Functional Hemodynamic Monitoring

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KEYWORDS

- Functional hemodynamic monitoring • Dynamic tissue O_2 saturation
- Positive-pressure ventilation • Stroke volume variation

KEY POINTS

- Functional hemodynamic monitoring reflects the assessment of the dynamic interactions of hemodynamic variables in response to a defined perturbation.
- Dynamic tissue O_2 saturation responses to complete stop-flow conditions (vascular occlusion test) assess cardiovascular sufficiency and distribution of microcirculatory blood flow.
- Dynamic inspiratory changes in central venous pressure during spontaneous ventilation identify both cor pulmonale and volume responsiveness.
- Dynamic changes in arterial pulse pressure (diastole to systole) and left ventricular stroke volume during positive-pressure ventilation reflect the degree to which the subject is volume responsive.
- Both pulse pressure variation (PPV) and stroke volume variation (SVV) quantitatively track volume responsiveness, with a threshold value of greater than 10% to 15% defining a subject whose cardiac output will increase by greater than 15% in response to a 500-mL fluid bolus.
- Dynamic changes in PPV and SVV cannot be used in the setting of atrial fibrillation, acute cor pulmonale, or when spontaneous breathing is forceful and erratic.
- Dynamic changes in cardiac output in response to a passive leg-raising maneuver also predict volume responsiveness.
- PPV/SVV defines central arterial stiffness or elastance, and can be used as a surrogate marker of vasomotor tone.

This work was supported in part by the National Institutes of Health grants HL67181 and HL073198.
Potential Conflicts of Interest: M.R. Pinsky is the inventor of a University of Pittsburgh US Patent "Use of aortic pulse pressure and flow in bedside hemodynamic management." Member of the Scientific Advisory Board for LiDCO Ltd. Stock options from LiDCO Ltd and Cheetah Medical, Ltd.
Receives institutional funding for research from Edwards LifeSciences, Inc.
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Crit Care Clin 31 (2015) 89–111
http://dx.doi.org/10.1016/j.ccc.2014.08.005 **criticalcare.theclinics.com**
0749-0704/15/$ – see front matter

INTRODUCTION

Hemodynamic monitoring is the active assessment of cardiopulmonary status by the use of biosensors that assess physiologic outputs. The simplest form of monitoring is the individual health care professional, inspecting the patient for consciousness, agitation or distress, breathing regular or labored, the presence or absence of central and peripheral cyanosis; touching of the skin of a patient to note if it is cool and moist, and if capillary refill is rapid or not; palpation of the central and peripheral pulses to note rate and firmness.

Although well established and important as bedside diagnostic tools, these simple "human-instrument" measures can be greatly expanded by the use of pulse oximetry to estimate arterial oxygen saturation (Spo_2), and the sphygmomanometer and auscultation to note systolic and diastolic blood pressure and identify pulsus paradoxus. These classic measures of hemodynamics, often referred to as routine vital signs, are central to the assessment of cardiorespiratory sufficiency and much of diagnostic bedside medicine is rooted in these important techniques.

However, with some exceptions, these simple and inexpensive measures do not have the discriminatory value in identifying patients as being stable or unstable when compensatory processes mask instability or when changes in physiologic state occur rapidly. Furthermore, they predict poorly who are at an early stage of an instability process, such as hypovolemia or heart failure, but compensating. Within the context of circulatory shock, tachycardia may or may not develop early and even if it is present, it is nonspecific. However, these simple measures can be markedly helped in their sensitivity to detect effective hypovolemia by making these same measures before and during an orthostatic challenge. For example, measuring blood pressure and pulse rate changes between lying supine, sitting, and standing markedly increase the diagnostic capability of the measures to identify functional hypovolemia. If heart rate increases and/or blood pressure decreases with sitting or standing, it is reasonable to presume that some degree of compatible hypovolemia exists. However, the other important concept in making these observations is that the measures themselves do not change, but their measured values change in response to a defined physiologic challenge: this is an example of functional hemodynamic monitoring.[1] Functional hemodynamic monitoring is the use of a defined physiologic stressor to access the physiologic reserve of the system.

Another example of functional hemodynamic monitoring is to use the morphology of the normal lead II electrocardiogram (ECG) to define ischemic heart disease. In practice, unless there is ongoing ischemia or prior infarction, the rhythm and morphology of the ECG signal is a poor marker of clinically relevant coronary artery disease. However, that same ECG signal, if monitoring during an exercise challenge that increases heart rate above a minimal amount defined by subject age, does not show any morphologic changes or arrhythmias, then it is highly unlikely that the subject has clinically significant coronary artery disease. It is important that as with the measure of pulse rate and blood pressure, ECG monitoring has not changed; it is the intervention that creates evolving hemodynamic parameters that markedly increase the sensitivity and specificity of hemodynamic monitoring to define cardiovascular state.

Using the functional hemodynamic monitoring principles described herein, it is possible for the bedside clinician to answer 4 interrelated and important questions of their patient[2]:

- Are they in compensated shock?
- Are they volume responsive?