EDITORIAL

Challenging the Right Ventricle: The Ups and Downs and Ins and Outs of Strain

Desafiando al ventrículo derecho: los altibajos y pormenores del strain

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The left ventricle (LV) may be perceived as the dominant heart chamber but, as is often the case, bulk is often associated with simpleness. The left ventricular chamber can simply be modelled as a prolated ellipse - or a bullet. Left ventricular remodelling is divided simply into volume loaded or eccentrically remodelled as in aortic and mitral regurgitation vs. pressure loaded, with concentric remodelling as in aortic stenosis or systemic hypertension. Either way, with the exception of a varying degree of sphericity, the basic shape does not dramatically change.

The right ventricle (RV), on the other hand, while displaying far less brawn than the LV is actually infinitely more complex. It must eject the same volume as the LV (in the absence of shunt), yet only has one fifth of the mass. Adding to the complexity, due to the right ventricular shape, its remodeling process is geometrically far less simple. (1) Of course, it hypertrophies and dilates, but the pattern is considerably challenging to predict or describe. Add to this the shared interventricular septum and the muscle fibers that the LV wraps around the right ventricular apex and we have a mathematical modeler's life challenge. (2)

So, how can we describe the RV when it is faced with pathology? The first thing to recognize is the difference between normal and abnormal. This was the main goal of establishing "cut-off" values through guideline documents. (3) But here again we see, as was demonstrated in Del Castillo et al.'s paper published in this issue of the Journal, (4) as well as in others, there is considerable overlap between so-called normal values and abnormal values. (5) Cut-off points are best viewed as values to suggest abnormality, as opposed to confirm normalcy. The next thing is to recognize that not all conditions will provoke the same kind of reaction. We know that clinically, volume overload conditions such as atrial septal defect (ASD), behave differently clinically than pressure-loaded conditions such as pulmonary hypertension, so it is likely their effects on the heart should be different.

With that in mind, Del Castillo et al. approached both these goals by describing the different ways of adaptation or maladaptation in a significant sample of volume-loaded patients with ASDs and pressure loaded patients with pre-capillary pulmonary hypertension secondary to schistosomiasis, comparing them with normal subjects. A number of interesting observations were made through their results. Firstly, they demonstrated that the LV is compromised physically by the dilated RV through compression and that its systolic function is somewhat reduced - not by considering LVEF- but when global longitudinal strain is used, likely because of the shared septum. As such, the RV-LV interaction is more than just compression from inter-chamber pressure gradients. Second, they demonstrated that the pressure overloaded ventricles demonstrated more severe dilatation than the volume loaded ones, as well as more significant systolic dysfunction when traditional parameters of tricuspid annular plane systolic excursion (TAPSE), S' wave and fractional area change (FAC) are used. The authors point out what was stated above: despite significant remodeling and dysfunction, many right ventricular pressure overload (RVPO) subjects had values within normal limits. They suggest that this might be related to presence of significant tricuspid regurgitation (TR) (data not shown), but in pulmonary arterial hypertension (PAH), the basal segments are often the last to fail. The finding of a small LV, reduced FAC and severely elevated systolic pulmonary artery pressure (sPAP) suggest clinically advanced PAH. In other words – normal does not connote normal, but abnormal is very bad.

There are few data comparing right ventricular remodeling and function with differing etiologies of pulmonary hypertension. Anecdotally, post-capillary etiologies result in a relatively preserved right ventricular shape, with dysfunction presenting only very late, or with concomitant pulmonary artery reactivity as in mitral stenosis. In addition, Grapsa et al., studied right

REV ARGENT CARDIOL 2016;84:511-513. http://dx.doi.org/10.7775/rac.V84.i6.10032
SEE RELATED ARTICLE: Rev Argent Cardiol 2016;84:556-562. http://dx.doi.org/10.7775/rac.v84.i6.9467

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ventricular remodeling in subjects with pulmonary hypertension related to PAH, chronic thromboembolic pulmonary hypertension (CTEPH) and mitral regurgitation (MR). Mitral regurgitation resulted in the least degree of right ventricular enlargement and dysfunction, with significant differences noted between PAH and CTEPH groups in a variety of parameters, including tricuspid leaflet tenting. (6) Mafessanti et al. elegantly demonstrated the loss of longitudinal motion in the post-operative state, with a more neutral impact on longitudinal strain or FAC – in part related to paradoxical septal motion. (7) They did not, however, look at radial strain, or inward motion. So it is clear that the RV responds to different challenges in a diverse manner, but also that we must alter our echocardiographic assessment by condition, and choose which parameters perform best for each pathology.

In the past decade, the development of novel analytic tools such as 2D speckle or feature tracking has allowed us to be able to do analyses that we couldn't in the past. The lack of angle dependence with this technique has overcome one of the principal limitations of Doppler-derived strain. In addition to the mainstream use of global longitudinal strain to assess left-sided conditions such as chemotherapy-induced cardiomyopathies (8), right ventricular free wall strain has proved valuable in predicting survival in conditions such as PAH (9). Using this technique, Del Castillo et al. were able to study the longitudinal and radial strain component of the RV in different conditions. First, one notes that radial strain is considerably lower in the RV as compared with the LV. Second, the strain values are, in fact, greater in right ventricular volume overload (RVVO) and RVPO subjects. This is consistent with the findings of Hayabuchi et al., (10) which showed greater longitudinal and circumferential strain in children with ASD and greater radial strain in tetralogy of Fallot subjects, as compared with controls. Smith et al., however, using 3-D speckle tracking, found that circumferential strain is reduced in PAH as compared with controls. (11)

The cause and the significance of this higher radial strain values in some RVPO patients is still unclear, though it is incumbent on us to reflect on the limitations of the technique used to study this. Most speckle-tracking algorithms were developed for the LV and have been "forced upon" the RV. The right ventricular wall is considerably thinner than that of the LV so it has fewer speckles for the algorithm to track – though less of an issue in pulmonary hypertension. This may become more of an issue in a thin-walled subject when insonating from the apical view, as the probe's lateral resolution is not as great as its axial resolution. It is for this reason that radial strain is more often performed on the LV from the parasternal short-axis view.

The authors detail in their introduction the complex relationships between the LV and RV with the interaction between multiple layers of muscle fibers, intertwined and interacting. The LV contracts towards a centroid of the chamber, with the radial motion adding significantly to this motion; the RV contracts much more in a base to apex motion. While the right ventricular myocardium does demonstrate deformation along its radial axis, the contribution towards right ventricular ejection is not well known, though we know that reduced radial strain connotes severe right ventricular dysfunction (11). The authors' data suggests a proposition that the decrease in longitudinal function might be maladaptive, while the increase in radial function might be adaptive.

So how can we reconcile the finding of decreased longitudinal strain with increased radial strain in the overloaded RV, and why was it seen in their patients and not in those of Smith et al.? In the LV, we know that longitudinal fibers both demonstrate reduced function and predict outcomes earlier in ischemia and more consistently than circumferential fibers in chemotherapy-induced cardiomyopathy, hence the use of GLS to detect early disease states. (12) One might expect this phenomenon to be magnified in the RV since the longitudinal fibers do the lion's share of the work with the base to apex motion. In PAH, circumferential fibers are shared with the LV, so they are protected from RVPO. When longitudinal fibers fail, an adaptive mechanism could be a greater contribution from the left ventricular fibers that have to do less work because of the underfilled LV, get more stretched as they wrap around the dilated RV and therefore via Frank-Starling, have increased contractility. Other factors to consider include the ubiquitous constant of time, and that of disease process. We do not know at what point Del Castillo's patients are in their disease process – compensated vs. decompensated. Perhaps as the disease progresses, radial strain will fall. Finally, we don't know whether schistosomiasis patients evolve or respond to pressure overload in identical manner as other PAH subjects.

Piece by piece, fiber by fiber, the RV is revealing its secrets. Del Castillo et al. may have provided another clue. To address the complex issue of adaptation, serial studies in pulmonary hypertension patients will be required to see the timeline of changes in strain -both longitudinal and radial. Three-dimensional strain can look at the complex interactions of different types of strain, including torsion and area strain. (11) In addition, ultrafast echo or magnetic resonance imaging will help us move beyond anatomical relationships to clarify the complex functional interrelationship between the LV and RV.

Conflicts of interest

Dr. L. Rudski declares a minor holding of stock in General Electric, outside of a managed portfolio.

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