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REVIEW TOPIC OF THE WEEK

Hemodynamics of Mechanical Circulatory Support

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JACC JOURNAL CME

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CME Objective for This Article: After reading this article, the reader should be able to: 1) describe the characteristics of the left ventricular end-systolic and end-diastolic pressure-volume relations and which of their features can be used to index contractility and diastolic properties; 2) describe how changes in preload, afterload, ventricular contractility, and heart rate impact the left ventricular pressure-volume loop (specifically end-diastolic volume and pressure, stroke volume, systolic pressure generation) and myocardial oxygen demand; and 3) describe anatomic and physiological differences between the different types of mechanical circulatory support currently in use clinically.

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Hemodynamics of Mechanical Circulatory Support

ABSTRACT

An increasing number of devices can provide mechanical circulatory support (MCS) to patients with acute hemodynamic compromise and chronic end-stage heart failure. These devices work by different pumping mechanisms, have various flow capacities, are inserted by different techniques, and have different sites from which blood is withdrawn and returned to the body. These factors result in different primary hemodynamic effects and secondary responses of the body. However, these are not generally taken into account when choosing a device for a particular patient or while managing a patient undergoing MCS. In this review, we discuss fundamental principles of cardiac, vascular, and pump mechanics and illustrate how they provide a broad foundation for understanding the complex interactions between the heart, vasculature, and device, and how they may help guide future research to improve patient outcomes.
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For patients with advanced heart failure, there are an increasing number of therapies, especially in the form of mechanical circulatory support (MCS). There are several classes of MCS devices, distinguished by hemodynamic characteristics of the pump, the sites from which blood is withdrawn and returned, the size of catheters and/or cannulas used, whether the insertion technique is percutaneous or surgical, and whether or not a gas exchange unit is used. Some devices are for short-term use, whereas others can be used for the duration of a patient's life. These characteristics contribute to determining the ease of deployment, ease of patient management while on the device, and overall safety profile, as reviewed recently in detail (1). To varying degrees, all available devices improve cardiac output and blood pressure (2-5), but their specific features result in different overall hemodynamic effects. The implications of these differences are only partially understood (6) and have not yet been researched in clinical trials.

Right heart catheterization with a pulmonary artery catheter (PAC) is the cornerstone of a standard clinical hemodynamic evaluation of patients undergoing MCS. However, widespread routine use of PAC has declined over the past decade and there is no consensus on systematic use of PAC data (7). As a result, important differences in hemodynamic effects of different forms of MCS may have gone unrecognized. A full understanding from advanced hemodynamic principles of the mechanisms of such differences has the potential to impact clinical practice and outcomes.

This review aims to provide a concise overview of advanced hemodynamic principles, including the basics of ventricular mechanics, ventricular-vascular coupling, and myocardial energetics (see [8-10] for detailed descriptions). We will then review how these principles can be applied to better understand the hemodynamic effects of MCS.

FUNDAMENTALS OF LEFT VENTRICULAR MECHANICS

Events occurring during a single cardiac cycle are depicted by ventricular pressure-volume loops (PVLs) (**Figure 1A**). Under normal conditions, the PVL is roughly trapezoidal, with a rounded top. The 4 sides of the loop denote the 4 phases of the cardiac cycle: 1) isovolumic contraction; 2) ejection; 3) isovolumic relaxation; and 4) filling. The loop falls within the boundaries of the end-systolic pressure-volume relationship (ESPVR) and the end-diastolic pressure-volume relationship (EDPVR). The ESPVR is reasonably linear, with slope Ees (end-systolic elastance) and volume-axis intercept Vo. The EDPVR is nonlinear and described by simple equations, such as: $P = \beta(e^{\alpha[V-Vo]} - 1)$ or $P = \beta V^{\alpha}$. ESPVR, and EDPVR shifts occur with changes in ventricular contractility and diastolic properties (remodeling).

The actual position and shape of the loop depend on ventricular pre-load and afterload. At the organ level, pre-load can be defined as either end-diastolic pressure (EDP) or the end-diastolic volume (EDV), which relate to average sarcomere stretch throughout the myocardium. Afterload is determined by the hemodynamic properties of the vascular system against which the ventricle contracts and is most generally characterized by its impedance spectrum (the frequency-dependent ratio and phase shift between