data to be too extensive to be explained only by salvage of the cells in the jeopardized zone. Concerning the data on treated patients the treatment was started 13 hours after the onset of chest pain, and it is hard to understand that the effect of treatment at this stage can be so extensive. According to the computed curves most of the irreversible damage is already done, and the cells that are candidates for salvage at this point seem to be only a small portion of the total. Yet the authors describe a reduction of infarct size from 53 CPK gram-equivalents to 27 in the reported case of trimetaphane treatment in a hypertensive patient.

The work referred to has gained much attention and in principle the hypothesis is extremely interesting. Based on the published data, however, the reader is left with serious doubts as to whether there is not an effect on the CPK activity of the already released amount of enzyme that overshadows the effect that the authors are really looking for.

We would appreciate an explanation from the authors on these two points.

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References


The authors reply:

Dr. Lorentsen and colleagues raise two related concerns. The first, namely that the prompt drop in CK, is not explicable by salvage of jeopardized tissue, fails to reflect available information regarding the elimination of enzyme from blood. It is now clear that the rate of disappearance of CK after infarction is substantially slower than disappearance after intravenous injection of enzyme, at least in part because of continued release of CK into the circulation after plasma CK has peaked. When zymosan is administered after myocardial infarction, inhibiting CK disappearance, observed plasma activity actually increases substantially even when zymosan is given after plasma CK activity has begun to decline. The decline of activity from blood is markedly decreased by even modest late release from the heart and does not produce striking deviations from linear fits of semi-log plots of CK time-activity curves. Since the observed elimination rate of CK from blood after infarction reflects not only the true CK disappearance rate but also continuing release from the heart with concomitant resupply of the vascular space, inhibition of enzyme release by an intervention would indeed cause the elimination rate to exceed that prevailing after a typical myocardial infarction.

The second concern is based on the unsupported assumption that interventions initiated relatively late will be totally ineffective. In hypertensive patients, continuing damage to the heart may occur for substantially longer intervals than in patients with infarction without hypertension. The time dependent evolution of necrosis cannot be deduced from computed curves, since such curves are based only on empirical associations between early and late plasma CK values in groups of patients. The prejudice that damage occurs early is one with which we concur, but we do not concur with the concept that damage occurs early exclusively. In addition to obser-

LV Aneurysms by M-mode Echo

To the Editor:

In their article, entitled "Detection of Left Ventricular Aneurysms by Cross Sectional Echocardiography" (Circulation 54: 936, 1976) Weyman et al. are describing what will undoubtedly become the most certain noninvasive method of detecting ventricular aneurysms. They are, however, in error when they state that "data suggesting that M-mode echocardiography might be useful in detecting ventricular aneurysms . . . have appeared only in the form of isolated case reports."

We described nine patients, eight of whom had aneurysms in-
LV aneurysms by M-mode echo.
J G Greenwald

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