tice should not be alarmed at the content of my article.

Yours sincerely,

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Laboratory Diagnosis of MI and AST Determination

Dear Sir,

I don't bother them very often, perhaps once in twenty years, so perhaps I am entitled to ask your readers for a few seconds of more than ordinary attention to this problem, discussed in two items in the October issue, with reference in turn to two papers in the July issue of the Annals.

On the face of it, all the outstanding questions can be easily resolved. Assay CK, CKMB, and/or myoglobin serially over the first few hours after admission and you will be able to achieve your diagnosis in time for thrombolytic treatment to make a difference. Aspartate aminotransferase and LDH, the old standbys, are not even in the league. The biggest outstanding difficulties are that we don't have a fully developed and nationally agreed protocol for exactly which of these analytes to choose, or how to measure them, or when: but it is clear that only management decisions remain.

All this is fine – as far as it goes, but I have a quite different problem, which starts because in my own admittedly limited experience, there are really rather few cases where the clinical diagnosis of acute MI, seen by a careful physician in the first few hours, is seriously in doubt at the time or even called into question afterwards. The purpose of these repeated and often urgent assays of CK, etc, is to document the diagnosis for later reference, or to provide justification for that same thrombolytic treatment, or evidence of its success. A more frequent truly diagnostic conundrum is how to make the diagnosis in a late-presenting case, days after the onset which may have been silent. It is too early to say that in such cases all these mini-proteins are useless, though that seems quite likely.

True, Total AST and Total LDH assays are also very blunt and imperfect weapons. As far as knowledge yet goes, the assays worth considering for delayed diagnosis of MI are LDH isoenzymes (H4 often stays up for many days) and the mitochondrial isoenzyme of AST. The demonstration that this latter remains elevated for extended periods, with an apparent half-life of about 7 days, is already over twenty years old[1] and has been confirmed more recently[2]. Don't all rush though. None of the existing techniques measures all the enzyme which is really present on either activity or mass basis[3]. (Yet more physicochemical studies are required even before field trials are planned.) My plea is only to defer the Gadarene rush to mind-made-up, managerially-convenient conformity and listen to the new science which is on its way to you.

Yours sincerely,

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