A representative of Baxter Dade Division responds:

To the Editor:

During discussions with one of the authors (G.H.W.), we became aware that the analyzer operating settings of the Stratus II® System used in this study had been altered from those supplied by the manufacturer. Because the Stratus II operating settings are set by the manufacturer to optimize the performance of each assay, we believe that the data presented in this study are not representative of the Stratus Free T4 assay performance as designed for the Stratus II System.

We have always supported the premise that control material should be selected so as to reflect the performance of patients’ specimens. Control materials and patients’ specimens are carefully evaluated during the optimization of each Stratus assay. When performing comparative studies, one must also ensure the quality of the control and patients’ specimens. Caution should be used if the samples have been stored for long periods or have been subjected to multiple freeze-thaw cycles.

After many years of experience with our products and extensive clinical trials, we stand by our claim of both the suitability and clinical utility of Stratus assays and Baxter Dade Control Materials.

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Creatine Kinase BB and Brain Lesions

To the Editor:

Estimation of brain lesion size by quantification of serum CK-BB (Clin Chem 1989;35:651–4 and 1989;35:208–9) seems of questionable value, because it is the site, not the size, of the cerebral lesion that determines neurological deficit. A pinpoint brainstem lesion can be fatal, whereas an extensive infarct in a “silent” area of the brain causes little or no obvious deficit.

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An author of one of the above-mentioned papers comments:

To the Editor:

We examined 51 patients with (a) primary or metastatic brain cancer, (b) brain infarction(s), or (c) brain confusion(s) and obtained each patient’s sera for quantification of creatine kinase isoenzyme BB (CK-BB) (Clin Chem 1989;35:651–4). We attempted to correlate the size of the brain lesion, measured by computerized axial tomography (CAT) scan, with the amount of CK-BB found in the sera.

Oftentimes patients brought to an emergency room are evaluated for brain injury. Patients with cancer are frequently evaluated (staged) for brain metastases. Usually the question concerning the physician is not the location of a brain lesion, but rather, if a brain lesion exists and, if so, how much tissue damage has occurred. Dr. Griggs is correct, however, in stating that it is the site, not size, of the cerebral lesion that determines the neurological deficit. Current state-of-the-art radiological procedures, including the CAT scan and magnetic resonance imaging, are not capable of detecting very small brain lesions (1–4) and we were hoping serum CK-BB would serve in this capacity. Unfortunately, we determined that there was poor correlation between the CK-BB monoclonal antibody kit we used to evaluate serum CK-BB and the size of the various brain lesions.

There are distinct advantages in using patients’ sera in place of radiological techniques, especially in determining the presence of brain metastases: e.g., decreased cost to the patient, and, of course, decreased exposure to radioactivity. More work is needed to clarify the conditions and transport mechanisms that can lead to leakage of brain isoenzymes into the blood (5) to successfully correlate the size of a brain lesion with the concentration of CK-BB in a patient’s serum.

References

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Measuring Cholesterol without Interference from Lipemia

To the Editor:

Interference of lipemia with cholesterol measurement procedures (1–3) may cause analytical errors greater than the current recommended bias limits of 5% or less (4), with substantial misclassification of patients into the normal, borderline high, or high cholesterol groups (5). Although fewer than 3% of serum samples from hospitalized patients are lipemic (6), the frequency of postprandial lipemia in a screened outpatient population may be higher because existing guidelines do not require fasting or diet restriction before venipuncture (7, 8).

The best approach to the problem of lipemic interference with cholesterol measurement is to select procedures that are unaffected by lipemia. We have found two procedures that are independent of lipemia: a manual procedure, Cholesterol C-system (Boehringer Mannheim Canada, Laval, Quebec, Canada); and an automated procedure with a Hitachi 737 analyzer and CHOD-PAP reagent (Boehringer Mannheim Canada). We have also investigated the underlying mechanism for such independence.

Twenty-eight consecutive lipemic sera from the Ottawa Civic Hospital, Ottawa, Canada, were visually classified according to their degree of turbidity: trace, 1+, 2+, or 3+ (creamy). Aliquots of the fresh specimens were used for cholesterol measurement with a Hitachi 737 automated analyzer. The remaining portions, stored at −20°C, were analyzed for cholesterol with the U.S. Centers for Disease Control (CDC) Abell–Kendall reference procedure (9) and the Cholesterol C-system manual procedure.

Figure 1 shows that cholesterol measurements on the Hitachi 737 correlate well with those obtained with the CDC Abell–Kendall reference method (Hitachi 737 values = 0.53 mmol/L + 0.975 × reference method values; r = 0.995, n = 28). Lipemia did not significantly interfere with chole-