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Editorial for “The Biventricular Dysfunction and Ventricular Interdependence in Patients with Pulmonary Hypertension: A 3.0 T Cardiac Magnetic Resonance Feature Tracking Study”

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Pulmonary hypertension (PH) is a condition defined by elevated pulmonary artery pressure (PAP) with a mean pressure ≥ 20 mmHg (1). It can be either idiopathic or secondary to a broad spectrum of underlying conditions. The main concern in PH is its impact on the right ventricle (RV), ultimately leading to RV dysfunction. Through ventricular interdependence, PH can ultimately lead to the involvement of the left ventricle (LV) (2). Cardiac magnetic resonance imaging (MRI) has now become the gold standard for LV functional assessment, including ejection fraction (EF), and is therefore a cornerstone in cardiac assessment in PH patients. Although ventricular function assessment has a prognostic value in PH, the identification of parameters for the detection of early impairment – before any overt functional impairment – is stimulating the scientific community. Strain imaging, historically more an echocardiography “first-hand” parameter, has been increasingly studied in cardiac MRI and has demonstrated its predictive power as an early parameter of cardiac dysfunction in many conditions (3). In this intent, strain has also emerged as an innovative tool for ventricular interdependence assessment. Initially studied through echocardiography (4, 5), the evaluation of bi-ventricular strain through cardiac MRI is a relevant avenue for refined functional assessment of cardiac degradations in PH.

In this issue of the Journal of Magnetic Resonance Imaging, Fang et al. proposed to investigate the role of RV and LV strain in biventricular dysfunction and ventricular interdependence assessment through cardiac MRI in PH patients (6). To do so, the authors have included patients with PH, subsequently subdivided into two groups – with or without RVEF dysfunction, based on a 40% cut-off, as opposed to a control group. Patients with group 2 PH (PH associated with left heart disease) were excluded from the study, to avoid any obvious confounding factor. In their study, the authors have successfully shown that RV and LV strain were lower in patients with PH compared to controls, and as expected even more in PH patients with reduced RVEF. More interestingly, the authors have shown that RV strain was independently correlated to LV strain, supporting the concept of ventricular interdependence and the key role of strain as an early marker of LV dysfunction in PH patients. The inter-ventricular dependence was highlighted by the asymmetrical distribution of impaired LV strain with a more severe dysfunction of the interventricular septum.

These findings are consistent with the results of other rare studies on the topic (7) but go beyond by distinguishing PH patients with and without preserved RVEF. The authors also address the mechanisms of the unfavorable ventricular interdependence and LV diastolic dysfunction in PH patients: i) from an anatomical and hemodynamic standpoint through the shared interventricular septum and its progressive noncompliance resulting in direct decrease of LV filling, and ii) from a cardiac systolic-diastolic phase standpoint through a shortened diastole due to increased RV afterload resulting in reduced LV preload. The

mechanistic consequences of this augmented pressure in the RV can include LV stiffness and LV remodeling towards a D-shaped septum, leading to a prolonged systolic phase and a concomitant degradation of the LV diastolic function. The correlations of mean PAP with all three components of LV strain confirmed the progressive global LV dysfunction: longitudinal, radial and circumferential strain.

It remains that PH patients in Fang et al. were mostly from the PH subgroup of pulmonary arterial hypertension patients (group 1). It would be interesting to investigate the biventricular interplay using strain cardiac MRI in the four other groups of PH patients, including in PH patients due to left heart disease in which altered hemodynamics might generate different strain patterns. Generally, further investigations are warranted to fully understand the fluid-structure relationship between flow energetics and myocardial strain. Recent advances to estimate PAP using 4DFlow MRI (8) appear very complimentary to strain measurements. Their combination allows to envision a fully non-invasive diagnosis, removing the need for right heart catheterization to identify and characterize PH.

Finally, measuring strain using non-invasive cardiac MRI holds potential for monitoring and tailoring novel therapeutic avenues for PH patients, such as specific types of tyrosine kinase inhibitors (TKIs). Although PH has been reported secondary to several TKIs cancer treatments, the promising results of the IMPRES trial (9) using imatinib, a unique TKI lowering pulmonary vascular resistance, to mitigate bi-ventricular remodeling encourage further investigations in PH patients. Interestingly in this trial, LV global and septal circumferential strain were the strongest correlates of cardiac improvement linked with the treatment. The role of cardiac MRI to support the evaluation of such novel therapies could be central, leveraging both strain and flow measurements.

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