

Despite these comments, we strongly agree with the authors: evaluation of RV function in ARDS is one of the cornerstones of the care and support of such patients, and RV protective measures such as prone position, inhaled nitric oxide, and extracorporeal membrane oxygenation could improve prognosis. Measurement of RV free-wall strain could help identify an early RV dysfunction in ARDS, rather than RV systolic impairment markers.

The authors have disclosed that they do not have any potential conflicts of interest.

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The authors reply:

We thank Petit and Bidar (1) for their insightful comments on our article (2), recently published in *Critical Care Medicine*. Our aim was to identify a common right ventricular (RV) phenotype that associated with mortality, such that RV protective measures trialed in this subgroup may improve outcomes. The problem lies in defining this phenotype: should we use RV dilation, systolic impairment or both to classify RV dysfunction (RVD)?

American and British Echocardiography Societies and numerous critical care studies have defined RVD through RV systolic impairment (low RV fractional area change [RVFAC], tricuspid annular plane systolic excursion, and S') with no consideration of RV size. However, as Petit and Bidar (1) highlight, due to the complex geometry of the RV, these parameters have been demonstrated as inaccurate markers of RV failure (3). In our study, patients with RV systolic impairment “without” RV dilation had a low mortality rate. This may be because the degree of RV systolic impairment was overestimated through using RVFAC when RV size was small. Newer markers of RV systolic function, such as RV free wall longitudinal strain, demonstrate promise (4), but are not routinely measured in current clinical practice.

If not RV systolic impairment, then what of RV dilation? Mekontso Dessap et al (5) demonstrated that RV dilation (RV:left ventricular end-diastolic area [LVEDA] > 0.6) with septal dyskinesia (termed acute cor pulmonale) did not independently associate with mortality in over 700 prospective acute respiratory distress syndrome (ARDS) patients. Our results support this:

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the mortality rate of patients with RV dilation was low, perhaps as preserved RV forward flow maintained left ventricle and thus cardiac output. Severe RV dilation (RV:LVEDA > 1) was associated with an increased mortality rate in both cohorts and, however, was comparably much rarer—with a prevalence of 5–7%, limiting its clinical utility. Thus, the current European Society of Intensive Care Medicine consensus definition of RVD includes RV dilation but coupled with evidence of systemic congestion (6). The latter, however, may be difficult to define!

Importantly, our study was the first to demonstrate a marked increase in mortality when RV dilation and RV systolic impairment were combined. This made pathophysiological sense: the inability to maintain RV forward flow in the setting of an already dilated RV may precipitate shock, organ dysfunction, and death. Furthermore, the inaccuracies of measuring RVFAC may be mitigated in the setting of a dilated RV.

So, could RV dilation with systolic impairment be the common RV phenotype that independently associates with mortality from ARDS? As highlighted in the article and the comments by Petit and Bidar (1), the limitations of this pragmatic, retrospective study mean that these findings require prospective validation, in larger, unselected coronavirus disease 2019 (COVID-19) and non-COVID-19 ARDS cohorts. The role of transesophageal echocardiography and of RV free wall longitudinal strain in identifying a prognostic RV phenotype should also be elucidated.

Last, we would like to add that although all patients did not receive prone ventilation, proning thresholds were rigidly protocolized according to an Fio_2 requirement of greater than 60% and not according to work-flow demands.

In conclusion, we propose to “bridge the gap” between groups using RV dilation and others using RV systolic impairment in isolation to define RVD: consider using both together!

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