whom 145 had some form of left ventricular (LV) dysfunction (inclusive of LV systolic/diastolic dysfunction) as well (Fig 1 in the article). Hence, it appears that the majority of patients in the study group had "biventricular" dysfunction, and it is unclear whether primary LV dysfunction led to RV dysfunction or vice versa? Second, the primary end point of 28-day mortality rates may potentially be within the "early-phase" of sepsis, when biventricular failure is more likely to prevail.[2] Third, since echocardiographic parameters are dynamic and may change continuously in patients with sepsis, both contradictory[3] and similar findings[4] to this study have been published. Fourth, it is unclear whether the mortality rate associated with RV dysfunction was due to the initial presentation of RV dysfunction or the persistence of RV dysfunction despite treatment. A follow-up echocardiogram to assess whether the RV dysfunction was transient and resolved after treatment would have helped clarify whether recovery of RV dysfunction leads to better outcomes in patients who present with RV dysfunction. Fifth, it has been established in animal models that the RV dysfunction seen in sepsis is due to endothelial damage and cytokine release rather than isolated pulmonary artery hypertension.[5] Hence, incorporation of other biomarkers along with echocardiographic parameters may help better delineate this sepsis-induced cardiomyopathy. We believe that future integrated precision medicine-based approach that would incorporate echocardiography, clinical parameters, biomarkers, cardiac enzymes, epigenetics, and genomics would help not only to differentiate RV dysfunction in sepsis as an epiphenomenon but also would help to identify various subphenotypes of sepsis-induced myocardial dysfunction in need of distinct clinical and therapeutic approaches.

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Response

To the Editor:

We thank Pal et al for raising several important questions regarding septic cardiomyopathy. They note that most patients with right ventricular (RV) dysfunction had concomitant left ventricular (LV) dysfunction and that the challenge of determining if one ventricular dysfunction preceded the other. We observed substantial overlap among LV systolic dysfunction, LV diastolic dysfunction, and RV systolic dysfunction, which is consistent with previous comparable reports of early septic cardiomypoathy.[1-3] We agree that our data are unable to answer whether LV dysfunction preceded RV dysfunction; however, given the distribution of overlapping and isolated ventricular dysfunction, it is clear that there is no single pathway for the cascade of organ failure that is seen in septic cardiomyopathy. Pal et al also comment that 28 days may be within the early-phase of sepsis. Although we disagree with the designation of 28 days as early-phase sepsis, we agree with the need for long-term outcome assessments in critically ill patients, who may incur complications, such as pneumonias, in the convalescent phase of sepsis.

We feel obliged to address the concern about seemingly contradictory findings of echocardiography in sepsis. The cited publication studied the ratio of RV basal diameter to LV end-diastolic diameter, which may be less indicative of systolic function and more indicative of loading conditions than the parameters studied in our article.[4] The two other large studies that investigated comparable measurements of contractility (tricuspid annular plane systolic excursion, fractional area change, free wall strain) in similar populations have demonstrated comparable

associations between RV systolic dysfunction and mortality rates.[2,3] Regardless, the observation that echocardiographic parameters change in the critically ill patient is absolutely correct and should be emphasized when the septic cardiomyopathy literature is being interpreted.

Determining causality in the setting of septic multiorgan dysfunction syndrome is challenging. It is unknown whether ventricular dysfunction is a primary contributor to death, or if it is an epiphenomenon of increased pulmonary hypertension or myocardial depression from circulating cytokines. Although we suspect that most of patients in our study did not have significant RV dysfunction at baseline, this suspicion remains unsubstantiated. We agree with Pal et al that assessment of biomarkers and repeating echocardiography in survivors are important aspects to consider in future investigation.

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Mortality Risk Prediction in Amyopathic Dermatomyositis Associated With Interstitial Lung Disease

Perhaps Some Potential Details to Consider

To the Editor:

We read with interest the article by Lian et al[1] in the October 2020 issue of *CHEST*. The authors designed a prediction model to assess survival in patients with amyopathic dermatomyositis-associated interstitial lung disease (ADM-ILD) and to guide clinical treatment. It is an exciting model because the prognosis of patients with ADM-ILD is very poor, and high-risk patients must be identified as early as possible. Nevertheless, we believe that there are still some details worth discussing.

First, it may not be appropriate to select the survival rate at 1 year as the observed outcome.[1] According to previous studies of ADM-ILD,[2] the disease progresses very rapidly; one-half of the patients died within 6 months, which is consistent with the performance of Kaplan-Meier follow-up survival curves in this study. The choice of time node can be more subdivided. Although the number of patients lost to follow-up was not disclosed, based on the existing data and survival curves, it was found that at least five patients in the high-risk group of the discovery cohort had been lost to follow-up in 1 year, which will create bias in the calculation of the survival rate.

Second, we found that patients in the high-risk group in this study[1] had a lower survival rate regardless of cohort (especially a higher mortality rate in the validation