Hemodynamic Support
Science and Evaluation of the Assisted Circulation with Percutaneous Assist Devices

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KEYWORDS

• Circulatory support • Pressure-volume relationships • Pressure-volume area • ESPVR • EDPVR • Cardiogenic shock • Contractility

KEY POINTS

- Four fundamentally different modes of circulatory support include aortic counterpulsation, left atrial-to-arterial pumping, right atrial-to-arterial pumping, and left ventricular-to-aortic pumping.
- Each approach has a distinct hemodynamic fingerprint with regard to effects on the ventricular pressure-volume loop and key hemodynamic and metabolic parameters.
- An understanding of the different modes of circulatory support may help guide the choice of which device is most appropriate for a given clinical setting.
- Understanding the different modes of circulatory support has the potential to guide future researchers in the optimization of therapies, to help generate hypotheses for clinical trials, and to help guide the choice of device for specific clinical trials that will ultimately provide evidence needed to guide therapeutic decision-making.

INTRODUCTION

The use of percutaneous devices to support the circulation in patients with various forms of hemodynamic compromise and for prophylactic use during high-risk coronary interventions where such compromise is believed likely to occur is becoming more common, which is especially the case as such devices become easier to deploy, safer to use, and hemodynamically more potent. As reviewed in the other articles of this edition, the number of devices and the range of clinical indications in which they are applied are growing. In addition to devices to assist the left ventricle, the value of percutaneous right-ventricular assist devices is becoming increasingly appreciated.

Because different devices have different modes of action, the clinician is faced with the task of choosing the appropriate device for each particular clinical setting. This task can be facilitated by an understanding of the fundamental hemodynamic principles that allow description of the nature and severity of changes in the heart and vascular function in different disease states and the nature and potency with which different devices interact with the heart and vasculature. The goals of cardiac support are different in different settings. For the elective setting of high-risk coronary intervention, the goal is primarily to maintain reasonably normal systemic blood flow and blood pressure during a transient period of coronary occlusion and myocardial dysfunction. In contrast, in more emergent settings, such as cardiogenic shock, pulmonary edema, and pulmonary dysfunction, the goal is often to take over the work, partly or wholly, of the failing right and/or left

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ventricle to ensure normal blood pressure, cardiac output, and pulmonary venous pressures over extended periods of time.[1–5] When these goals are attained, end-organ perfusion and function are maintained, blood can be adequately oxygenated by the lungs, and diuresis is promoted in states of volume overload. Furthermore, by resting the heart and simultaneously ensuring end-organ perfusion in these settings, the odds of native heart recovery without permanent end-organ damage may be improved.

In addition to direct hemodynamic effects, the impact of different devices on coronary blood flow and myocardial oxygen demand can be important, especially in settings of acute coronary syndromes where preservation of myocardial function and viability are of primary concern to maximize the chances of recovery.

FUNDAMENTAL HEMODYNAMIC PRINCIPLES

The ventricular pressure-volume (PV) framework provides a foundation for understanding cardiac and vascular properties, myocardial energetics, and the impact of different modes of percutaneous circulatory support strategies. This framework allows representation of ventricular preload, afterload, lusitropy, and contractility and their respective roles in determining cardiac output, blood pressure, and pulmonary venous pressures. Details of this approach have been summarized previously[6,7] and are reviewed here in brief. The basic concepts are summarized by the pressure-volume relations displayed in **Fig. 1**. (Note that this and all other figures and quantitative values presented in this article have been derived from a previously described and validated real-time, interactive cardiovascular simulation.[8]) The normal PV loop (shown in blue) is a plot of instantaneous ventricular pressure and volume throughout the cardiac cycle. The 4 major phases of the cardiac cycle are readily identified. Starting at end-diastole (bottom right corner), these phases are as follows: isovolumic contraction, ejection, isovolumic relaxation, and filling. The PV loop is bounded inferiorly by the end-diastolic pressure-volume relationship (EDPVR) and superiorly by the end-systolic pressure-volume relationship (ESPVR). The EDPVR uniquely defines the passive diastolic properties of the LV and the slope (E_{es}) and volume axis intercept of the ESPVR provide a load-independent index of ventricular contractility.

In this construct, ventricular preload is indexed by either end-diastolic volume (EDV) or end-diastolic pressure (EDP). Ventricular afterload is indexed by effective arterial elastance (Ea), which is the slope of the line connecting the point on the volume axis at the EDV to the end-systolic PV point. Ea is mainly determined by total peripheral arterial resistance (TPR) and the duration of the cardiac cycle (T) according to Ea ≡ TPR/T.[9]

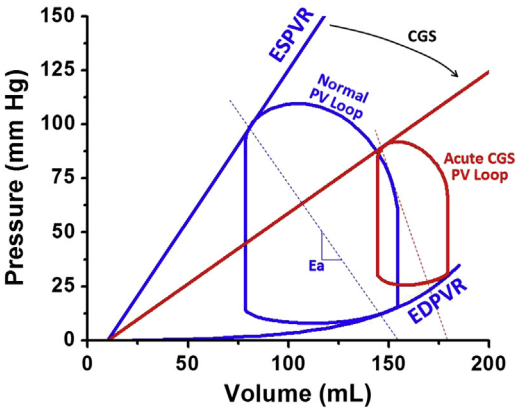

Fig. 1. Prototypical pressure-volume (PV) loops from a normal adult (*blue*) and from a patient in cardiogenic shock (CGS, shown in *red*). The loops are bound by the end-systolic and end-diastolic pressure-volume relations (ESPVR and EDPVR, respectively). The downward shift of the ESPVR is a reflection of the reduction in left ventricular contractility that underlies the development of CGS. Effective arterial elastance (Ea), an index of ventricular afterload, which depends primarily on total peripheral resistance and heart rate, can also be derived from the loop. See text for further details.

One additional hemodynamic parameter of interest is cardiac power output (CPO), which is defined as the product of stroke work (SW, the area inside the PV loop) and heart rate (HR). However, because it is not possible to precisely quantify SW noninvasively, SW is approximated as the product of mean arterial pressure (MAP) and stroke volume (SV). Accordingly, CPO ≡ SW × HR ≈ MAP × SV × HR = MAP × CO. This parameter integrates information related to 2 fundamental functions of the heart: its ability to generate blood pressure and its ability to generate cardiac output. In addition, interest in this parameter has stemmed largely from the fact that for patients presenting with cardiogenic shock (CGS), CPO has an inverse relation to in-hospital and short term survival.[10–12]

Myocardial Oxygen Demand and Supply

Many clinicians think that myocardial oxygen consumption is related to SW (on a per beat basis) or to CPO (on a per unit time basis). However, this is not the case because SW does not quantify all of the work done by the heart with each contraction. Myocardial oxygen consumption has been shown