Inadequate tissue oxygenation leads to a disturbance in intracellular energy metabolism and inability of mitochondria to form ATP. The accumulating AMP is readily degraded to various purine catabolites, which can cross cellular membranes and enter the circulation. Measurement of HX concentration in plasma may provide an indirect estimation of tissue reperfusion injury induced by superoxide (O_2^-) free radicals. HX is a natural substrate for XO, an enzyme widely distributed among tissues, under its own catalysis. HX is reduced from its natural dehydrogenase form towards its oxidase form (6), which produces from O_2 the superoxide free radical, a potent inducer of post-ischemic tissue injury.

Future HX measurements directly in the coronary sinus may reveal whether myocardial hypoxia accounts for the total amount of HX released after declamping. There being no simple routine methods for tissue oxygenation measurement currently available, an automated enzymatic assay for HX, a major purine metabolite, could offer an important diagnostic tool.

References
3. Sollevi A, Schmidt W, Jansson E, Bomfim V, Kaijser L. Adenine nucleotide degra

More on Methods for CK-MB

To the Editor:
With regard to the article "Clinical and Analytical Evaluation of Different Methods for Measurement of Creatine Kinase Isoenzyme MB" (Clin Chem 1989;35:130–4), I would like to make the following comments about the Seradyn CK-MB results. Diagnosis of an acute myocardial infarction is determined by several factors in addition to the absolute value of the CK-MB. This is referred to in our package literature and described graphically in our monograph.

Specifically these factors include: (a) the total CK must be abnormal for an AMI to be considered, and (b) the proportion of CK-MB must be between 4.5% and 14%. By these criteria, no AMI would have occurred in patients 4 and 24 (Table 2) because their value for total CK is normal. Patients 2, 10, 11, 12, 16, 25, and 29 all have <4.5% CK-MB, and thus would be considered AMI-negative. The total CK value for patient 7 is only marginally high, and thus an unusual CK-MB would bear reinvestigation.

This leaves only two of 32 patients whose results are in disagreement, a considerably better correlation than indicated by the authors' conclusion.

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The authors of the article respond:

To the Editor:
We have used the Seradyn CK/MB kit in our laboratory as a screening tool for myocardial damage for the past seven years. We utilize an immunoradiometric assay for quantifying CK-MB Monday through Friday and the Seradyn screen at night and on the weekends. We will usually not perform a CK-MB screen unless the patient's total CK is ≥150 U/L. However, if the physician clinically suspects an acute myocardial infarction (AMI), we will perform the screen regardless of the total CK. We have on numerous occasions diagnosed AMI's (meeting all the clinical criteria) with "normal" total CK values.

On various occasions we have telephoned the Seradyn company for technical assistance concerning our CK/MB screen. We inquired whether most of the Seradyn clients used a "number" or a "number" (i.e., 16.6 U/L) as a cutoff for a normal vs abnormal result. The Seradyn CK/MB package insert suggests that >3–4% CK-MB is an abnormal result and it lists the normal range as 5.7–16.6 U/L. We explained to the Seradyn company that we were getting numerous false-positives and false negatives using % and they suggested that we use the upper limit of Seradyn's normal range (16.6 U/L) as our cutoff value. They stated that most of their customers use Seradyn's printed normal range (rather than establishing their own). At no time did Seradyn suggest sending us a copy of the monograph or using the scheme presented in their monograph.

article (Clin Chem 1989;35:130–4) were those with discrepant values between the four assays. In retrospect, we have utilized both the Seradyn monograph and the "% CK-MB" (>4%) suggested by Seradyn for all 32 of our patients' samples. Both have resulted in numerous false-positive and false-negative results, as well as in lower sensitivities and specificities than our original cutoff value of 16.6 U/L.

We therefore continue not to favor the Seradyn CK-MB as a screening method in our institution.

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Indocyanine Green Interference in the Kodak Ektachem Determination of Total Bilirubin

To the Editor:
Indocyanine Green (ICG; Hynson, Wescott & Dunning, Hunt Valley, MD 21030), a non-toxic tricarbocyanine dye used to monitor hepatic blood flow and function (1, 2), is exclusively cleared by the liver and excreted in an unconjugated form in bile without undergoing enterohepatic recirculation (3). We recently had occasion to measure ICG and bilirubin in plasma taken from patients with significant liver disease. When present, ICG interfered with the Kodak Ektachem 700 (Eastman Kodak, Rochester, NY 14650) assay for total bilirubin. This observation led to the following study, performed according to the guidelines for interference testing established by the NCCLS (4).

We added ICG to fresh pooled plas-