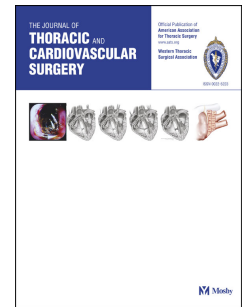


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Preoperative Right Ventricular Dysfunction Should Not Preclude Surgical Ventricular Restoration

Constantine L. Athanasuleas, MD. FACC



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Constantine L. Athanasuleas MD. FACC.
Professor of Surgery
Division of Cardiothoracic Surgery
University of Alabama at Birmingham

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.

TEXT

The STICH trial concluded that adding SVR (surgical ventricular restoration) to CABG did not improve outcomes in the treatment of ischemic cardiomyopathy.¹ The trial was controversial and contradicted extensive registry data.² A sub analysis reported preoperative RV dysfunction in 9-12% of patients and was associated with larger ventricles and worse early outcomes compared to CABG alone.³ In this issue of the Journal, Couperus reported that preoperative RV dysfunction was even more frequent in SVR patients as determined by RV fractional area change (RVFAC, 21%) tricuspid annular plane systolic excursion (TAPSE, 20%) and RV longitudinal peak systolic strain (RV LVPSS, 27%).⁴ Any of these was present in 39%. A higher the number of impaired RV parameters worsened 30-day survival with heart failure as the cause of death. Antegrade cardioplegia was used in all patients. The authors concluded that refraining from SVR should be considered when RV dysfunction is present.

The cause of RV dysfunction in SVR patients 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 responsible for about 20% of RV output, whereas the oblique septal fibers shorten and lengthen producing 80% of RV systolic force.^{5, 6} The septum is the main driver of RV function: when RV transverse fibers (free wall) are cauterized⁷ or even removed and replaced with a patch⁸, 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 CMR and might be a helpful tool in predicting reversibility of function.¹¹ Patients with preoperative RV failure without septal viability are not SVR candidates.

RV dysfunction is related to large hearts and LV volume reduction will improve longitudinal strain provided that the septum is not injured during surgery. Postoperative RV dysfunction has been reported in the majority of 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 studied RV function in surgical aortic valve replacement (SAVR) and transcatheter replacement (TAVR). In the SAVR group, RV longitudinal contraction (TAPSE) decreased and RV transverse contraction increased with no net loss of RV

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