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Clinical Reviews

OPTIMIZING OXYGEN DELIVERY IN THE CRITICALLY ILL: ASSESSMENT OF VOLUME RESPONSIVENESS IN THE SEPTIC PATIENT

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☐ **Abstract—Background: Assessing volume responsiveness, defined as an increase in cardiac index after infusion of fluids, is important when caring for critically ill patients in septic shock, as both under- and over-resuscitation can worsen outcomes. This review article describes the currently available methods of assessing volume responsiveness for critically ill patients in the emergency department, with a focus on patients in septic shock. Objective: The single-pump model of the circulation utilizing cardiac-filling pressures is reviewed in detail. Additionally, the dual-pump model evaluating cardiopulmonary interactions both invasively and noninvasively will be described. Discussion: Cardiac filling pressures (central venous pressure and pulmonary artery occlusion pressure) have poor performance characteristics when used to predict volume responsiveness. Cardiopulmonary interaction assessments (inferior vena cava distensibility/collapsibility, systolic pressure variation, pulse pressure variation, stroke volume variation, and aortic flow velocities) have superior test characteristics when measured either invasively or noninvasively. Conclusion: Cardiac filling pressures may be misleading if used to determine volume responsiveness. Assessment of cardiopulmonary interactions has superior performance characteristics, and should be preferentially used for septic shock patients in the emergency department.** © **2014 Elsevier Inc.**

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☐ **Keywords—volume responsiveness; volume assessment; critically ill; critical care; septic shock**

INTRODUCTION

Critically ill patients with circulatory failure are commonly encountered in the emergency department (ED) setting, and initial resuscitation often involves an aggressive approach to volume expansion. The ultimate goal of volume expansion is to improve cardiac output and ultimately, oxygen delivery. Whereas under-resuscitation of the hemodynamically compromised patient can result in further end-organ dysfunction, over-resuscitation can lead to volume overload and prolong the need for mechanical ventilation, intensive care unit length of stay, and increase mortality (1–3). Assessment of volume status, or more appropriately, volume responsiveness (i.e., whether a patient's cardiac index will be responsive to increased circulatory volume) is a skill of paramount importance to the emergency physician, so that resuscitation may be performed in a rational manner. The means of assessing volume responsiveness have been the subject of great controversy and active research. This review will describe the currently available methods of volume assessment, with a focus on the septic patient in circulatory failure.

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DISCUSSION

Cardiac Filling Pressures and the Single-Pump Model of the Circulation

Cardiac filling pressures (central venous pressure [CVP] and pulmonary artery occlusion pressure [PAOP]) have been used as a guide for optimizing preload and fluid resuscitation since the 1950s, as first described by Hughes and Magovern (4). Cardiac filling pressures are thought to obey the Starling principle, which states that increased stretch (preload) of a cardiac myocyte will increase the contractility until you reach a plateau (Figure 1) (5,6). When applied to the cardiovascular system of a patient in shock rather than a single myocyte or cardiac chamber, the assumption is that changes in right atrial pressure (preload) lead to changes in cardiac output (contractility), and has been shown to be true in healthy male volunteers as central venous pressure changes linearly with either hemorrhage or transfusion for the first several minutes (7).

This is consistent with a single-pump model of the circulation, which ignores the right ventricle and pulmonary circulatory effects on left ventricular output, and assumes that restoration of central venous pressure leads to restoration of circulating volume. Previous researchers subscribing to this model report only three possible explanations for shock: loss of vascular tone, decreased contractility, and volume depletion (8,9). Thus CVP has gained popularity as the endpoint of choice for volume resuscitation and is incorporated into management algorithms and guidelines for the care of the septic shock patient (10–12).

Unfortunately, cardiac filling pressures have been unable to differentiate volume responders from nonresponders with an area under the receiver operating characteristic (ROC) curve for CVP 0.58 and PAOP 0.63 (13). With an area under the ROC curve of 0.58, CVP is only slightly better than chance at predicting volume responders from nonresponders, with chance being 0.50 and a perfect prediction 1.0. Additionally, achieving target cardiac filling pressures shows poor correlation to improvement in either left ventricular performance (end-diastolic volume [EDV], stroke volume [SV], or cardiac index [CI]) or circulating blood volume (14,15). Lastly, even CVP at the extremes of range (low or high) fails to differentiate responders from nonresponders (13). In a comprehensive literature review up to 2006, and repeated in 2012, Marik et al. conclude that the measurement of CVP, whether to determine circulating blood volume or as a response to volume challenge, is not useful, with very poor test characteristics (ROC of 0.56) (16,17).

These data suggest that cardiac filling pressures are not effective parameters for assessing whether a hemodynamically compromised patient will respond to volume infusion. Rather than a single pump with CVP affected by only three variables, the right ventricle and pulmonary circulation in fact do play an important role. Thus, right ventricular compliance, the presence of tricuspid regurgitation or stenosis, and pulmonary hypertension all influence central venous pressure. Additionally, left ventricular compliance, intrathoracic pressure, chest wall compliance, and vascular tone also contribute to central venous pressure, exclusive of intravascular volume (18). Given these confounders, cardiac filling pressures cannot reflect the actual right and left end diastolic volumes exclusively, and thus will not be able to predict volume responsiveness clinically.

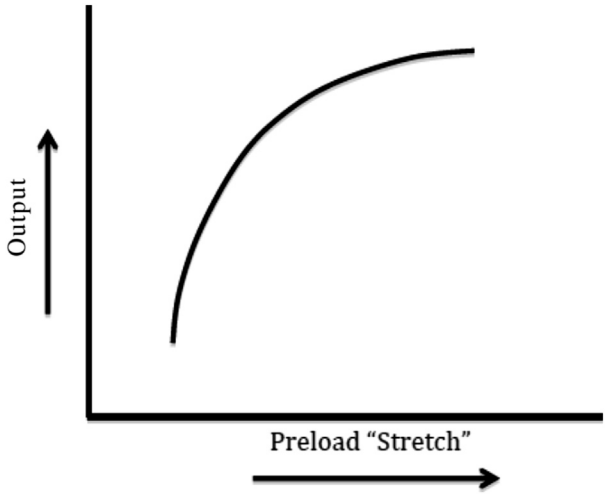

Figure 1. The "Starling Curve" demonstrating changes in contractility per change in preload. The "Starling Curve" states that as you increase the preload, or stretch, of a muscle (i.e., left ventricle) the output will increase. Once the preload limit is reached, further increases in preload lead to only minimal increases in output. Adapted from Michard F, Teboul JL. Using heart-lung interactions to assess fluid responsiveness during mechanical ventilation. Crit Care 2000;4:282–9, Figure 1 (24).

Cardiopulmonary Interactions and the Dual-Pump Model of the Circulation

A dual-pump model that accounts for right ventricular, pulmonary vascular, and intrathoracic influence on cardiac performance is a more accurate description of the forces that determine volume responsiveness. The right ventricle (RV) is a low-pressure, highly compliant, flow-based chamber, as opposed to the left ventricle (LV), which is a high-pressure, low-compliance chamber (19,20). As a result, the RV is far more sensitive to changes in afterload than the LV, which can maintain stroke volume in the face of large increases in systemic vascular resistance. RV stroke volume, however, is sensitive to variation in venous return, and RV afterload