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EDITORIAL COMMENT

Atrial Septostomy for Left Ventricular Unloading*

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Propelling blood from the venous system, through an oxygenator, and into the arterial system, thus bypassing the heart and lung, is realized in venoarterial extracorporeal membrane oxygenation (VA ECMO), thereby supporting systemic perfusion in severe cardiac failure. At first glance, most would intuitively assume that such a direct bypass also unloads the left heart to relieve ischemia and decrease the tension imposed on the myocardium. However, after a closer look, the contrary—increased mechanical loading—may be the rule rather than the exception in VA ECMO support (1-5). Yet comprehensive analyses reveal that the mechanical devil is in the details.

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One way to potential address increased left ventricular loading is explored by Mlcek et al (6) in their elegant experimental study in this issue of *JACC: Cardiovascular Interventions*. Their results are in agreement with published clinical case series (7,8). The investigators also suggest "permissive hypotension" as an important clinical unloading strategy, as they are identifying arterial blood pressure as a major determinant of left ventricular oxygen consumption both with and without atrial septostomy. This is in line with earlier simulation studies (5), although it should be noted that individualized intensive care therapy in complex circulatory failure supported by VA ECMO cannot be standardized, and a detailed understanding of the effects of VA ECMO is imperative. Every case requires careful consideration of all available options, accounting for volume status, right- and left-sided contractility, ventricular interdependence, and cardiac afterload, as emphasized by the investigators (5,9).

A mechanistic simulation study can demonstrate that although the left ventricle often dilates with increasing ECMO flow (5,9), the right ventricle is usually being slightly unloaded. These net effects are the consequences of a complex interplay including changes in preload and afterload as well as serial, septal, and pericardial actions, all of which are elements of the overarching interdependencies of the right and left heart.

Right-sided filling pressures inherently tend to be reduced by the central venous drainage limb of the VA ECMO circuit, but much less than anticipated (10). Notably, after venous efflux, the extracorporeal blood flow re-enters the same native circulation and is not pooled in a venous reservoir as during routine surgical cardiopulmonary bypass. Effectively, the net reduction of venous volume is thus confined to the blood volume needed to increase systemic pressures in the arterial system. In cardiogenic shock, this increment of perfusion pressure is obviously very desirable to secure vital organ function and integrity, although concurrent concerns may arise pertaining to detrimental overload of the failing left heart. In this context, it is important to realize that the percutaneous balloon atrial septostomy described by Mlcek et al (6) acts by partly diverting left-sided pulmoatrial blood flow to the right atrium, yet the circulatory system supported by VA ECMO remains a closed loop.

The augmented systemic afterload imposed by the ECMO flow is often assumed to be related to the retrograde flow originating from the femoral arterial cannula, at times streaming far up into the proximal

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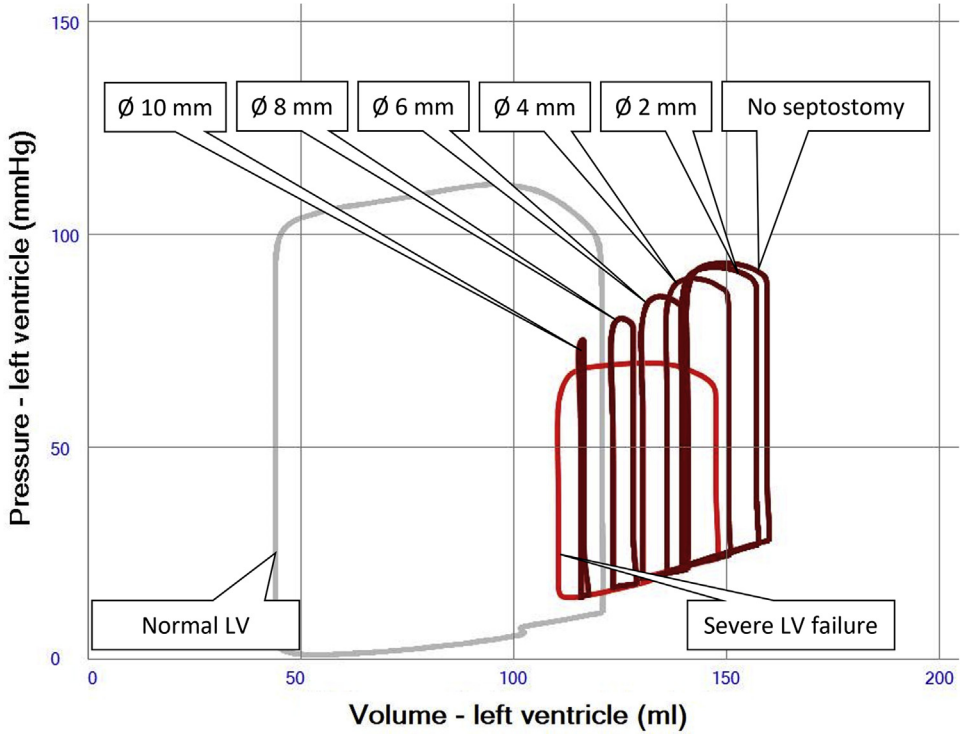
FIGURE 1 **Simulated LV Pressure-Volume Loops Illustrating Effects of Atrial Septostomy in Severe LV Failure Supported by VA ECMO**



Normal left ventricular (LV) function is shown in **gray** and severe LV failure without support in **bright red**. The **dark red curves** show loops during VA ECMO support. The rightmost **dark red loop** is without atrial septostomy. The **dark red loops** are moving to the left, indicating less dilatation with increasing size of the septostomy (diameter 2, 4, 6, 8, and 10 mm). The 10-mm septostomy imposes a risk for a nonejecting left ventricle with risk for LV thrombus formation, as indicated by the loop's resembling a vertical line with almost no difference between systolic and diastolic volumes, equivalent with a stroke volume close to zero.

aorta. Yet experimental evidence suggests that afterload is more related to aortic and peripheral arterial pressures per se than to the actual direction of blood flow (11). These findings are in agreement with the results of the present study by Mlcek et al (6).

In daily clinical practice, further confusion may arise while evaluating the adequacy of the patient's circulation on the basis of venous saturations. During peripheral, bifemoral VA ECMO, it should be realized that there is no anatomical site within the venous system where truly mixed venous blood (mixed venous oxygen saturation [SvO_2]) reflecting circulatory adequacy can be sampled. Samples taken before the oxygenator and in the inferior caval vein blood overestimate SvO_2, whereas superior caval, right atrial, and pulmonary artery blood sampling sites underestimate SvO_2 (12). It should also be noted that oxygen delivery can substantially be increased by draining the least oxygenated blood, as usually found in the superior caval vein, into the venous limb of the ECMO circuit (12,13). Interpretation of venous oxygen saturation is further complicated when atrial left-to-right shunting is created, as in the present study.

Each different aspect of the patient's treatment and the optimal tuning of different components of the various support modalities involved may be quite understandable. Yet overall optimization by integration of all these elements when looking at the bigger picture of the individual's state of disease may be very cumbersome, especially in severe cardiogenic shock with impending or overt multiorgan failure. Traditional evidence-based medicine approaches cannot sort out how patient-specific, therapeutic details such as flow settings, cannulation modes, and a well-educated choice among the available spectrum of adjunct unloading techniques should be fitted to the individual case. Here, recent advances in cardiovascular computational modeling come into play, heading toward the idealized digital twins of our critically ill patients. We are moving forward by leaps and bounds

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while aiming to bridge the gap between the mechanistic complexity of modern care and support technologies dynamically interplaying with the patient-specific manifestation of life-threatening disease (5,9). Mlcek et al (6) point to the use of patient-specific computer simulation as a tool to understand and optimize clinical treatments (14). The complexities of doing an atrial septostomy while running VA ECMO in a patient with left heart failure is a realistic clinical scenario, in which patient-specific simulation may significantly contribute to our understanding as a versatile clinical tool toward the best possible individualized care in the coming future (**Figure 1**).

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