the reverse is true for patient 4. In addition, the impressive magnitude of this correlation is somewhat dampened by the very small sample size. The standard error for a correlation coefficient with this sample size (n) is 1 / √n−1, or 0.5. Even a coefficient of unity here cannot exceed twice the standard error; thus one cannot exclude the possibility that the expressed r value arose by chance, despite its magnitude. It is doubtful whether reasonable conclusions can be achieved with such a small sample size.

In addition, the poor correlation (r = 0.33) between control PCW and control LAD seems hardly avoidable given the narrow physiologic range of PCW compared with LAD, which may vary significantly with body size. It is of interest that, despite poor correlations, plasma expansion with dextran sufficient to raise PCW in the 11 patients with normal control PCW is associated with decreased PTFV1 (i.e., increased P wave “negativity”), as well as with increased LAD in 8/11 patients. Directional changes in PCW and LAD, and PTFV1 are similarly appropriate following unloading with furosemide and/or nitroprusside. That is, a decrease in PCW is associated with decreased and increased PTFV1 in 5/5 patients, despite the negative correlation (r = −0.98) of elevated control PCW with LAD.

We believe that the study design employed by the authors offers much potential in establishing relations between left atrial pressure, size, and conduction, but that the small number of studies performed as well as errors in data analysis, interfere with realization of that potential.

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References

The authors reply:

To the Editor:

Doctors Gottdiener, Di Blanco, and Fletcher are correct in pointing out that the correlation of echocardiographic left atrial dimension (LAD) with an elevated control mean pulmonary artery wedge pressure (PAWP) in our patients with acute myocardial infarction was r = −0.98, not r = 0.98. Our subsequent experience has demonstrated a poor correlation between an elevated PAWP and the LAD. These data reinforce our major conclusion that the LAD cannot be used to quantitate the PAWP before intervention.

Analysis of the data using the LAD corrected for body surface area did not significantly change any of the correlations.

After Dextran infusion, only 3 of 11 patients (27%) with acute myocardial infarction had an abnormally increased LAD, and only 3 of 11 patients (27%) had an abnormally increased P wave terminal force in V1 (PTF-V1). After furosemide and/or nitroprusside therapy, the LAD remained abnormally increased in four of five patients with acute myocardial infarction, and the PTF-V1 changed from an abnormally increased value to a normal value in only one of five patients. Therefore, the data indicate as we stated in our paper that neither the PTF-V1 nor the LAD can be used to assess the PAWP after intervention.

Of interest, a very recent paper by Josephson and associates (Am J Cardiol 39: 967, 1977) demonstrated that in patients with coronary artery disease, the electrocardiographic pattern termed left atrial enlargement was unrelated to either left atrial pressure (PAWP) or volume (LAD) overload.

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Infarct Sizing Controversy

To the Editor:

In their recent comparison of histologic and enzymatic estimates of infarct size, Roe and colleagues reported a poor correlation (r2 = 0.05) between the two approximations of infarct size; however, they observed that when the calculations were confined to the MB-CK fraction in what they call the “cardiac zone” for small infarcts the correlation coefficient was 0.94 (r2 = 0.91). These data are inconsistent with data relating enzyme changes to infarct size obtained by several observers over many years and may be explained by methodologic differences in their animal model.

La Due and co-workers, in 1954, described the close relation between peak enzyme activities and anatomic infarct size; this relation has been confirmed several times and conforms to clinical experience. The one-compartment model utilized to analyze serum CK changes was proposed to improve the correlation between enzyme changes and infarct size. Experimentally, analysis of serum CK by this model correlates with infarct size estimated by total myocardial CK depletion. Myocardial CK depletion reflects blood flow reduction and histologic evidence of necrosis. In experiments performed on conscious unmedicated dogs, the group from Harvard University showed a correlation coefficient of 0.76 between serum CK infarct size estimates and anatomic estimates; similar experiments from the Duke group showed a correlation of 0.87 between serum CK and anatomic estimates of infarct size. In man, serum CK estimates of infarct size correlate with patient mortality, postinfarction morbidity, incidence of arrhythmias, degree of ventricular dyskinesia, postinfarction hemodynamic dysfunction, and, importantly, anatomic estimates of infarct size.

Roe and colleagues, in their experiments, used a different method of calculating CK release than we originally reported. In calculating CK release (CK-R), they used the equation:

$$\text{CK-R} = E(T) + Kd \int_{0}^{T} E(t) dt$$

where E(T) is the last observed CK activity instead of the originally described form:

$$\text{CK-R} = \sum \left[ \frac{\Delta E}{\Delta t} + Kd \int E(t) \, dt \right] \Delta t$$

to perform their calculations. The form of the equation they used exaggerates the influence of the last data point and will overestimate infarct size when the last value is not close to zero. Since the total CK activity in the majority of the animals studied by Roe et al. did not return to zero (table 1), they would frequently overestimate infarct size; moreover, the studied dogs had control CK values ranging from 31 to 914 mIU/ml with 11 of the 14 animals over the normal CK for the dog (50 mIU/ml) indicating that the dogs had not recovered from the surgical procedures. For example, they calculate in dog #1 a CK-infarct size of 114.6 CK-gm-eq using the first equation above, while a recalculation using the second equation shows a value of 1.7; the histologic infarct size in this animal was 0.1, the preocclusion “control” value was 914 mIU/ml, and the final observed serum CK activity in that animal was 948 mIU/ml instead of zero. Recalculation of their data utilizing our original equation.
Infarct sizing controversy.
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