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## Right Ventricular Longitudinal Strain

A New Prognostic Tool for COVID-19?



We have read with interest the recent paper by Li et al. (1) regarding the prognostic value of right ventricular longitudinal strain in patients with coronavirus disease 2019 (COVID-19) (1). As the morbidity and mortality of the ongoing pandemic COVID-19 are posing a serious threat to health care systems all over the world, the urge to find prognostic factors to better stratify patients and offer an adequate level of medical assistance to everyone is of the utmost importance.

However, we have some remarks, both from a clinical and methodological point of view.

First, the authors report a remarkable incidence of deep vein thrombosis (DVT) (41% of the population), with surprisingly no documented cases of pulmonary embolism (PE). We wonder how the diagnosis of DVT and PE was performed (i.e., was there an extensive screening protocol for both DVT and PE?) and how these findings were interpreted by the authors. Do the authors think that eventually undiagnosed or sub-clinical PE events may explain right ventricle (RV) echocardiographic parameters modification in patients with bad prognosis?

A methodological remark regards the significant variability of the timing of the echocardiographic study, with an interquartile range of 3 to 10 days between admission and examination. We wonder if this could have affected the results, possibly increasing the prognostic values of RV dysfunction for tests performed during the detrimental phase of ARDS. Moreover, it should be stated if echocardiography was performed during invasive or noninvasive ventilation or in oxygen, as it is well known how ventilation itself could significantly modify echocardiographic parameters, including RV longitudinal strain (2). This could represent a potential bias, as severe patients were more likely to undergo ventilation support, a condition that possibly makes RV echo parameters just innocent bystanders.

Finally, the proposed cutoff values for right ventricular longitudinal strain, (RVLS) tricuspid annular plane systolic excursion (TAPSE), and right ventricular fractional area change (RVFAC) are largely within the normal range in healthy subjects. We wonder how the authors interpreted the biological plausibility of their findings. Is it possible to speculate that the active phase of the disease leads to a form of “overload” of the RV, where the inability to

overcompensate (resulting in increased RV systolic function parameters) carries a worse prognosis?

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Cardiovascular Imaging* [author instructions page](#).

## REFERENCES

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## THE AUTHORS REPLY:



We appreciate the comments by Dr. Fukui and colleagues about our paper (1), and we fully agree with some of their views. However, there are some issues that need to be clarified and discussed.

We acknowledge and agree that right ventricular (RV) function is closely linked with the afterload. In our cohort study, 40 (34.2%) patients developed acute respiratory distress syndrome (ARDS), which contributed to increase RV afterload. Furthermore, both ARDS and right ventricular longitudinal strain (RVLS) were found to be associated with mortality, consistent with the previous study (2).

It is difficult for us to evaluate the right ventricular to pulmonary artery (RV-PA) coupling by measuring a ratio between tricuspid annular plane systolic excursion and pulmonary artery systolic pressure (PASP), as suggested by Cavalcante (3). Noninvasive PASP measurement depends on the presence of tricuspid regurgitation (TR), and there were only 61 patients (50.8%) had interpretable TR jet signal in our study. Among them, the majority was mild or trivial TR, which may affect the accuracy of PASP assessment. As the right heart catheterization was not available in our designated treatment hospital, PASP derived from