Incidence of “Flipped” Lactate Dehydrogenase Isoenzyme Pattern (LD₁ > LD₂) in Specimens with Normal Total Lactate Dehydrogenase from Coronary-Care Patients

To the Editor:

The effectiveness of analysis of lactate dehydrogenase (LD; EC 1.1.1.27) isoenzymes in diagnosis of myocardial infarction is well documented in the literature. The “flipped” pattern (LD₁ > LD₂) seen on LD fractionation is commonly regarded as an indicator of myocardial infarction (1). At our hospital, clinicians frequently request that serum LD isoenzyme analysis be performed on samples from Coronary Care Unit (CCU) patients, even when the total serum LD activity is normal. In investigating the usefulness of performing this test under these circumstances, we could not find data in the literature regarding the frequency of a “flipped” pattern in patients with normal serum LD activity. Therefore, we reviewed LD isoenzyme determinations done on serum samples from a series of patients admitted to the CCU at Madigan Army Medical Center from 15 September 1981 through 15 February 1983.

All serum samples were stored at room temperature and LD isoenzymes were determined within five days. Hemolyzed specimens and refrigerated or frozen specimens were excluded from the study. Some specimens were repeat samples on the same patients but obtained on different days. Total LD was measured with the SMAC (Technicon, Inc., Tarrytown, NY), with Technicon reagents. LD isoenzymes were determined by the lactate dehydrogenase isoenzyme (fluorometric) method of Corning Medical, Medfield, MA; only Corning Medical reagents were used. Results for total LD were separated into normal and above-normal groups, and each group was examined for the presence or absence of a “flipped” pattern (LD₁ > LD₂).

Our results are summarized in Figure 1. Serum samples with a normal total LD activity showed a “flipped” pattern in four of 156 (2.5%) samples, compared with 114 of 274 (42%) with an increased total LD activity. All four patients with normal total LD who showed the “flipped” pattern also had creatine kinase (CK) MB isoenzyme. Therefore, had we not determined LD isoenzymes in those samples, we still would have noted enzyme evidence of myocardial infarction, because total CK activity and CK isoenzymes are determined for all CCU patients at our hospital. Moreover, these four patients had total LD activity > 75% of our upper limit of normal. Had this been the cutoff value below which isoenzymes would not be determined, still none of the patients with a “flipped” pattern would have been missed.

We conclude that LD isoenzyme determinations are not necessary for serum samples with a total LD activity of < 75% of the upper limit of normal, if one is evaluating a patient only for myocardial infarction. In fact, given the incidence percentages mentioned above, it may not be an effective use of resources to determine LD isoenzymes for any sample with normal total LD activity from patients with suspected myocardial infarction, especially if CK is also being monitored.

The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

Reference


Stephen H. Koopmeiners
J. David Turnbull
Dept. of Pathol.
Madigan Army Med. Center
Tacoma, WA 98431

Phenacemide Interference with Jaffé-Type Determination of Creatinine in Three Automated-Analysis Systems

To the Editor:

Phenacemide [(phenylacetylimidene), an anticonvulsant drug, reacts with alkaline picrate to produce a chromogen that interferes in the Jaffé method for determination of creatinine (1–3). Here we report the extent of interference of phenacemide with creatinine in three different analytical systems: SMAC (Technicon Instruments Inc., Tarrytown, NY 10591), aca (Du Pont Instruments, Wilmington, DE 19898), and ASTRA 8 (Beckman Instruments, Inc., Brea, CA 92624).

We dissolved 0.56 and 1.49 mg of phenacemide (USP Reference Standard; United State Pharmacopeia, Rockville, MD 20852) in 4 mL of pooled serum, then measured creatinine with each instrument, using the methods and reagents supplied by the respective manufacturers. We also measured creatinine in an aliquot of the pooled serum without added phenacemide, by the three analytical systems.

Phenacemide interfered positively with determination of creatinine in the ASTRA 8 and the SMAC, but negatively with the aca method (Table 1 and Figure 1), interference similar to that noted with some cephalosporin and cephamycin antibodies. The interference was least with the SMAC method, as also reported for cefoxitin and cephalothin (4, 5); cephalothin, like phenacemide, causes a negative interference with determination of creatinine with the aca method (5).

The greater positive interference