illness. The T4/T3 ratio was about as useful as values for reverse T3 in detecting this apparently abnormal metabolism of T4.

Additional studies are required to gain a clearer understanding of the changes that occur in the metabolism of thyroid hormones in non-thyroidal illness.

Several recent papers have compared FTI with direct methods of measuring free T4 (6–9). Chopra et al. (6) did not use any of the methods we report here. Witherspoon et al. (7) tested the Damon and Clinical Assays FT4 kits and found that values correlated well with those by equilibrium dialysis for all their samples; they also found better agreement between FTI and both CPT4 and DPT4 than we have. Lawlor and Blaustein (8) state that the Clinical Assays method correlates well with equilibrium dialysis in samples from patients with non-thyroidal illness, but of their samples, however, had normal T4 concentrations. Our samples from patients with non-thyroidal illness had decreased values for total T4, normal thyrotropin, and high reverse T3, so the differences between our data and those of others may be attributable to differences in the sample population tested.

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Sufficiency of One Control: A Survey of Three Small Chemistry Analyzers

To the Editor:

It has become common practice to include both a “normal” and an “abnormal” control with each chemistry run, and accrediting agencies strongly suggest we do it. For small hospitals in which a run may consist of only three or four specimens the additional testing significantly increases costs.

To see if two-control testing is really necessary, we studied a year’s performance of three small semi-automated analyzers, the System S (Gilford Instrument Laboratories, Inc., Oberlin, OH 44074), the VP (Abbott Laboratories, Diagnostics Division, Dallas, TX 75247), and the Gemeni TM (Electronucleonics, Inc., Fairfield, NJ 07006) for a single test, serum calcium, on two lot numbers of commercial control serum. One was a “normal” control with a mean value of 99 mg/L, the other an “abnormal” one with a mean value of 135 mg/L. Both were included in each patient run. There was no deterioration of either specimen throughout the year.

Of 698 pairs of results, only seven (1%) were discordant by deviating from the respective mean values in opposite directions, and in each of these the run would have been rejected because the “normal” control exceeded its acceptable limits. In all others when one was high the other was proportionately high, as were all the patient results in the run.

These conclusions were reinforced by “delta checks” (1) of 166 patients who had more than one serum calcium done during the year. Some of these inpatient patient results were discrepant enough to achieve clinical significance until corrected for differences in serum total protein (2) and for the normal control value of the run in which it was included. Variation of corrected values was similar to that reported by others for biological variation (3).

We conclude that two control speci-
mens are not necessary in each run for the instruments tested, although they may be required with others (4). Furthermore, comparisons of repeat values from any patient may be made more useful by minimizing the effect of analytical error through use of correction factors based on a single control tested at the same time. Quality-control costs are substantial (5) and unneeded ones should be eliminated.

One control per run is essential. Two seem to be redundant.

References

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More on Lactate Dehydrogenase in Myocardial Infarction

To the Editor:

We wish to respond to the Letter of Gordesky and Winsten (Clin. Chem. 28: 1239, 1982) commenting on our study (1) of lactate dehydrogenase isoenzyme (LD-1) after myocardial infarction. They did not find the ratio of LD-1 to LD to be useful in a study of 47 patients, in sharp contrast with our results in a larger series (1) and our subsequent experience involving more than 100 patients. These differences reflect two major differences in approach:

1. The large proportion of false-positive results reported in their Letter were found by using a cutoff point of 27.5% for the LD-1/LD ratio, whereas we used a cutoff point of 40%. Their lower cutoff point surprises us, because the two studies involved the same immunochemical reagents, the same assay temperature (30 °C), and a similar reagent with pyruvate as substrate (2). In our experience, a cutoff of about 40% is ap-
propriate with this assay and, indeed, Gorsedsky and Winfield found only one value above 40% among their patients without infarction.

2. They reported LD-1/LD values >40% in only 16 of 28 patients with infarction, whereas we found values >40% in all patients with infarction. This difference appears to reflect, in part at least, differences in blood-sampling times. We obtained samples at intervals of 4 to 8 h during the first 24 h of hospitalization, whereas Gorsedsky and Winfield used only one sample obtained on admission and one the following morning at 0800 hours. These are two very different definitions of measuring the “peak value in 1st 24 hrs after admission” as stated on the Figure accompanying the Letter. In Gorsedsky and Winfield’s definition, this meant merely samples obtained “at least 7 h” after admission. We are not surprised that many infarcted patients had normal LD-1 results with this sampling protocol (see Figure 3 of reference 1).

Use of the ratio of LD-1/LD, rather than use of LD-1, is problematical. We presented results for both LD-1/LD and LD-1 and saw only a slight advantage of the ratio, as evidenced in Figure 2 of reference 1. LD-1 may be increased before the LD-1/LD ratio, and we present our time-course data using the more conservative LD-1/LD ratio. In a just-completed study of 100 patients in a coronary care unit (CCU), both LD-1 and LD-1/LD again were highly sensitive and specific for the diagnosis of myocardial infarction, but again we saw no clear advantage to using the ratio rather than LD-1 alone. We have also seen isolated patients with congestive failure (high LD-5) after infarction in whom the ratio was normal while LD-1 was clearly increased. Thus the ratio—like all clinical laboratory tests—is best interpreted in light of the clinical findings.

Finally, we agree that studies of LD-1 need to be done in settings other than the CCU and that such studies are difficult. These studies appear warranted because studies of LD-1 in the CCU setting suggest that it is a useful diagnostic determinant for myocardial infarction (see, e.g., 1, 3, 4). We will present a study of LD-1 in cardiac surgical patients at the 1982 national meeting of the AACC (5) and we look forward to seeing studies performed outside the CCU setting by other investigators.

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Temperature Conversion Factors for Total and isoenzyme 1 Activities of Lactate Dehydrogenase

To the Editor:

Although national and international standardizations of enzyme determination specify the temperature of the incubation mixture, some instruments unfortunately do not have the capability to allow one a free choice of temperature to use. One must therefore convert the results from one temperature to another, to be able to compare results with others reported. Because a frequent objection to this practice is that the different composition of isoenzymes does not allow the mathematical conversion from one temperature to another, I studied the temperature dependencies of the activity of total lactate dehydrogenase (LD, EC 1.1.1.27) and of the “heart-isoenzyme” (LD-1) to see whether a temperature-conversion factor can be used in this specific case.

The LD and LD-1 activities were measured according to the French proposals (1) with minor modifications. I used the Cobas-Bio centrifugal analyzer according to the manufacturer’s specifications. LD-1 was separated from the other LD isoenzymes according to the method of Usategui-Gomes et al. (2).

The reagents (Isomune-LD) were kindly supplied by F. Hoffmann–La Roche & Cie., CH-4002 Basel, Switzerland. Serum samples, obtained from the local hospital, consisted of 50 sera with increased LD activities and 20 with normal activities. In 11 sera the measured isoenzyme activity exceeded the total activity, because of distortions of anti-

References
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