TO THE EDITORS: Joubert et al have made a commendable effort to elucidate the pathophysiology of pulmonary and myocardial edema in various preeclampsia phenotypes. Nevertheless, we do have a few alternative explanations. The authors ruled out overt left ventricular systolic dysfunction (LVSD) as a cause of pulmonary edema in preeclampsia. Therefore, the absence of LVSD despite the presence of myocardial edema is indeed noteworthy. Nevertheless, a normal LV systolic function was demonstrated in several previous studies that reported myocardial edema. Acute-onset myocardial edema without myocardial injury may not reflect systolic dysfunction. The ejection fraction is ubiquitously reported as an indicator of systolic function, and it has its limitations. Myocardial strain analysis is now frequently used to detect subclinical systolic dysfunction. Ejection fraction tends to normalize in the initial stages of evolving myocardial pathophysiology owing to alterations in the LV end-diastolic volume, wall thickness, circumferential strain, longitudinal strain, preload, afterload, and chronotropic activity. Moreover, the increased afterload (elevated systemic vascular resistance) increases LV contractility and normalizes ejection fraction through the Anrep effect. Therefore, subclinical LVSD in these patients cannot be ruled out without a detailed myocardial strain analysis.

The left atrial volume index (LAVI) was 38.9 mL/m² in women with preeclampsia and pulmonary edema. In these patients, the LAVI exceeded the cutoff (>34 mL/m²) suggested to diagnose diastolic dysfunction. The presence of at least 1 echocardiographic parameter beyond cutoff (tricuspid regurgitant velocity, E/e' [early mitral inflow filling velocity/mitral annular early diastolic velocity], or septal or lateral e' velocity) would have established the presence of diastolic dysfunction. Pulmonary edema has been commonly noted in patients with heart failure and preserved ejection fraction (HFpEF). In these subsets of patients, elevated LV end-diastolic pressure and subclinical LVSD could be implicated in the causation of pulmonary edema.

In this study, systemic blood pressure appears to be positively correlated with pulmonary and myocardial edema development. The preeclampsia with pulmonary edema phenotype had higher systolic (189.4 mm Hg vs 163.9 mm Hg) and diastolic blood pressure (110.6 mm Hg vs 103.6 mm Hg) than that without edema. Furthermore, the preeclampsia with severe features phenotype had significantly higher diastolic blood pressure (122 mm Hg vs 110.6 mm Hg vs 92.1 mm Hg) than that with pulmonary edema and without systemic features. Therefore, the presence of pulmonary edema and a greater extent of myocardial edema in patients with higher mean blood pressure are not surprising. An elevated capillary hydrostatic pressure could partly explain the mechanism of myocardial and pulmonary edema owing to higher mean blood pressure. In addition, we expect a lower plasma oncotic pressure because of protein loss in preeclampsia. Besides, disruption in the endothelial barrier could lead to a higher efflux of fluid in preeclampsia.

Therefore, based on the findings of this study, we can reasonably speculate on the pathophysiology of pulmonary and myocardial edema in preeclampsia. Moreover, the addition of a few echocardiographic indices (tricuspid regurgitant velocity, E/e', septal or lateral tissue doppler velocity, or myocardial strain), and biochemical parameters (serum albumin, protein) could have helped reexplore the pathophysiology of pulmonary and myocardial edema further.