The Myocardium in Mitral Regurgitation
A Tale of 2 Ventricles

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Muscles (from the tensor tympani to the left ventricle [LV]) are in the business of generating force. In cardiac mechanics, the innate ability to generate force independently of preload is called contractility. A central tenet of cardiology is that the prognosis of most cardiac diseases is determined in large part by the health or disarray of the myocardium conferred by this property of contractility. Before the era of molecular biology, a search for the perfect index of contractility had become a Holy Grail of cardiology. Such an index would be independent of preload, afterload, and cardiac volume and mass; sensitive to changes in inotropy; reproducible; and easy to apply. Perhaps the most accurate of these is end-systolic stiffness (the modulus of systolic stress and strain),1 but the tedium of its clinical use is so daunting that it is rarely used. Thus, although dozens of indexes have been proposed, none fulfilled all of the above requisites, and the search was largely abandoned. Thus, the cardiology world has settled on ejection fraction (EF) with all of its foibles as the preferred index of cardiac function. It measures chamber movement that would be independent of preload, afterload, and cardiac vortility had become a Holy Grail of cardiology. Such an index is available at http://circ.ahajournals.org

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The Present Study

MR obviously affects a volume overload on the LV; therefore, the LV has been the target of most studies of MR. Increased preload, together with usually normal afterload, increases LV EF in MR so that the normal EF in patients with severe MR is probably ≈70%. By the time EF is reduced to 60% or even 64%, prognosis worsens, presumably because myocardial damage causing reduced contractility has ensued.5,6 The present study by Le Tourneau et al adds to our knowledge of the pathophysiology of MR in several ways. First, it confirms and emphasizes previous observations that right ventricular (RV) dysfunction plays a direct role in MR prognosis.6 Furthermore, it provides insight into the mechanism of RV dysfunction.

To reiterate, EF is determined by preload, afterload, wall thickness, and contractility, and all 4 factors vary from patient to patient. The increased LV filling pressure in MR, together with reflexive pulmonary vasoconstriction, often leads to pulmonary hypertension, thus afterloading the RV. It is often presumed that RV ejection performance, when impaired, is due to increased pulmonary pressure. Because the geometric vagaries of the RV make calculation of systolic wall stress (thought by many to be the gold standard of afterload) extraordinarily difficult, it is rarely measured and was not measured here. However, estimated pulmonary pressure barely correlated with RV EF, suggesting that RV afterload was not a major cause of reduced RV ejection performance.

More important was the effect of MR on the interventricular septum. Reduced septal function played a major role in the pathogenesis of myocardial injury in MR.10-14 The Present Study

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patients with reduced LV and RV EF probably had contractile dysfunction of both ventricles, a finding that again raises a neurohumoral hypothesis. Because both ventricles are bathed in the same humoral soup, if catecholamines can damage the LV, they likely could damage the RV as well, perhaps as shown in the Figure.

Mitrval regurgitation creates an intricate interplay between LV volume overload, LV remodeling, RV–LV interaction, pulmonary hypertension, neurohumoral activation, and myocardial damage that should be avoided or intercepted before the LV or both ventricles become permanently damaged, points dramatically emphasized in this work by Le Tourneau et al.15

Disclosures
None.

References

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