most likely cause for this anomaly the presence of surfactant in the calmagite reagent, which results in air bubbles and scattering of incident light. A complete set of results will be invalid if an inappropriately high absorbance occurs in either the standard cuvets (all results falsely low), or the reagent blank cuvet (where values less than the standard concentration will be falsely low and other values falsely high). Individual results may be falsely high in other instances. After having the mix cycle on the centrifugal analyzer verified for correct operation, we resolved this problem by extending the time for final absorbance measurements to 4 min, thus improving within-run precision to approximately 1% by the elimination of gross errors.

We have noted this problem with other procedures used in this instrument when large amounts of surfactant are involved, and we recommend that, during method development, unstable absorbance readings be investigated as a possible reason for unacceptable imprecision. Failure to do this may result in a potentially reliable method being unnecessarily rejected.

Effects of Needle Size and Storage Temperature on Measurements of Serum Potassium, L. Verresen, R.L. Lins, H. Neels, and M.E. De Broe (1 Dept. of Nephrology-Hypertension, Algemeen Ziekenhuis Stuivenberg, and University of Antwerp and Affiliated Hospitals, Antwerp, Belgium; 2 Dept. of Clinical Biochemistry, Algemeen Ziekenhuis Stuivenberg.

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Various sampling, processing, and storage conditions reportedly affect the measurement of potassium in blood. The aim of this study was to evaluate the effect of the puncture needle size and the storage temperature of the blood.

Using a 23- or 19-gauge needle we collected blood specimens into polystyrene tubes for clotted blood, then measured the potassium with an Hitachi automatic analyzer. The results were analyzed by the Wilcoxon matched-pairs signed ranks test.

The mean (± SEM) concentration of serum potassium in samples drawn with a 19-gauge needle (n=10) was 4.1 ± 0.5 mmol/L and in those drawn with a 23-gauge needle (n=10), 4.4 ± 0.6 mmol/L. The mean difference between the two groups was 0.3 ± 0.2 mmol/L (p <0.001). The mean concentration of potassium in blood analyzed immediately (n=30) was not significantly different from the value observed in the samples stored for 16 h at 18 °C (n=30): 4.5 ± 0.8 and 4.6 ± 0.7 mmol/L, respectively. In contrast, there was a significant difference (p <0.001) between the untreated samples and the samples stored at 4 °C for 16 h (n=30) (6.2 ± 0.8 mmol/L).

The accuracy and precision of determinations of serum potassium decline rapidly when samples are stored. In particular, when serum is not separated from erythrocytes, there is a constant interaction between serum and cells. One of the mechanisms is an active transport system, coupled with the absorption and phosphorylation of glucose, that pumps potassium into the erythrocytes (TS Danowski, J Biol Chem 1941;139:693–705). Reducing the temperature results in nonspecific enzyme inhibition, blocking this trans-