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Functional Hemodynamic Monitoring:

Current Concepts in Critical Care

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Abstract

Purpose—Functional hemodynamic monitoring is the assessment of the dynamic interactions of hemodynamic variables in response to a defined perturbation.

Findings—Fluid responsiveness can be predicted during positive pressure breathing by variations in venous return or left ventricular output using numerous surrogate markers, like arterial pulse pressure variation (PPV), left ventricular stroke volume variation (SVV), aortic velocity variation, inferior and superior vena cavae diameter changes and pulse oximeter pleth signal variability. Similarly, dynamic changes in cardiac output to a passive leg raising maneuver can be used in any patient and measured invasively or non-invasively. However, volume responsiveness, though important, reflects only part of the overall spectrum of functional physiological variables that can be measured to define physiologic state and monitor response to therapy. The ratio of PPV to SVV defines central arterial elastance and can be used to identify those hypotensive patients who will not increase their blood pressure in response to a fluid challenge despite increasing cardiac output. Dynamic tissue O_2 saturation (StO_2) responses to complete stop flow conditions as can be created by measuring hand StO_2 and occluding flow with a blood pressure cuff, assesses cardiovascular sufficiency and micro-circulatory blood flow distribution. They can be used to identify those ventilator-dependent subjects who will failure a spontaneous berating trial or trauma patients in need of life saving interventions.

Summary—Functional hemodynamic monitoring approaches are increasing in numbers, conditions in which they are useful and resuscitation protocol applications. This is a rapidly evolving field whose pluripotential is just now being realized.

Keywords

cardiovascular physiology; minimally-invasive monitoring; non-invasive; resuscitation Shock

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Introduction

Increased interest in a more proactive use of monitoring technologies has emerged because clinical trials have consistently documented that the dynamic response of a measured output variable to a defined stress allows the bedside clinician to define the physiological state of the patient and manage them more proactively. This field when applied to the assessment of cardiovascular state is referred to as Functional Hemodynamic Monitoring: the assessment of the dynamic interactions of hemodynamic variables in response to a defined perturbation [1]. Such dynamic responses result in emergent parameters of these commonly reported variables, such as arterial pulse pressure and stroke volume, that greatly increase the ability of these measures to define cardiovascular state and predict need for and response to therapy. Since our last review of this topic for this series [2], many new studies and increased insight into functional hemodynamic monitoring has occurred. This review will highlight some of the studies since that time, placing them within context into the larger picture of diagnosis and management of the critically ill.

Presently, functional hemodynamic monitoring has proven useful in clinical trials at predicting volume responsiveness, defining loss of arterial tone and in identifying occult cardiovascular insufficiency (compensated shock). Like any test used for clinical decision making, its sensitivity and specificity improves if its pre-test probability is higher. Thus, application of functional hemodynamic monitoring approaches will be improved when placed within the appropriate clinical context.

Predicting volume responsiveness

A primary question asked in the management of a patient in shock is whether or not the patient will increase their cardiac output in response to intravascular volume infusion. Volume responsiveness has been arbitrarily defined as a ≥15% in cardiac output in response to a 500 ml bolus fluid challenge [3]. Michard et al. documented that the dynamic variations in arterial pulse pressure during positive-pressure breathing (8 ml/kg) when averaged over at least 3 breaths, referred to as pulse pressure variation (PPV), accurately predicts which patients would be volume responsive [3]. Many studies over the past 14 years since the original publication of this sentinel paper have validated the usefulness of such dynamic variations on blood flow induced by positive-pressure breathing to define volume responsiveness. For a detailed review of the evolution of PPV as a monitoring tool over this 14 year interval the reader is referred to a recent review on this topic [4].

Importantly, all the different measures including not only PPV, but also left ventricular (LV) stroke volume variation (SVV), changes in inferior and superior vena cavae diameters, systolic pressure variation and pulse oximeter pleth density variation are based on fundamental physiological principles underlying heart-lung interactions. And all have been documented to be robust predictors of volume responsiveness [5].

The physiologic basis for these parameters follows. During the inspiratory phase of positive pressure ventilation, intrathoracic pressure increases passively increasing right atrial pressure causing venous return to decrease, decreasing right ventricular (RV) output, and after two or three heart beats, LV output, if both RV and LV are volume responsive [3].

Thus, in preload dependent patients cyclic changes in LV stroke volume and its coupled arterial pulse pressure are seen and the magnitude of the changes is proportional to volume responsiveness. The actual calculation of PPV and SVV from the commercially available minimally-invasive monitoring devices quantified these two parameters in various ways (e.g. PiCCO, LiDCO, FloTrac). In general both PPV and SVV are defined as the ratio of the maximal minus the minimal values to the mean values, usually averaged over 3 or more breaths. We showed that increasing the sampling window to include at least 5 breaths minimizing sampling error in estimating both PPV and SVV [6]. Since all commercially-available devices reporting PPV and SVV use a sampling window of 15-20 second, they all incorporate this into their measure. Numerous studies have documented that a SVV >10% or a PPV> 13-15% on a tidal volume of 8 ml/kg or greater is highly predictive of volume responsiveness [3,7,8]. Clearly, changes in tidal volume, chest wall compliance and contractility will all affect these measures [9]. When ventilation includes spontaneous breathing or irregular heartbeats, PPV and SVV become inaccurate. Still, one study in septic shock patients showed that if the threshold values for PPV were increase to >15% the test still predicted volume responsiveness in spontaneously breathing patients [10]. Also, PPV remains predictive in elderly patients with varying degrees of diastolic heart failure [11]. Still, caution needs to be used wen interpreting these parameters when spontaneous breathing efforts are exaggerated. One debate that has arisen comes from the interpretation of PPV or SVV values in the “grey zone’ of 10-15%. Since many patients are ventilated with low tidal volumes and may also be vasodilated due to general anesthesia, such values often exist. Lakhal et al. [12] underscored that reality in their study of intraoperative patients with presumed hypovolemia. They found that increasing the threshold values to >23% markedly increased the positive predictive value of these test to identify volume responders. Under these conditions either a volume challenge or a passive leg raising (PLR) maneuver to assess dynamic increases in cardiac output can be done [13]. Thus, the bedside clinician has the option to examine real-time PPV or SVV during positive-pressure breathing or the dynamic changes in cardiac output in response to a PLR maneuver in assessing fluid responsiveness without the need to give a fluid bolus. A clear example of using PLR to guide fluid therapy in a difficult patient was recently presented as a case conference [14]

Since the perturbation causing these cyclic changes in flow is dependent of the cyclic changes in intrathoracic pressure, tidal volume, a major determinant of changes in intrathoracic pressure, needs to be great enough to alter central venous pressure. Thus, tidal volumes of ≥6 ml/kg decrease the sensitivity but not specificity of this parameter [9,15]. Similarly, in the presence of intra-abdominal hypertension, chest wall compliance is markedly decreased. This must alter venous return and blood flow distribution. Still, PPV and SVV remain sensitive and specific if tidal volume is maintained [16,17]. However, intra-abdominal hypertension does reduce the sensitivity of the passive leg raising (PLR) test to identify volume responsiveness [18], presumably because intra-abdominal hypertension increases during the PLR maneuver altering unstressed blood volume [18].

Although PPV can be computed at the bedside by direct analysis of the arterial pressure waveform signal [3] and by personal software analysis of such signals [19] most clinicians use commercially-available minimally-invasive monitoring devices that used arterial pressure waveform analysis from an indwelling arterial catheter or an estimate of the arterial

waveform using a plethysmographic signal from a finger cuff. Hadian et al. compared the three most commonly used minimally-invasive devices: PiCCO, LiDCO and FloTrac to each other and a paired thermodilution cardiac output measured from a pulmonary artery catheter in 17 post-operative surgical patients. They showed that although all devices gave similar mean cardiac output values, their trending and dynamic responses correlated poorly with each other. In a recent review, Stagt examined the accuracy of the latest generation software Flotrac system and found it acceptable for clinical use in the operating room [20]. This is important, because FloTrac is often used to compare cardiac output and SVV accuracy of other devices. For example, others showed that a similar minimally-invasive device, IntelliVue MP was as accurate as FloTrac in defining mean cardiac output values in 47 septic shock patients [21]. Most likely any new devise that used arterial pulse pressure to estimate flow will show similar degrees of general accuracy and poorer degrees of trending accuracy. Similarly, Vos et al. [22] compared the ability of a non-invasive plethysmographic measure of arterial pressure (Massimo Radical) with invasive FloTrac to estimate cardiac output in 30 patients undergoing major hepatic resection. They found that the new non-invasive device gave similar cardiac output and SVV values. In contrast to these authors, Monnet et al. [23] showed that when arterial tone was altered by norepinephrine in critically ill patients, FloTrac poorly tracked changes in cardiac output. Presumably the FloTrac algorithm accuracy degrades when large changes in arterial impedance occurs, as may occur with the use of norepinephrine or when acute endotoxin induces shock [24].

Another insightful study was done by Marik et al. [25] who used a combined non-invasive estimate of cardiac output by bioreactance (NICOM) with regional measures of cerebral blood flow by carotid Doppler in 34 critically ill patients. Volume responsiveness was assessed by the PLR maneuver in non-ventilated patients and SVV in 19 mechanically ventilated patients. Only half their patients were volume responsive. Importantly, only the volume responders displayed an increase in carotid flow (79±32%). Furthermore, if carotid flow were used as the non-invasive estimate of volume responders to a PLR maneuver, a threshold value of 20% separated responders from non-responders with a high sensitivity and specificity. Since sustaining adequate cerebral blood flow is an important resuscitation target, these data suggest that in severely ill patients measures of carotid blood flow changes in response to a PLR maneuver may greatly augment the functional hemodynamic useful of these measures.

Fluid therapy is one of the first steps in the goal-directed therapy [26] and is a central part of the Surviving Sepsis Guidelines [27]. Thus, an adequate assessment of fluid responsiveness should improve the therapy. One study [19] conducted in high risk surgery patients showed that a volume loading guided to the goal of PPV minimization improved postoperative outcome and decreases length of hospital stay. Patients in the interventional group received more fluid than the control group and had also less postoperative complications, lower duration of mechanical ventilation and lower stay in the intensive care unit.

Assessing arterial tone

Although PPV and SVV cannot be accurate interpreted as measures of volume responsiveness in patients with atrial fibrillation, their ratio always defines dynamic central

arterial elastance (Ea)[28]. If the arterial circuit becomes stiffer, then for the same stroke volume change, arterial pulse pressure will change more and vice versa. Using this approach, Monge et al. [29] assessed the effect of volume loading on arterial pressure in hypotensive septic shock patients whose PPV predicted that they were volume responsive. All patients increased their cardiac output, as expected, in response to the fluid challenge, but only those patients with normal or increased Ea also increased their arterial pressure. Importantly, they could not predict who would increase their arterial pressure based on pre-challenge measures of systemic vascular resistance, mean arterial pressure or the ratio of arterial pulse pressure to stroke volume, only the PPV/SVV slope defining Ea predicted responders from non-responders. They found that Ea <0.9 reflected a severely vasodilator state. In support of this study, Hadian et al. [30] demonstrated that when post-operative cardiac surgery patients were given vasodilator therapy they significantly decreased Ea from 1.44 to 1.13. Collectively, these data strongly support the use of PPV/SVV estimates of Ea as part of the overall assessment strategy of critically ill patients. Indeed if arterial tone is markedly decreased PPV may not reliably track SVV at all [31].

Identification of cardiovascular insufficiency

Cardiovascular insufficiency is characterized by an inadequate O_2 delivery relative to the metabolic demands. Shock can be, in the early stages, compensated by autonomic mechanisms, such regional vasoconstriction, in an attempt to maintain central blood pressure and vital organ perfusion above an anaerobic threshold. In this stage of compensated shock, microcirculatory measures like arterial pressure or cardiac output are often inside the range of values defined as normal and, therefore, insensitive as early predictors of subsequent decompensation due to the increased risk of tissue ischemia and subsequent development of multi-organ failure and death. However, microcirculation alterations in muscle and skin blood flow already occur in these early stages and measures of tissue cardiovascular reserve should be a sensitive early warning measure of impending cardiovascular collapse. Thus, a valid method to assess the microcirculatory status such the non-invasive measurement of tissue oxygen saturation (StO_2) when coupled to a Functional Hemodynamic Monitoring test, such as the Vascular Occlusion Test (VOT), may allow early identification of compensated circulatory shock and thus guide initial resuscitation efforts.

Non-invasive measurement of StO_2 using near-infrared spectroscopy (NIRS) has been shown as a valid method to assess the microcirculation status, especially in septic and trauma patients. The absolute StO_2 value has a limited discriminating capacity because StO_2 remains within the normal range until shock is quite advanced. But the addition of a dynamic ischemic challenge such as the VOT, improves and expands the predictive ability of StO_2 to identify tissue hypoperfusion [32]. The VOT measures the effect of total vascular occlusion-induced tissue ischemia and release on downstream StO_2. StO_2 is measured on the thenar eminence and transient rapid vascular occlusion of the arm by sphygmomanometer inflation to 30 mm Hg above systolic pressure is performed either for a defined time interval, usually 3 min, or until StO_2 declines to some threshold minimal value, usually 40%. The deoxygenation rate (DeO_2) reflects the local metabolic rate and mitochondrial function, and the rate of reoxygenation rate (ReO_2) reflects local cardiovascular reserve and microcirculatory flow.

Microcirculatory failure during shock is a major component of the end-organ dysfunction. Such microcirculatory dysfunction can be characterized by oxygen shunting, vasoconstriction, thrombosis and tissue edema. The flow distribution within the tissue is impaired [33] but improves rapidly in septic shock survivors whereas patients dying by organ failure have a lower percentage of perfused small vessels [34].

Creteur et al [35] showed that the alterations in VOT StO_2 response are related to the outcome in patients with either severe sepsis or septic shock. Furthermore, when comparing to hemodynamically stable patients without infection (controls) and healthy volunteers, the difference in the septic patients were striking. Using NIRS VOT StO_2 they assessed the slope of increase in StO_2 release as well as by the difference between the maximum StO_2 and the StO_2 baseline (Δ). Both, the slope of ReO_2 and the Δ were significantly lower in septic patients than in controls and healthy volunteers. In the sample of septic patients, the slopes were also significantly lower in the ones who had cardiovascular insufficiency. ReO_2 slopes were higher in survivors than in non-survivors and also tended to increase during resuscitation in survivors but not in non-survivors. Finally, the ReO_2 slope was found to be a good predictor of ICU death, with a cut-off value of 2.55%/sec (sensitivity 85%, specificity 73%). These data confirm that the alterations in VOT StO_2 ReO_2 are related more to the sepsis process itself and its severity than to mean arterial pressure or vasopressor agent's dose. Importantly, the magnitude of this ReO_2 slope alteration is directly related to the septic disease and their presence in the first 24 hours of septic process and their persistence of delayed ReO_2 slope is related to patient's outcome. Still, if the StO_2 ReO_2 does reflect inadequate tissue perfusion then it should also sensitive of an impending cardiovascular insufficiency state (compensated shock) if matched with other static measures of tissue ischemia.

To address this issue further, Mesquida et al. [36] followed the StO_2 VOT in septic patients showing that impaired ReO_2 predicted organ failure. StO_2 VOT-derived estimation of cardiovascular stress during a spontaneous breathing trials (increased DeO_2) also identified patients who subsequently failure that trial [37]. Furthermore, Guyette et al. [38], measured both the VOT StO_2 as baseline serum lactate, known to define existing cardiovascular insufficiency in trauma, in a cohort of trauma patients during the air transport to the Trauma Center. The aim of the study was to see if the StO_2 measurement, including a VOT, was feasible in the prehospital environment and useful to predict in-hospital death and intensive care unit (ICU) admission. Not surprisingly, they did not find differences in baseline StO_2 between survivors, non-survivors and patients admitted to the ICU, they showed significant differences in DeO_2 and ReO_2 slopes between survivors and non survivors, as well as between patients who need ICU admission and patients who did not. Furthermore, only one of the five patient deaths in their sample had prehospital vitals signs that would have met the protocolized criteria for resuscitation (heart rate >120 bpm, systolic blood pressure < 90 mmHg). Importantly, serum lactate alone was no better than lowest systolic pressure in predicting those in need of Life Saving Interventions (LSI) or death, but if the baseline serum lactate was >1.7 mmol/dl the ReO_2 was 100% specific for the need of LSI. This study shows the usefulness of the microcirculation dynamic assessment in the early stages of the trauma injury, when the cardiovascular insufficiency is not suspected with the

macrocirculatory indexes, providing the possibility to start early the appropriate treatment and decide the in-hospital disposition.

Conclusions

Functional hemodynamic monitoring is the pluripotential approach to interpolation of physiological data using a proactive intervention to create emerging parameters of robust sensitivity and specificity to identify cardiovascular insufficiency, volume responsiveness and vasomotor tone. When coupled with effective treatment goals it can markedly improve the accuracy y and simplicity of decision rules needed to drive resuscitation.

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Key Points

- Functional hemodynamic monitoring is the pluripotential approach to interpolation of physiological data using a proactive intervention to create emerging parameters of robust sensitivity and specificity to identify cardiovascular insufficiency, volume responsiveness and vasomotor tone.
- It needs to be considered within the broader aspects of risk stratification to reach its full potential.
- Since early goal-directed therapy algorithms need to resuscitate to circulatory sufficiency, defining end-points become as important as defining treatments to initiate.
- This field is rapidly expanding and has potential application for assessment of regional perfusion and function and across all acute care disciplines.