the RV on LV performance. This compelling work sparks some new questions that deserve attention.

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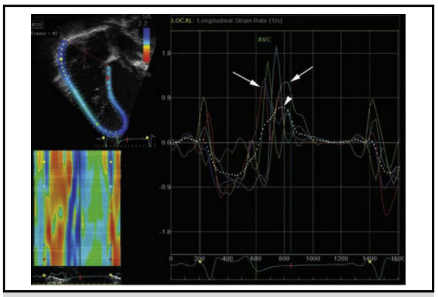
Commentary: Does right ventricular dysfunction cause left ventricular dysfunction in tetralogy of Fallot? The quest continues

William M. DeCampli, MD, PhD

Schafer and colleagues[1] present results of an analysis of left intraventricular flow dynamics (LVICF) in a group of 11 patients with repaired tetralogy of Fallot (TOF) with chronic pulmonary regurgitation (PR) and moderate increase in right ventricular end systolic volume index and right ventricular end diastolic volume index. They found that, compared with an age-matched control group of so-called normal subjects, the TOF group had abnormal flow patterns suggestive of left ventricle (LV) diastolic dysfunction. They showed that LVICF correlated with right ventricular end systolic volume index and end diastolic volume index and RV ejection fraction, suggesting the possibility of a causal relationship between abnormal RV function (in turn suggested to be due to PR) and abnormal LV diastolic function.



Tissue Doppler can detect LV dysfunction in TOF. MRI-based flow partitioning can detect this, too.

CENTRAL MESSAGE

Left ventricular dysfunction is common after tetralogy of Fallot repair. The challenge is to definitively determine the role of the right ventricle in causing the dysfunction.

Because these patients otherwise did not have conventional indications for pulmonary valve replacement, the authors suggested that LVICF measurements might be used to time earlier intervention on PR.

The authors used a method based on magnetic resonance imaging flow tracking developed by Eriksson and colleagues[2] to look at patterns of flow in the LV during diastole. These patterns are determined by the functional behavior of myocardial relaxation and recoil during diastole and in normal hearts optimize the efficiency of subsequent ejection. In effect, these patterns optimize alignment of fluid momentum to be efficiently additive to the momentum imparted to the fluid during systole. Any underlying cause of abnormal LV diastolic function will adversely

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influence this flow pattern and subsequent systolic efficiency.

It has long been speculated that abnormal RV function may be a cause of abnormal LV diastolic function in repaired TOF. This causal relationship is suggested, but not proven by associations of indices of RV function with those of LV function. A definite mechanism has not been determined. For example, in 2012 Friedberg and colleagues[3] showed that lower LV early diastolic radial and circumferential strain rates (by tissue Doppler echocardiography) were associated with greater RV volumes and greater PR in a study of 53 patients with repaired TOF and moderately increased RV volumes.[3]

In the present study, associations between LVICF and RV function indices are the basis for the suggestion that RV and LV dysfunction may be causally related. The calculated correlations should be carefully interpreted. The correlation analyses were performed using the combined cohort ($n = 22$). Although TOF patients will be scattered somewhat about the plot, the normal subjects will tend to cluster in 1 corner of the plot, almost surely yielding a significant linear correlation. Additional information could be obtained by computing the correlation within the TOF group alone (albeit it with larger sample size). Another problem (mentioned by the authors) is that the choice of normal subjects for the control group did not permit delineation of factors that could influence LV and RV function independently. Examples include the nature of the congenital heart defect, effects of surgical intervention, and conduction abnormalities. Better control groups might consist of patients who underwent a successful valve-sparing TOF repair, or patients who underwent ventricular septal defect repair.

The authors should be applauded for continuing to aggressively pursue sensitive and reliable indicators of ventricular dysfunction after repair of congenital heart defects—TOF in particular. Our quest to circumvent LV dysfunction simply by changing something about the RV, however, must rely on well-controlled studies that establish causality and a mechanism the basis on which we can target intervention. Intuitively, we believe the target is a competent and durable pulmonary valve placed early in life. It might be more complicated than that.

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