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EDITORIAL FOCUS

Assessing right atrial function in pulmonary hypertension: window to the soul of the right heart?

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Assessing right ventricular (RV) function is a fundamentally different task from assessing left ventricular (LV) function as the RV and LV have distinct cardiodynamics. RV contraction is sequential, peristaltic, and mainly longitudinal compared with LV contraction, which is uniform with twist and torsion. Under volume or pressure overload, the RV becomes more uniform in contraction (3, 4). Physiologically, the right atrium (RA) contributes to pulmonary hemodynamics by modulating RV filling through the interplay of atrial reservoir, conduit, and contractile (booster pump) function (5). Intrinsic atrial properties affect each phase of atrial function: atrial relaxation impacts the reservoir component, atrial chamber stiffness the conduit component, and atrial contractility the pump component. Deformation analysis is increasingly used to measure atrial strain and strain rate with the goal of better understanding the overall function of the atrium and the contributions of each component toward atrial function.

RA strain is more sensitive and an earlier predictor of RV pressure or volume overload (6). Thus, any condition with an adverse effect on the RV will also inherently reduce RA function before RA size changes. Reduced RA strain is considered a marker of reduced RA reservoir and conduit functions, even when RA contractile function is preserved. Pulmonary hypertension (PH) is a disease with high mortality; so early diagnosis is critical for starting therapies that can improve outcome (2). In early stage PH, the RV responds to the high afterload with increased contractility and little or no change in RV size (7).

Echocardiographic application of RA speckle-tracking strain has been reported in many studies, including PH (1). However, accurate echocardiographic assessment of RA strain is not always feasible, mainly due to ultrasound window limitations, specifically in patients with PH. Among other diagnostic imaging tools, cardiac magnetic resonance (CMR) provides excellent visualization of the RA with high spatial resolution and wide field view. Feature-tracking algorithms have been adapted to measure RA strain using standard CMR cine images, permitting highly reproducible quantitative assessment.

In this issue of the *American Journal of Physiology,* Tello et al. (8) report the correlation of cardiac CMR feature tracking-derived RA phasic function with CMR feature tracking-derived RA phasic function with elastance (E_{ed}) and RV-arterial coupling [end-systolic/arterial elastance (E_{es}/E_a)] from invasive RV pressure-volume (P-V) loops. The authors prospectively included 54 patients with severe PH and excluded patients with atrial flutter or fibrillation. P-V/Swan-Ganz catheterization was performed 1 day after CMR imaging. In addition to conventional CMR measurements, RA myocardial feature tracking was performed to examine the association of RA phasic function, including RA reservoir and passive and active strain, with indexes of RV function. The authors are to be commended for a meticulously performed study and their effort to uncover alterations in RA phasic function in patients with PH. They demonstrate that impaired RV diastolic function appears to be the dominant determinant of altered RA phasic performance, which in turn contributes to backward venous flow and systemic congestion. They directly measured load-independent RV function via the acquisition of RV P-V loops with the assessment of RV diastolic stiffness and afterload and described RV-arterial coupling using gold-standard methodology (CMR imaging and P-V/Swan-Ganz catheterization).

Efficient right heart performance depends on optimal RA-RV coupling, so RA dysfunction and noncompliance may reduce ventricular output. To maintain stroke volume in PH, the RV adapts through either increased contractility (homeometric autoregulation) and/or increased volume (heterometric autoregulation). Interestingly, the present study found the impairment of RA phasic function to be predominantly related to alterations of RV lusitropy rather than RV contractility or coupling. The study also showed a significant correlation of RA phasic strain with RA size and inferior vena cava diameter, indicating that the loss of reservoir function leads to backward venous flow. Thus, RA phasic function is altered in relationship to impaired diastolic function of the chronically overloaded RV and contributes to backward venous flow and systemic congestion. Assessment of both changes in contractility and volume, as well as indexes of phasic RA function derived from longitudinal strain, is a significant strength of this study. In addition to the limitations that the authors highlight in their article, it is also important to note the strong association between RV and RA function. Therefore, an assessment of RV global longitudinal strain may provide additional information on right heart function, as such measurements are becoming more routine with CMR feature-tracking algorithms.

There are a few important differences in a strain values between the LA and RA that require further characterization across different disease states (Fig. 1):

1. The RA has higher reservoir strain values than LA. As a reservoir, the RA atrium stores caval venous and the LA stores pulmonary venous return during ventricular contraction and isovolumetric relaxation. Physiologically, the RV endocardial layer (the main source of strain-derived power)

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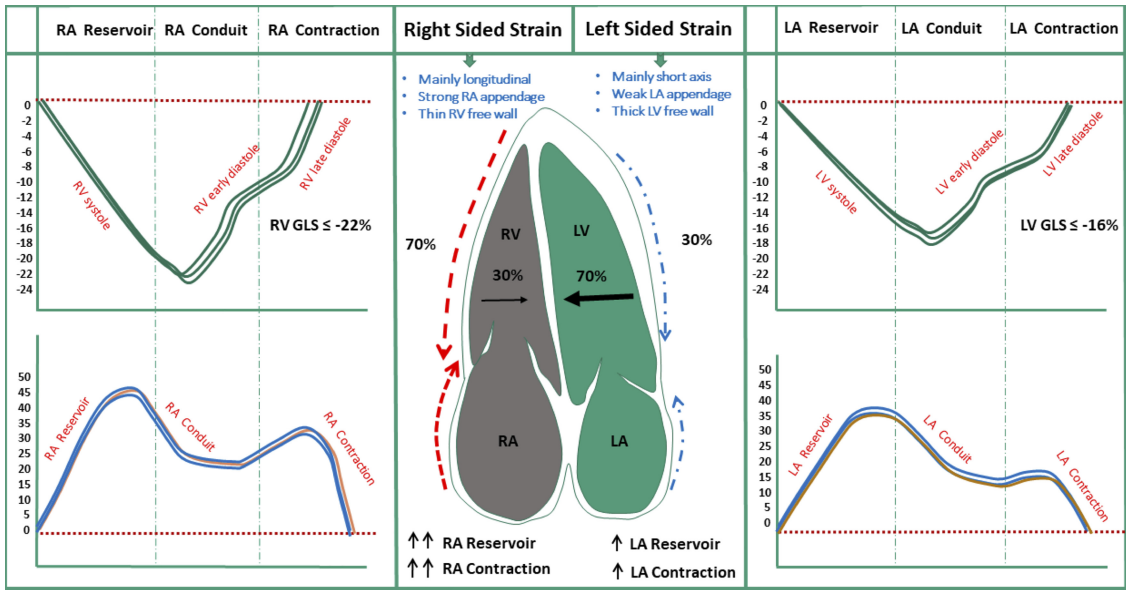

Fig. 1. Differences in right and left heart function by strain imaging. Notably, the right atrium (RA) has higher reservoir strain values than left atrium (LA), RA conduit strain values may be similar to LA strain values, and the RA has higher active contraction strain values than the LA. RV, right ventricle; GLS, global longitudinal strain.

is primarily comprised of longitudinal muscle loops; therefore, shortening of the RV is much greater longitudinally than radially. This is in contrast to the LV, which has a helical structure formed by ascending and descending obliquely oriented loops. Thus, the longitudinal shortening of the lateral tricuspid annulus toward the apex in systole is much higher than the shortening of the lateral mitral annulus. Therefore, the dragging effect of the RV to the RA is higher, and the RA will expand more in diastole (atrial diastole/reservoir phase) than the LA.

2. RA conduit strain values may be similar to LA strain values. Conduit function refers to the phase of passive blood flow from the atrium into the ventricle. As direct strain measures for conduit function are not possible, it makes physiological sense to use a concept of volume for this function, where conduit function is equivalent to the volume obtained from subtraction of the reservoir volume value from the atrial contraction phase divided by stroke volume. As strain values are more vulnerable to artifact than volumes, any inaccurate measurement in the reservoir or contraction phases can affect conduit strain values.
3. The RA has higher active contraction strain values than the LA. Anatomically, the RV has a thinner free wall than the LV. Moreover, it has a larger and more muscular RA appendage than the LA appendage. Therefore, it would be expected to be easier for the appendage to pull the RV free wall during RA contraction compared with the effect of a small and less muscular LA appendage against a thicker LV wall.

Atrial strain is a sensitive tool for detecting atrial dysfunction in the setting of ventricular diastolic dysfunction. To date, the assessment of RA function by CMR feature tracking has been largely confined to research studies. Feature tracking may add value to existing CMR techniques for the noninvasive functional assessment of right heart function. The results of Tello et al. (8), along with the findings from earlier studies (1), support the concept that RA strain is a useful parameter to be part of the management and follow-up of patients with PH. Correlation of feature tracking derived RA strain with clinical outcomes in PH will be key to establishing its prognostic value.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

F.A., S.R., and S.K. drafted, edited, revised, and approved final version of manuscript.

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