Screening Assay for Ethylene Glycol in Serum

To the Editor:
The recent paper by Eder et al. (1) recalls, among other things, the problems that a clinical STAT laboratory has to face in the case of ethylene glycol poisoning. As an alternative to the precise and accurate gas chromatographic technique, the enzymatic assay with glycerol dehydrogenase (EC 1.1.1.6) and NADH as tracer has been proposed by various authors [see, e.g., (2–4)]. The relative nonspecificity of glycerol dehydrogenase from Enterobacter aerogenes toward diols and triols allows interference with this test by propylene glycol, glycerol, 1,2-butanediol, and 2,3-butanediol (2, 5, 6). Nonetheless, its usefulness in emergency screening for ethylene glycol has been stressed, particularly for its technical simplicity (2).

Rynder et al. (2) used an analyzer, the Du Pont aca® SX (presently distributed by Dade Behring Inc., Deerfield, IL), that is particularly suited for emergency situations. Presently this instrument, together with the more recent one, the aca STAR, is still widely used in laboratories both in this country and in the United States. Unfortunately, the product that, following the method of Rynder et al., we were using to avoid interference from endogenous triacylglycerols and glycerol (triglyceride test kit for Monarch, cat. no. 35190, Instrumentation Laboratory) is no longer available in our country.

Because skilled personnel are not available at all times in our emergency laboratory and because cases of ethylene glycol poisoning are infrequent but not uncommon, I decided to continue to use the aca method whenever the gas chromatographic technique was not available. The modification I make is trivial: I cut compartment no. 2 (ingredients: lipase and stabilizers) of the aca triglyceride analytical test pack (Du Pont, cat. no. 702230901), wash it with deionized water, and firmly reseal the hole with transparent adhesive tape. I calibrate the test with a 16 mmol/L solution of ethylene glycol in pooled sera that are free of ethylene glycol, as assessed by gas chromatographic analysis, and use the same pooled sera as a blank. Linearity was studied by serial dilutions of the calibrator in ethylene glycol-free pooled sera down to the concentration of 1.6 mmol/L. The linear regression analysis of the results gave the following statistics: dose = 0.955 × response − 0.04 mmol/L, n = 10, r = 0.995, S_0/5 = 0.41 mmol/L. The test is recalibrated every month.

In our laboratory, we are aware that the results obtained in this way need cautious interpretation and gas chromatographic confirmation; however, this test permits us to screen economically for ethylene glycol with a typical turnaround time <30 min.

References

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The Director of Regulatory Affairs and Quality Assurance for the Dade Behring Chemistry Group replies:

To the Editor:
As manufacturer of the aca® discrete clinical analyzer and the TGL test pack described in this Letter to the Editor, Dade Behring appreciates the opportunity to comment. As described in our labeling, the intended use of the TGL test pack is to measure quantitatively the triglycerides in serum. Dade Behring cannot support non-standard use of the TGL test pack for ethylene glycol testing because the TGL test pack has not been optimized for this use. Furthermore, because we have not filed a premarket notification with the US Food and Drug Administration for use of TGL test packs in ethylene glycol testing, we are restricted from promoting or endorsing the product for this use.

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Why is Troponin T Increased in the Serum of Patients with End-Stage Renal Disease?

To the Editor:
Haller et al. (1) have strengthened the argument that the increased serum cardiac troponin T (cTnT) found in many patients with end-stage renal disease (ESRD) originates in heart muscle and not regenerating skeletal muscle, as suggested by others (2). We recently measured serum cTnT using the Elecsys 2010 immunoassay system (Boehringer Mannheim), a method that is specific for cTnT (3), and creatine kinase MB isoenzyme (CK-MB) mass (Dade Stratus II immunoassay system) in 71 stable patients with ESRD. The serum cTnT was >0.1 µg/L in 37 (52%) of them, and interestingly, cTnT and CK-MB were significantly correlated (Fig. 1); in all 14 patients with serum CK-MB >4 µg/L the serum cTnT was >0.1 µg/L. This is consistent with a common source of these proteins, although of course it does not indicate whether the common source is heart or skeletal muscle.

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