eter added to 4 mL of blood with normal activities for total CK, LD, and AST yielded respective enzyme concentrations of 6070, 675, and 65 U/L (normal range =200, =440, and =65, respectively). The above finding should therefore be taken into consideration as a possible cause of a clinically unexplained increase in total CK.

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Influence of Dopamine on Peroxidase-Based Assays

To the Editor:

During the investigation of discrepant laboratory results, we detected an unusual interference in several of the biochemistry tests. The patient’s specimen had been obtained from a catheter in the jugular vein. Results of all analytes were discordant from those for a sample drawn 10 h previously. The cause of the discrepancy was thought to be contamination with isotonic saline containing 100 g/L dextrose. The dilution ratio of intravenous fluid to serum was 1:10, as substantiated by several results obtained with an Ektachem 700 analyzer (Johnson & Johnson, Rochester, NY), including: sodium, 132 vs 140 mmol/L; potassium, 4.4 vs 4.8 mmol/L; chloride, 102 vs 112 mmol/L; and glucose, 21.8 vs 9.5 mmol/L. However, the creatinine result (96 vs 221 mmol/L) did not conform to the dilution ratio.

When the specimens were further analyzed with a Boehringer Mannheim/Hitachi 917 analyzer (Boehringer Mannheim, Laval, Quebec), and using the manufacturer’s reagents, we noted a considerable decrease in the values for triglycerides (0.73 vs 1.23 mmol/L), cholesteryl (1.3 vs 1.7 mmol/L), and uric acid (126 vs 528 mmol/L).

Upon investigation, we found that the sampling catheter was being used to administer dopamine at a rate required to maintain a steady-state plasma concentration of ~62 μg/L (renal dose). A review of the affected chemistries revealed that each of these analyses depended on the generation of peroxide for the indicator reaction, and that dopamine might be functioning as a reducing agent, resulting in the depletion of peroxide. To investigate our hypothesis, we analyzed three different sera with dopamine (or saline) added. Fig. 1 shows the mean decrease in analyte concentration. Control assays such as albumin and total protein, which do not rely on hydrogen peroxide, showed no interference (data not shown). All procedures were performed in accordance with the ethical standards of the institutional review board of the hospital.

As Fig. 1 shows, the interference starts at concentrations of dopamine ~1 mg/L. The steady-state concentration of dopamine in plasma is ~0.11 mg/L when administered for its cardiac inotropic effects and about one-half of that when given for renal vascular effects [1]. At these concentrations, dopamine does not appear to affect hydrogen peroxide-based assays. The extent of the interference appears to vary, depending on the amount of hydrogen peroxide generated in the initial reaction. Weber and van Zanten [2] have previously noted that dopamine has a negative influence on the enzymatic creatinine assay. To our knowledge, there have been no other investigations of this interferent.

Although the practice of obtaining blood specimens from intravenous lines is not ideal, it is convenient. We recommend that blood should not be drawn from intravenous lines when dopamine is being infused. Relatively small quantities of dopamine may affect assays that rely on hydrogen peroxide generation for their indicator reaction.

References

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