LETTERS TO THE EDITOR

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Altered Left Ventricular Diastolic Properties

It is now well-established that the left ventricular (LV) diastolic pressure-volume curve is shifted upward during pacing-induced angina. Based on comparative measurements of LV diastolic pressure-dimension relations with and without the pericardium, we have suggested that the observed shifts in the pressure-volume curve are due to changes in right ventricular (RV) and pericardial pressures.[1-3] In a recent paper, Mann et al.[4] measured pressures in both ventricles and noted that the increase in LV end-diastolic pressure in patients paced to angina was greater than the increase in RV end-diastolic pressure. They concluded that these observations preclude an important role for the pericardium.

We suggest that this conclusion derives from an undue emphasis on end-diastolic pressure to the exclusion of the rest of diastole. In their figure 1, RV mid-diastolic pressure increases about 3 mm Hg, while LV mid-diastolic pressure increases about 4 mm Hg. As we have already pointed out,[2] the greater increase in LV diastolic pressure is due to the fact that the left ventricle is thicker than the right ventricle. These changes in pre *a*-wave diastolic pressure are consistent with an increase in pericardial pressure. But how can LV end-diastolic pressure increase so much more than pericardial pressure? The relationship of a change in pericardial pressure to a change in the height of the *a* wave is apparent when left atrial pressure is considered directly. It should not be surprising to find a greater increase in peak left atrial pressure than the increase in pericardial pressure, since left atrial contraction is an active process that causes a pressure gradient across the atrial wall. Further, although the left ventricle is still in diastole during atrial contraction, the works of Rankin et al.[5] and of others[6, 7] suggest that viscoelastic effects may produce pressures well above the passive elastic diastolic pressure-volume curve.

Mann et al.[4] report that the change in RV end-diastolic pressure was "not significant." This is misleading, since their data (which they analyzed with a two-tailed *t* test) gave a *p* value of 0.051. Furthermore, since the question they asked was whether RV pressure *increased* during angina, they might more properly have used a one-tailed test. Then they would have reported a significant increase in RV pressure ($p = 0.026$).

Regardless of whether RV end-diastolic pressure increased or not their most important observation is that LV end-diastolic pressure increases more than RV end-diastolic pressure and, presumably, more than pericardial pressure. LV end-diastolic pressure may change more than pericardial pressure because atrial contraction is active and the ventricular myocardium has significant viscous properties. The mechanisms of the variable *a* wave bear further investigation but are, to a degree, separable from those mechanisms which shift the passive LV diastolic pressure-volume relation. It seems reasonable to conclude that parallel upward shifts in the diastolic portion of pressure-volume loops (e.g., fig. 4, Mann et al.[8]) may be due to increases in pericardial pressure, and that the disparate rise in end-diastolic pressures does not preclude an important role for the pericardium.

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The authors reply:

To the Editor:

We thank Drs. Tyberg, Misbach and Glantz for their interest in our study. We are familiar with their previous work concerning the influence of right ventricular (RV) and pericardial pressure on the left ventricular (LV) diastolic pressure-volume relationship, and we agree that these influences are important during acute alterations in afterload and preload. However, we feel that the results of our study, as well as the findings of others, do not support the participation of such a mechanism in the shift in LV pressure-volume relations during pacing-induced angina pectoris.

First, we disagree with Drs. Tyberg, Misbach and Glantz concerning the findings in figure 1. The rise in LV mid-diastolic pressure is clearly greater than the rise in RV mid-diastolic pressure; these pressures are clearly separated during angina, while they are nearly equal in the control state. Thus, it is not only end-diastolic pressure, but also early and mid-diastolic pressure in the left ventricle that increase with angina. Given the interdependence of the ventricles, why couldn't the rise in RV diastolic pressure result from the increased LV diastolic pressure? We previously showed that there is a small but significant increase in LV volumes with angina, and this could set the stage for the rise in RV diastolic pressure in some patients in our study. Second, the RV mid-diastolic and end-diastolic pressure actually declined in some patients (patients 16 and 21), while LV mid- and end-diastolic pressures rose substantially. Third, since some concern has been expressed concerning our statistical treatment of the data, we have consulted a statistician who suggested that we use the Wilcoxon test for paired samples, since in a situation where the data are not normally distributed and the sample is small, a *t* test is more likely than the Wilcoxon to show spurious differences (the *t* test has a greater alpha error). Using the Wilcoxon test, there was no significant difference in RV diastolic pressure before and after pacing.

Fourth, while we certainly agree that the mechanism of this altered diastolic distensibility needs further study, a substantial body of evidence favors a role for residual diastolic interaction between contractile elements in the ischemic heart. This may be due to a combination of incomplete or impaired relaxation,[1-8] altered diastolic tone,[9-14] tension prolongation during recovery from hypoxia,[15-17] and even partial ischemic contracture of some myofibrils within the distribution of the stenotic or occluded coronary arteries.[13, 14, 18-21]

Altered left ventricular diastolic properties.

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