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Editorial for : « Differential Adaptation of Biventricular Myocardial Kinetic Energy in Patients with Repaired Tetralogy of Fallot Assessed by Cardiac Magnetic Resonance Tissue Phase Mapping»

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Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart disease.¹ Repair of tetralogy of Fallot (rTOF) with right ventricular reconstruction can cause pulmonary regurgitation with right ventricular dilation and consequent right ventricular (RV) and left ventricular (LV) contractile dysfunction that predisposes to ventricular arrhythmias and sudden death.² In this context, sensitive imaging markers of RV and LV dysfunction may help guide therapy, because early detection of RV and LV dysfunction could improve selection of patients for pulmonary valve replacement.³

Cardiac magnetic resonance imaging (MRI) is considered the gold standard for evaluation of rTOF patients, and various approaches have been developed to monitor biventricular dysfunction associated with pulmonary regurgitation in order to improve detection and prediction in this condition. Four dimensional (4D) flow MRI has been shown to detect abnormal flow pattern in rTOF patients.^{4,5} The calculation of kinetic energy (KE) derived from 4D flow MRI has been proposed in rTOF patients⁶ and disturbed KE in patients with rTOF and PR has been shown in both the RV and LV.⁷ Tissue phase mapping (TPM) (time resolved 2D phase contrast MRI with three-

directional myocardial velocity encoding) provides quantification of regional biventricular three-directional myocardial velocities⁸ and is able to detect regionally abnormal LV and RV motion in rTOF patients.⁹

In this issue of JMRI, the authors use tissue phase mapping derived myocardial KE as an innovative indicator.¹⁰ The working hypothesis is that RV and LV myocardial KE may have differential adaptation mechanisms because of the presence of pulmonary regurgitation (PR) in rTOF patients. The myocardial KE is calculated in radial (KE_r), circumferential (KE_ø), and longitudinal (KE_z) directions. The proportions of KE in each direction to total KE_{røz} (%KE_r, %KE_ø, %KE_z) in left and right ventricles (LV and RV) are also calculated. The myocardial KE combines mass and velocity information as an integrated measure of the biomechanics of the myocardium. The study relies on a significant number of patients and normal controls (49 rTOF patients and 47 controls). The authors show different KE adaptation in left and right ventricle. The adaptation of the KE proportion appears to occur earlier than that of the KE amplitude, and the biventricular balance of %KE is disrupted in rTOF patients. Several KE parameters are associated with the severity of pulmonary regurgitation. These results document RV structural remodeling in rTOF patients with pulmonary regurgitation and suggest significant RV adaptation in rTOF. Myocardial KE appears to be a useful parameter for studying myocardial adaptation in patients with rTOF and may help determine the optimal timing of pulmonary valve replacement. Future studies should determine the clinical usefulness of myocardial KE for earlier detection of alterations compared to techniques currently used. A comparison with techniques such as feature tracking strain and 4D flow MRI would be worthwhile.

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