

Review

Functional hemodynamic monitoring

Michael R Pinsky¹ and Didier Payen²

¹Professor of Critical Care Medicine, Bioengineering and Anesthesiology, Department of Critical Care Medicine, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA

²Professor of Anesthesiology, Department of Anesthesiology and Critical Care Medicine, Lariboisière Hospital, University of Paris VII, Paris, France

Corresponding author: Michael R Pinsky, pinskymr@com.upmc.edu

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Abstract

Hemodynamic monitoring is a central component of intensive care. Patterns of hemodynamic variables often suggest cardiogenic, hypovolemic, obstructive, or distributive (septic) etiologies to cardiovascular insufficiency, thus defining the specific treatments required. Monitoring increases in invasiveness, as required, as the risk for cardiovascular instability-induced morbidity increases because of the need to define more accurately the diagnosis and monitor the response to therapy. Monitoring is also context specific: requirements during cardiac surgery will be different from those in the intensive care unit or emergency department. Solitary hemodynamic values are useful as threshold monitors (e.g. hypotension is always pathological, central venous pressure is only elevated in disease). Some hemodynamic values can only be interpreted relative to metabolic demand, whereas others have multiple meanings. Functional hemodynamic monitoring implies a therapeutic application, independent of diagnosis such as a therapeutic trial of fluid challenge to assess preload responsiveness. Newer methods for assessing preload responsiveness include monitoring changes in central venous pressure during spontaneous inspiration, and variations in arterial pulse pressure, systolic pressure, and aortic flow variation in response to vena caval collapse during positive pressure ventilation or passive leg raising. Defining preload responsiveness using these functional measures, coupled to treatment protocols, can improve outcome from critical illness. Potentially, as these and newer, less invasive hemodynamic measures are validated, they could be incorporated into such protocolized care in a cost-effective manner.

Introduction

Hemodynamic monitoring is a cornerstone of care for the hemodynamically unstable patient, but it requires a manifold approach and its use is both context and disease specific. One of the primary goals of hemodynamic monitoring is to alert the health care team to impending cardiovascular crisis before organ injury ensues; it is routinely used in this manner in the operating room during high-risk surgery. Another goal of hemodynamic monitoring is to obtain information specific

to the disease processes, which may facilitate diagnosis and treatment and allow one to monitor the response to therapy.

The effectiveness of hemodynamic monitoring depends both on available technology and on our ability to diagnose and effectively treat the disease processes for which it is used. The utility of hemodynamic monitoring has evolved as it has merged with information technology and as our understanding of disease pathophysiology has improved. Within this context, hemodynamic monitoring represents a functional tool that may be used to derive estimates of performance and physiological reserve that may in turn direct treatment. However, no monitoring device can improve patient-centered outcomes unless it is coupled to a treatment that improves outcome. Thus, hemodynamic monitoring must be considered within the context of proven medical therapies, success of which is dependent on the clinical condition, pathophysiological state and ability to reverse the identified disease process.

Rationale for hemodynamic monitoring

A progression of arguments supporting the use of specific monitoring techniques can be proposed. At the basic level, monitoring can be defended on the basis of historical controls. In this regard, prior experience with similar monitoring techniques indicates that they can identify known complications that are undetectable with less invasive means. Clearly, the mechanism by which the benefit is achieved need not be understood or even postulated.

Further support for hemodynamic monitoring comes from an understanding of the pathophysiology of the process being treated, such as heart failure or hypovolemic shock. Weil and Shubin [1] defined circulatory shock as decreased ability of blood flow to meet the metabolic demands of the body. Using their classic approach, four basic groups of circulatory shock can be defined: hypovolemic, cardiogenic, obstructive, and

CVP = central venous pressure; LV = left ventricular; PAC = pulmonary artery catheter; $P\text{CO}_2$ = partial carbon dioxide tension; $P\text{pao}$ = pulmonary arterial occlusion pressure; RV = right ventricular; Scvo_2 = central venous oxygen saturation; $\text{S}\bar{\text{v}}\text{O}_2$ = mixed venous oxygen saturation.

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