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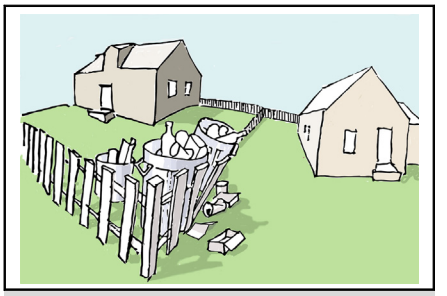
CONG

Commentary: Ventriculo-ventricular interaction: A bad neighbor brings down the neighborhood

David P. Bichell, MD



Right ventricular chaos affects the neighboring left ventricle's mechanical efficiency.

CENTRAL MESSAGE

After tetralogy of Fallot repair, compromise of the left ventricle occurs even with only modest right ventricular enlargement. Implications for pulmonary valve intervention deserve attention.

Once thought benign, pulmonary insufficiency after tetralogy of Fallot (TOF) repair starts the right ventricle (RV) down a path of certain decline but also toward being an influential bad neighbor to the left ventricle (LV). Intervening with pulmonary valve replacement (PVR) may mitigate the degeneration of both ventricles. The question "is PVR indicated?" has evolved into "when is PVR indicated?" The answer is earlier and earlier, for the sake of the whole neighborhood.

The threshold for PVR is moving earlier based mostly on measures of RV decline. A QRS duration of >180 milliseconds predicts ventricular tachycardia, and QRS duration stabilizes after PVR.[1] Thresholds for PVR based on QRS prolongation have moved from 180 toward 140 milliseconds.[2] Functional recovery of the RV after PVR is less likely if the preoperative RV end-diastolic volume is >170 mL/m^2, and that threshold is moving toward 150 mL/m^2.[2] Exercise criteria, RV mass-to-volume ratio, and other RV metrics support lowering the threshold.[3]

RV dilation, by way of a leftward septal shift in diastole, perturbs presystolic LV flow patterns and contributes to a fall in LV performance. Impaired filling momentum, RV-induced geometric changes in the LV are linked to both diastolic and systolic mechanical inefficiency.[4] LV impairment may occur early in the decline of RV performance and may join the list of reasons for earlier PVR.

Time-resolved phase-contrast magnetic resonance imaging with 3-dimensional velocity coding produces spectacular particle-tracking maps of vascular hemodynamics.[5] Schäfer and colleagues,[6] in the current issue of the *Journal*, use 4-dimensional flow magnetic resonance imaging to elegantly show a color-coded disorganization of presystolic blood flow in the LV of patients with repaired TOF, with a decline of LV mechanical efficiency early in the evolution of measurable decline of the culprit RV. Partitioning LV flow into 4 phases, 4-dimensional flow images show that patients with TOF and only mild-to-moderate RV enlargement already have reduced LV direct flow and increased residual volume when compared with unoperated controls. Do these findings support nudging the threshold for PVR earlier still?

Demonstrating LV chaos early on the timeline of RV impairment is important to think about, but questions remain of its clinical relevance and what role PVR has in restoring LV health. If PVR can reverse LV inefficiency, how much LV impairment is too much to reverse? If PVR only arrests progression of LV impairment, might this compel even earlier, preventive PVR? Whether the findings of this study affect any decision at all about PVR remains confounded by some uncertainty that pulmonary insufficiency is the culprit. The TOF repair includes ventricular septal defect closure under cardioplegic arrest. Although reasonable to expect that pulmonary insufficiency causes the LV mechanical inefficiency, the noncompliant ventricular septal defect patch and myocardial scarring from cardioplegic arrest are not exonerated, and unoperated control subjects cannot secure an indictment of the pulmonary valve alone. Schäfer and colleagues[6] have contributed a new level of understanding of the scope of influence of

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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

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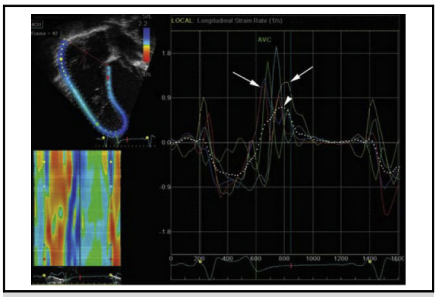
Schafer and colleagues[1] present results of an analysis of left intraventricular flow dynamics (LVICF) in a group of 11 patients with repaired tetralogy of Fallot (TOF) with chronic pulmonary regurgitation (PR) and moderate increase in right ventricular end systolic volume index and right ventricular 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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