Exploring Hemodynamics

A Review of Current and Emerging Noninvasive Monitoring Techniques

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KEYWORDS

• Stroke volume • Hemodynamics • Cardiac output • Hypovolemia

KEY POINTS

- The lack of randomized controlled trials suggesting improved outcomes with pulmonary artery catheter use and pressure-based hemodynamic monitoring has led to a decrease in pulmonary artery catheter use.
- An increasing amount of literature supporting stroke volume optimization (SVO) has given rise to a paradigm shift from pressure-based to flow-based techniques.
- Regardless of the device chosen, the SVO algorithm approach should be considered, and volume challenges should be guided by dynamic assessments of fluid responsiveness. Although SVO requires further study in extubated and medical intensive care unit populations, a mortality benefit has been observed in high-risk surgical patients.
- The use of SVO is supported by more evidence than for central venous pressure for fluid resuscitation.

INTRODUCTION

Over the last 15 years, hemodynamic monitoring has evolved. Monitoring technologies and resuscitation strategies are transitioning from pressure-based parameters (such as central venous pressure [CVP]) to flow-based parameters (such as cardiac output and stroke volume [SV]) as the supporting evidence increases for a protocolized approach to hemodynamic optimization rather than traditional approaches. Early goal-directed therapy protocols are an emerging standard of practice that is no longer limited to the care of patients with sepsis. This article provides a general overview of the paradigm shift occurring in hemodynamic monitoring, including current monitoring techniques, resuscitation end points, and supporting evidence. In contrast with many other hemodynamic review articles, this article also focuses on practical application

Conflict of Interest: None.
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Crit Care Nurs Clin N Am 26 (2014) 357–375
http://dx.doi.org/10.1016/j.ccell.2014.05.001 **ccnursing.theclinics.com**
0899-5885/14/$ – see front matter

and systematic use of technology so that cardiac output and SV can be optimized regardless of the device used.

Before deliberating over the emerging trends and latest devices in hemodynamic monitoring, it is worthwhile to discuss the goals of monitoring and hemodynamic optimization. The goals of this concept are largely concerned with 2 objectives: (1) optimizing the macrocirculation (eg, cardiac output, SV), and (2) optimizing the tissue oxygenation via end points such as blood lactate, central venous oxygen saturation ($ScvO_2$), and mixed venous oxygen saturation (SvO_2) levels. This article discusses the macrocirculatory parameters of hemodynamic optimization as they relate to current and emerging trends in hemodynamic monitoring.

FROM CARDIAC PRESSURES TO PARAMETERS BASED ON BLOOD FLOW

In the past, cardiac filling pressures were associated with a proportionate cardiac filling volume. However, recent studies suggest that cardiac pressures do not consistently correlate with cardiac volume. A systematic review of the literature was published in *Chest* in 2008 by Marik and colleagues.[1] That study examined every article published to date on the ability of the CVP to predict preload responsiveness or cardiac output after a fluid challenge in humans (24 studies met the inclusion criteria, n = 803). In that review, Marik and colleagues[1] could find no data to suggest that the CVP correlated well with intravascular volume status or preload. The investigators thus concluded that the CVP should no longer be used to make clinical decisions regarding fluid management. The pooled correlation coefficient between CVP and measured blood volume was 0.16 (95% confidence interval, 0.03–0.28), which suggests that the accuracy of CVP for measuring blood volume is like flipping a coin.[1] Similar studies have been published regarding the poor correlation between the pulmonary artery occlusive pressure (PAOP) and left ventricular end-diastolic volume.[2–5] Several clinical factors can exacerbate this poor correlation between pressure and volume (ie, factors that alter the pressure-compliance curve of the myocardium). These factors include mechanical ventilation, aging, obesity, history of myocardial infarction, diabetes, and sepsis.[6] The primary reason to administer a fluid challenge is to increase the SV (or cardiac output), not static cardiac filling pressures such as PAOP or CVP. The PAOP and CVP were intended to be guides to better optimize cardiac output and SV.

Meanwhile, the last 10 years have produced other studies that have highlighted the slow-to-change and misleading nature of other commonly used monitoring parameters in critical care. In 1996, Hamilton-Davies and colleagues[7] published an observational study that showed the limitations of traditional vital signs as early indicators of volume depletion. Six healthy volunteers were phlebotomized of a mean 25.3% (standard deviation [SD], 3.5%) of their respective blood volumes over 90 minutes. Systolic blood pressure, heart rate, and SV were among the parameters continuously monitored. After the 90-minute target time, the only statistically significant change in these parameters was in the SV. Average SV decreased by a mean of 16.5 mL (SD, 15 mL; $P<.01$). Systolic blood pressure and heart rate failed to show consistent or significant changes (**Fig. 1**). These findings suggest that pressure-based parameters and traditional vital signs may not equate to blood flow–based parameters, and that flow decreases before pressure decreases as hypovolemia worsens.

USE OF SV TO ASSESS FLUID RESPONSIVENESS

SV Optimization

The study by Hamilton-Davies and colleagues[7] suggests a temporal order of events in hypovolemia, beginning with a decrease in SV when a decrease in overall circulating