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UNLOADING THE LEFT VENTRICLE IN VENOARTERIAL EXTRACORPOREAL LIFE SUPPORT: THE URGENT NEED OF SPEAKING THE SAME LANGUAGE! To the Editor:

We have read the review article entitled "Controversies in ECMO: Immediate versus Watchful Waiting for VA-ECMO Venting" by Cavayas and colleagues,¹ published in the Special Issue of Invited Presentations: Adult: Mechanical Circulatory Support: Invited Expert Opinions of JTCVS Open December 2021. We congratulate the authors for this nice review article and, at the same time, provide some personal comments.

The authors described the pathophysiology of left ventricle (LV) overload and correctly underlined the role of the preload in this complex mechanism. LV preload has been often underestimated as an important determinant of LV distension during venoarterial (VA) extracorporeal life support (ECLS). Indeed, the patient's venous return usually exceeds the ECLS drainage and subsequently passes through the pulmonary circulation. As a consequence, the residual transpulmonary blood flow and bronchial venous return may not be counterbalanced by the reduced LV ejection due to impaired contractility and retrograde ECLS-generated blood flow toward the aortic valve, leading to LV dilation and pulmonary congestion.²

A meticulous fluid–balance management should be strongly recommended. Continuous renal-replacement therapy may be adopted to avoid fluid overload when diuretics resistance occurs.³

Furthermore, the authors highlighted the urgent need of a common LV overload definition during VA ECLS and clearly stated its absence in the literature. On one hand, we strongly believe this gap should be immediately solved to better analyze and elucidate the ongoing research on this

topic. On the other hand, a common LV overload definition has been already provided. Among 184 peripheral VA ECLS retrospectively investigated at the Maastricht University Medical Center, we developed a multiparametric approach to appropriately detect and monitor the LVrelated hemodynamics and potential overload appearance during VA ECLS.⁴

First of all, this definition considered direct and undirect parameters, which should be always assessed in relationship with the others. None of them is sufficient for diagnosing the LV overload.

Echocardiography plays a central role. This evaluation should quickly achieve an anatomical/functional overview of the heart. Besides the dimensions, volumes, and LV ejection fraction, the echocardiogram must include the aortic valve inspection. Protracted closure or incomplete valve opening are the most important markers of LV overload. As a consequence, the LV might be not able to eject, exceeding the aortic pressure caused by VA ECLS flow.⁵ Therefore, this may lead to dangerous blood stasis at the aortic root and LV cavity, potentially predisposing to cavity thrombosis,⁶ which can be "announced" by an increased "smoke-like" effect in the heart chambers or proximal aortic tract.

In addition, there are several echocardiographic semiquantitative or quantitative parameters that might be applied to better define the grade of LV impairment. Among them, the velocity time integral at the LV outflow tract is certainly one of the most commonly used. Ten centimeters should be proposed as the lowest cut-off,⁷ even if we suggest evaluating its trend over time.

Table 1^{8,9} shows the updated definition of LV overload that has been improved with new parameters and divided in 2 clinical scenarios based on VA ECLS implementation. Some parameters have been reserved to the preimplementation scenario. Central venous pressure and oxygen saturation, as well as the echocardiographic inferior vena cava evaluation, cannot be properly evaluated during full VA ECLS support, as a consequence of the venous cannula position. These parameters should be considered before ECLS, as marker of advanced fluid overload and severe myocardial disjunction. Likewise, the postcapillary wedge pressure should also be more useful either before ECLS placement or in the weaning phase.

Cavayas and colleagues¹ further declared that the LV can often be "medically" unloaded. Nevertheless, this statement might lead to underestimation of the current issue. In fact, in a cardiogenic shock animal model, we performed a full monitoring of the LV hemodynamic and workload.¹⁰ Although the relevant clinical parameters did not show any signs of LV overload or end-organ perfusion impairment, the pressure–volume analysis revealed increased LV filling pressure and diastolic volume, as well as raised

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	Grade of severity		
Method	Mild	Moderate	Severe
Pre-ECLS implementation			
Central venous line			
ScvO ₂ , %	75-55	55-45	<45
CVP, mm Hg	8-12	12-16	>20
Echocardiogram			
AoR or MR	Mild	Moderate	Severe
LV distension	Mild	Moderate	Severe
LA distension	Mild	Moderate	Severe
"Smoke-like" effect	Mild	Moderate	Severe
IVC dilatation, cm*	1.5-2.5	>2.5	>2.5
IVC collapse [†]	<50%	<50%	No change
VTI LVOT	13-17	8-12	<u>≤</u> 7
E/E'		>15 (increased LV filling pressure)	
Swan-Ganz Catheter (Edwards LifeSciences)			
PCWP, mm Hg	13-18	18-25	>25
During ECLS			
Arterial line			
Arterial pulsatility	Mild reduction	Moderate reduction	Almost (<8 mm Hg)
	(10-15 mm Hg)	(8-10 mm Hg)	or pulseless
Echocardiogram			
AV	Opening every 2 bpm	Opening every 3-4 bpm	Closure
LV distension	Mild	Moderate	Severe
LA distension	Mild	Moderate	Severe
"Smoke-like" effect	Mild	Moderate	Severe
VTI	13-17	8-12	≤7
Chest radiograph			
Congestion [‡]	Alveolar edema	Interstitial edema	Redistribution

ECLS, Extracorporeal life support; $ScvO_2$, central venous blood oxygen saturation; CVP, central venous pressure; AoR, aortic regurgitation; MR, mitral regurgitation; LV, left ventricle; LA, left atria; IVC, inferior vena cava; VTI, velocity time integral; LVOT, left ventricle outflow tract; E/E', early diastolic velocity of mitral annular motion/early diastolic velocity of mitral inflow; PCWP, postcapillary wedge pressure; AV, aortic valve; bpm, beats per minute. *IVC diameter in inspiration (Whitson and Mayo⁸). †IVC collapse in expiration (Whitson and Mayo⁸). ‡IVC collapse in expiration (Whitson and Mayo⁸).

myocardial oxygen consumption, as confirmed by other investigators. 11,12

Overall, we thank the authors for the opportunity to highlight some urgent needs in the LV management during VA ECLS. First, the absence of a shared definition of LV overload does not allow one neither to act consistently nor to compare the outcomes recorded in different centers. Second, the effectiveness of each venting strategy cannot be properly judged due to the lack of consensus and standard LV monitoring under VA ECLS. Furthermore, until well-designed and performed randomized trials are performed, it will be extremely difficult to consider beneficial or detrimental any strategy in this respect based on the large amount of confounding negative factors present in a patient undergoing VA ECLS. Therefore, imputing the actual impact or influence of LV unloading procedure on the ultimate patient outcome will be very difficult.

All these topics, in particular the elaboration of a common indication for unloading the LV, should be a priority for our scientific organization.

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