It takes two to tango: Right ventricular failure after left ventricular surgery

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When a colleague refers a patient for heart surgery, how often do we ask, “What is the right ventricular (RV) ejection fraction?” I’ve never asked. We probably should pay closer attention, as evidenced by the article in this issue of the Journal by Couperus and colleagues.1 Quantifying RV function is not easy, and if the tricuspid valve is the “forgotten valve,” then the RV is the “forgotten ventricle.” RV geometry is complex, the afterload and preload can shift dramatically, transthoracic echocardiography is technically challenging, and even 3-dimensional echocardiography and magnetic resonance imaging may not provide precise data such as we use to assess left ventricular function and volumes. Aphoristically, not everything that matters can be measured, and not everything that is measured matters. RV function is hard to measure, but it matters. Various studies have indicated that patients with cardiomyopathy and severe RV dysfunction have a worse prognosis.2,3 RV dysfunction before mitral surgery adversely affects late outcomes,4 RV dysfunction affects outcomes after aortic valve operations and transcatheter aortic valve replacement,5 and RV dysfunction has previously been shown to predict long-term outcome in patients with heart failure undergoing surgical left ventricular restoration.6 It seems intuitively obvious. RV dysfunction can cause low cardiac output; elevated right heart pressures may cause liver and renal dysfunction with ascites and edema; and the progressive downward spiral from tricuspid regurgitation gradually creates more RV dilatation and dysfunction.

So, what did we learn from this study? The 30-day mortality was high (11%), and Couperus and colleagues1 appear to have operated on a lot of patients with RV dysfunction (39% had ≥1 parameters). The Surgical Treatment for Ischemic Heart Failure (STICH) trial had a 30-day mortality of 13.6% in patients with RV dysfunction and a 30-day mortality of 4.7% in those without RV dysfunction.7 When I was at the Cleveland Clinic, we were avoiding patients with RV dysfunction, which we considered a contraindication to this operation, and we achieved a 1% 30-day mortality.8

What didn’t we learn from this study? Since Couperus and colleagues1 have only reported 30-day results, we don’t know whether RV dysfunction impacts late survival as well. Perhaps nonfatal but ongoing RV dysfunction limits late clinical improvement and quality of life. Or, more optimistically, RV dysfunction may improve, as it does in some patients with cardiomyopathy, and survival and clinical outcomes will improve as well.3

What do we do differently because of this article? For patients undergoing left ventricular reconstruction, further testing of RV function is warranted. Perhaps those with the most advanced RV dysfunction should be turned down for conventional surgery, and transplantation or mechanical support should be considered. Since our measurement techniques are limited, however, perhaps we should use our clinical clues (liver dysfunction, renal dysfunction, severe edema and ascites that do not respond to diuretics, etc) to select patients. We need more data and studies like this. We need to pay closer attention, because in the dance between the two ventricles, it takes two to have a successful tango.
References


