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**REPLY FROM
AUTHORS: DEFINING
VENTRICULAR
DISTENSION ON
EXTRACORPOREAL
MEMBRANE
OXYGENATION AND
TRIGGERS TO**



INTERVENE

Reply to the Editor:

We thank you for your kind words and your thoughtful comments.¹ We completely agree that left ventricular (LV) distension can result in catastrophic effects such as thrombus formation, compromise LV recovery, and that its importance should be emphasized in the management of patients with cardiogenic shock supported with venoarterial extracorporeal membrane oxygenation (ECMO). A commonly agreed-upon definition of LV distension is central to future research efforts aiming to provide evidence-based management strategies. The very comprehensive definition of LV distension you propose should be adopted in that sense, as proposed in the latest version of the Extracorporeal Life Support Organization Guidelines.²

“Medical” unloading strategies described in the article³ aim to address this very important phenomenon while avoiding the potential complications associated with mechanical unloading in selected patients in which the baseline risk of LV distension did not warrant pre-emptive mechanical unloading. Current evidence, in our opinion, does not clearly support the superiority of a prophylactic in all patients compared with a more tailored approach mechanical unloading strategy. From a purely LV loading perspective, mechanical unloading is probably always

superior to medical unloading. However, multiple factors beyond the LV should be taken in consideration when managing patients in cardiogenic shock on venoarterial ECMO. Sometimes “less is more.” None of the mechanical unloading strategies are without potential severe adverse effects—may it be limb ischemia, hemolysis, or abdominal organ hypoperfusion. These complications may worsen organ dysfunction, compromise access to long-term therapies in the absence of recovery, and therefore counterbalance the potential benefits associated with decreased LV work. Until stronger evidence comes to support prophylactic mechanical unloading strategies, we advocate for an individualized approach, with careful monitoring of signs of LV distension in patients without pre-emptive unloading, proactive medical management, and rapid escalation to mechanical unloading if medical strategies fail.

The strength of definition proposed by Meani and colleagues¹ is that it provides clinicians with a constellation of signs on which to base management decisions. Isolated reduction in stroke volume as indicated by a left ventricular outflow tract (LVOT) velocity–time integral of <10 cm/s may reflect a profoundly depressed LV systolic function that may predispose to the development of LV dilatation and pulmonary congestion. However, it may also be an appropriate response to reduced LV filling in the setting of relatively high ECMO flows that drain in the circuit a high proportion of venous return. For instance, in a patient with a LVOT diameter of 2 cm, an LVOT velocity–time integral of 8 cm/s corresponds roughly to a stroke volume of 25 mL. With a heart rate of 80 beats per minute, this would still result in a cardiac output of 2 L/min. In a euvolemic patient with high ECMO flows, this may be enough to handle the limited residual transpulmonary blood flow and bronchial artery blood flow. Without overt signs of blood stasis, LV dilatation, increased LV filling pressures, and/or pulmonary congestion, it could be appropriate to hold mechanical unloading and proceed with medical management with close monitoring.

We thank the authors for the opportunity to further discuss this important issue and warmly welcome their efforts to standardize the definition of LV distension.

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