Right ventricular suction: an important determinant of cardiac performance

André La Gerche¹,²* and Piet Claus²

¹Baker IDI Heart and Diabetes Institute, 75 Commercial Road, Melbourne, VIC 3004, Australia; and ²Department of Cardiovascular Sciences, KU Leuven, Leuven, Belgium

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This editorial refers to ‘The role of elastic restoring forces in right-ventricular filling’ by C.P. Del Villar et al., pp. 45–55.

It has long been stated that researchers can be divided into lumpers and splitters. With respect to cardiac physiology, there is a simple appeal in lumping all of the inter-related factors together and assessing global measures of cardiac performance. On the other hand, splitting the cardiac cycle into its individual mechanical properties provides an opportunity to identify specific determinants of global function. Del Villar et al. focus on the right ventricle (RV) and split this chamber’s filling properties into active and passive components and, in doing so, identify RV restoring forces as being critical to overall cardiac performance.¹ RV restoring forces act during the very earliest phases of diastole, but are determined by afterload and contractile function of both the RV and the left ventricle (LV). It would seem that the splitters have identified a key index of function, which would please the lumpers in their search for a measure which impacts globally.

The concept of early diastolic suction has been the source of debate and a clear definition of this phenomenon has proved elusive.²,³ Defining suction as performing work to draw blood into the ventricle is only consistent with a negative transmural pressure, relative to intra-pericardial pressure.⁴ However, Del Villar et al.¹ have taken a different approach and calibrated against atmospheric pressures, fixed to zero pressure in their model. It is unlikely that this would affect the conclusions of this study, which focuses on acute changes during one single experiment, but does introduce challenges for repetitive measurements in chronic experiments. Del Villar et al.¹ established the existence of early diastolic restoring forces in the RV in a number of different conditions. Interestingly, they demonstrated that RV restoring forces were stronger than in the LV, implying a greater role in filling the RV than for the LV. This hints at the fact that the RV uses more mechanisms to adapt to changes in loading conditions (both pre- and afterload), similar to changes seen in systolic RV function with acute RV loading.

It is becoming increasingly clear that RV function is a critical determinant of global cardiac function and clinical outcomes. Not only is RV function an important determinant of outcomes in pathologies affecting the pulmonary circulation,⁵ but it is also an important prognostic marker in conditions that have traditionally been regarded as primarily left ventricular pathologies such as congestive heart failure⁶,⁷ and acute myocardial infarction.⁸,⁹ This is likely due to the fact that the RV sits ‘upstream’ of the pulmonary circulation, left atrium, LV, and systemic circulation such that the afterload of the RV is an accumulation of increased filling pressures from any of these components. In addition to this ‘in series effect’ by which vascular pressures are transmitted up the chain, RV function can be impaired as a result of direct ventricular interaction. Using a derived index of RV geometry, Del Villar et al. demonstrated that an important adaptive response to acute RV failure is a septal shift towards the LV which enables the RV to reset to a larger zero-pressure volume (V₀), thereby preserving RV suction (Figure 1). There have been a number of studies which have demonstrated that RV failure causes a septal shift which impairs early diastolic filling of the LV,¹⁰,¹¹ but the current data of Del Villar et al. place an interesting twist on this relationship by demonstrating the benefits that a flatter interventricular septum has on RV function. Indeed, this represents a major mechanism for acute RV adaptation to afterload. In the normal RV with its crescent shape geometry, contractility and afterload are such that the contraction results in end-systolic volumes (ESV) below the zero-pressure volume (V₀) and the myocardium builds up elastic forces later to be released as restoring forces resulting in good suction (Figure 1). An acute increase in RV afterload and/or decrease in contractility result in higher ESV, therefore not falling below V₀ anymore and prohibiting the myocardial restoring forces to occur (Figure 1). Here, the septal shift comes in as a major mechanism of adaptation. A leftward shift of the septum enables the RV to increase V₀ and maintain RV suction, but at the expense of a ‘squashed’ LV with a lower LV V₀ and a loss of suction in that chamber. Thus, as a result of pericardial constraint, acute changes in load will have a significant impact on both RV and LV suction, with a balance needing to be found. With chronic compensatory adaptation, improved RV contractility enables ESV to again fall below V₀ without a major shift in the septum. Thus, the impact on the LV is less and overall cardiac performance is improved (Figure 1). In the acute interventions studied by Del Villar et al., the resetting of V₀ with concomitant changes in ventricular geometry (both LV and RV) is highlighted as a major mechanism of adaptation, but the potential molecular remodelling contributing to this process is not considered. With the progression towards heart failure, both changes in titin expression and extracellular matrix will influence this resetting.

* Corresponding author. Tel: +61 3 8532 1111; fax: +61 3 8532 1100, Email: andre.lagerche@bakeridi.edu.au

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remodelling could differently affect the elastic restoring forces (compression of the tissue, i.e. at volumes below $V_0$) and the passive stiffness (stretching of the tissue, i.e. at volumes beyond $V_0$), changing or even disrupting the relationship between both components observed in normal myocardium. It could be expected that chronic adaptations in tissue properties in response to load will contribute to changes in structure, $V_0$, and suction.

Heart failure represents the clinical symptoms which result when the heart is unable to meet the metabolic demands imposed upon it. Most commonly, patients experience symptoms during physical exertion when the metabolic demand of the working muscles requires an increase in cardiac output. Data of Del Villar et al. remind us that cardiac output is only as good as the worst ventricle and it would be intriguing to know the extent to which RV restorative forces enhance or constrain cardiac output during exercise. Nonogi et al. used direct invasive LV catheterization to demonstrate that early diastolic pressures fell during exercise to become increasingly negative relative to left atrial pressure, thereby increasing LV suction. It is likely that the restorative forces described by Del Villar et al. in the RV would be of similar or greater importance during exercise. A non-invasive means of assessing this process would be ideal, and there are a number of echocardiographic and magnetic resonance RV measures which may be suitable surrogates. RV isovolumic relaxation time, for example, is relatively graphic and magnetic resonance RV measures which may be suitable. This process would be ideal, and there are a number of echocardio-

**References**


