Preoperative right ventricular dysfunction should not preclude surgical ventricular restoration

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The Surgical Treatment for Ischemic Heart Failure (STICH) trial concluded that adding surgical ventricular restoration (SVR) to coronary artery bypass grafting did not improve outcomes in the treatment of ischemic cardiomyopathy.¹ The trial was controversial and contradicted extensive registry data.² A subanalysis reported that preoperative right ventricular (RV) dysfunction occurred in 9% to 12% of patients and was associated with larger ventricles and worse early outcomes than seen with coronary artery bypass grafting alone.³ In this issue of the *Journal*, Couperus and colleagues⁴ report that preoperative RV dysfunction was even more frequent in patients undergoing SVR, as determined by RV fractional area change, (21%) tricuspid annular plane systolic excursion (20%), and RV longitudinal peak systolic strain (27%). Any of these was present in 39%. A higher number of impaired RV parameters worsened 30-day survival with heart failure as the cause of death. Antegrade cardioplegia was used in all patients. Couperus and colleagues⁴ concluded that refraining from SVR should be considered when RV dysfunction is present.

The cause of RV dysfunction in patients undergoing SVR and how operative technique may affect RV function can be understood by examining RV structure and function. The RV is composed of a free wall consisting of transverse muscle fibers at its base and a septum composed of helical fibers that are obliquely oriented. The transverse fibers constrict or compress to cause a bellows motion, which is responsible for about 20% of RV output, whereas the oblique septal fibers shorten and lengthen to produce 80% of RV systolic force.^{5,6} The septum is the main driver of RV function: when RV transverse fibers (free wall) are cauterized,⁷ or even removed and replaced with a patch,⁸ there is no RV dysfunction.

Septal fiber orientation affects biventricular function and explains how left heart failure begets right heart failure.^{9,10} Septal viability is best determined by gadolinium-enhanced cardiovascular magnetic resonance imaging and might be a helpful tool in predicting reversibility of function.¹¹

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Central Message

Search for the cause of preoperative RV dysfunction in potential SVR candidates. Think anatomy and pathophysiology. A cardioplegic strategy that protects the septum is crucial.

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Patients with preoperative RV failure without septal viability are not SVR candidates.

RV dysfunction is related to large hearts, and left ventricular volume reduction will improve longitudinal strain so long as the septum is not injured during surgery. Postoperative RV dysfunction has been reported in most patients after heart surgery¹² and is an expression of impaired septal function (paradoxical septal motion) that is reported in 40% to 60% of patients undergoing heart surgery.^{13,14} Keyl and colleagues¹⁵ studied RV function in surgical aortic valve replacement and transcatheter replacement. In the surgical aortic valve replacement group, RV longitudinal contraction (tricuspid annular plane systolic excursion) decreased and RV transverse contraction increased with no net loss of RV function. In the transcatheter aortic valve replacement group, there were no changes in either parameter. This is likely because of inadequate myocardial protection at operation.

It has been proposed that septal dysfunction after cardiac surgery is related to inadequate myocardial protection and that it can be prevented by specific cardioplegic techniques.¹⁶ We have also shown that antegrade cardioplegia alone caused reduction in longitudinal strain, a variable that was normal after integrated antegrade and retrograde

cardioplegia. The uninjured septum prevents RV dysfunction.^{17,18}

In the study of Couperus and colleagues,⁴ induced septal injury by inadequate myocardial protection may have worsened an already dysfunctional RV, which may explain the poor clinical outcomes. Preoperative RV dysfunction should not preclude SVR.

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