Letters to the Editor

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Comments on “Analysis of Systolic Bulging: Mechanical Characteristics of Acutely Ischemic Myocardium in the Conscious Dog”


Dr. Akaishi and colleagues have provided valuable data on the relationship between systolic bulging and loading conditions in experimental myocardial ischemia. However, there are two aspects of their paper that we think merit further discussion.

In our own (unpublished) experiments, we have found that the magnitude of systolic bulge of severely ischemic myocardial segments in open-chest anesthetized dogs declines by about 50%, compared with the extent of bulging immediately after coronary artery ligation, over approximately 90 minutes. We attribute this to gradual reduction in compliance of the infarcting tissue.1 In the experiments of Akaishi et al., unloading (venesection) always preceded loading (infusion) by a time that is not stated but must have exceeded 30 minutes because infusion was given over this time. Thus, it is possible that the reduction in bulge that the authors found after infusion was due in part to time-dependent fall in compliance after coronary artery ligation.

Our second criticism concerns the acceptance by the authors on the basis of their data that shortening of the ischemic myocardial segment that occurs after ejection is a purely passive phenomenon. We agree that the exponential downstroke of the tension-length loop, which is superimposed on the upstroke and does not vary with loading, suggests that shortening during isovolumic relaxation (late systolic shortening) is passive. But shortening continues after mitral valve opening (shown in Figure 1 of the paper of Akaishi and colleagues), and therefore must extend into the phase of ventricular filling (postsystolic shortening).

Shortening of ischemic segments during early ventricular filling has been described in the intact human ventricle.2 Moreover, late systolic shortening is a well-recognized phenomenon in isolated papillary muscle and occurs both during and after ischemia.3-4 The cause of postsystolic shortening is unknown, but could be active (delayed relaxation) or passive (elastic recoil). However, the presence of latent contractile function in severely ischemic myocardium5 suggests that an active process may exist. We think that it would be premature, on the basis of the data submitted by Akaishi et al., to conclude that all of the shortening of severely ischemic myocardium is a completely passive phenomenon.

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References


Reply to the Previous Letter

The authors thank Drs. Norris, Brown, and Takayama for their letter. We agree that the compliance of the infarcted segment decreased over time. The decline in bulge at the time of volume infusion could not be caused by this, however. First, it was always related temporally exactly to the changes in loading. Second, we have now noted this phenomenon in multiple other experiments with varying loading states and different time courses. Third, the segments remained on similar and overlapping tension-length curves (Figure 4 of our paper).

We did not state, do not believe, and could never show that all ischemic segments show only passive shortening in late systole and early diastole. However, if a segment remains on a uniform exponential tension-length curve without external work, then there is no evidence, from the point of view of mechanics, of active segment function, even though shortening during late systole and early diastole may be substantial.

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