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REVIEW

Advanced Hemodynamic Monitoring: Principles and Practice in Neurocritical Care

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Abstract Advanced hemodynamic monitoring is necessary for many patients with acute brain and/or spinal cord injury. Optimizing cerebral and systemic physiology requires multi-organ system function monitoring. Hemodynamic manipulations are cardinal among interventions to regulate cerebral perfusion pressure and cerebral blood flow. The pulmonary artery catheter is not any more the sole tool available; less invasive and potentially more accurate methodologies have been developed and employed in the operating room and among diverse critically ill populations. These include transpulmonary thermodilution, arterial pressure pulse contour, and waveform analysis and bedside critical care ultrasound. A thorough understanding of hemodynamics and of the available monitoring modalities is an essential skill for the neurointensivist.

Keywords Hemodynamic monitoring · Subarachnoid hemorrhage · Traumatic brain injury · Mechanical ventilation · Cardiac output · Transpulmonary thermodilution · Bedside echocardiography

Introduction

Hemodynamic monitoring comprises one of the cornerstones of intensive care medicine. Neurocritical care patients often require such monitoring, specifically to optimize cerebral blood flow (CBF) and brain tissue oxygen delivery in addition to managing conditions commonly seen in critically ill patients including shock states and acute lung injury (ALI/ARDS). Manipulation of the cardiac output (CO), mean arterial pressure (MAP), systemic filling pressures, and volumes as well as dynamic markers of fluid responsiveness, requires continuous monitoring, thorough understanding of the modalities employed and proper interpretation of data acquired. Traditionally, invasive hemodynamic monitoring is equated to the use of a pulmonary artery catheter (PAC). Swan-Ganz catheter usage has more recently been tempered secondary to a lack of evidence on improving outcomes in randomized controlled trials [1–3] and also due to the poor performance of filling pressures as indicators of fluid responsiveness [4]; nevertheless PAC use may still be appropriate in selected patients and its use has been reviewed elsewhere [5]. In this review, we will focus on less invasive modalities including single-indicator transpulmonary thermodilution, arterial waveform and pulse contour analysis as well as bedside real-time cardiorespiratory sonography.

Cardiac Output Derivations

Traditional calculation of CO via PAC employ a temperature–time curve derived from measurements obtained in the pulmonary artery after injection of cold saline. During single-indicator (thermal) transpulmonary thermodilution (TP), CO measurements are based on cold saline injection through a regular central venous catheter (internal jugular or subclavian vein). The thermistor is located at an arterial site (radial, axillary, or more commonly in the femoral artery) comprised by a 4–5 Fr catheter; in similar fashion, a temperature–time curve is constructed and the CO

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calculated via the Stewart-Hamilton principle [6]. Estimations of CO via PAC as compared with transpulmonary thermodilution have shown high degree of correlation in both experimental [7] and clinical settings such as in the operating room and the intensive care unit (ICU) [8, 9]. Currently, the method commonly used for transpulmonary thermodilution employs the PiCCO system (Pulsion Medical Systems, Munich, Germany and Philips Medical Systems) which also uses arterial pulse contour analysis for the continuous display and monitoring of CO (http://www.pulsion.com/index.php). Via mathematical modeling that incorporates vascular resistance, impedance and capacitance, a flow-time curve is created on the basis of a given arterial trace [10–12]. Thermodilution-generated CO measurements are then employed to calibrate the pulse-contour continuous CO analysis, a procedure done in scheduled time intervals (e.g., every 8 h). Recalibration is also advisable when the hemodynamic/physiologic vascular state of a given patient changes to ensure accurate continuous CO monitoring [13, 14]. Mutoh et al. [15] compared PAC measurements of CO to transpulmonary thermodilution using PiCCO derived CO in a neurocritical care population, patients with aneurysmal subarachnoid hemorrhage (SAH). In this study, simultaneous measurements were performed in 16 patients. A total of 263 measurements were recorded over a mean of 6 days. Results of analysis of pooled data for cardiac index (CI) values showed high correlations and close agreement among PAC-CI, TPCI, and TP calibrated pulse-contour CI (bias and precision according to the Bland–Altman plot were 0.05 and 0.11 l/min/m^2 and $\pm$0.25 and $\pm$0.33 l/min/m^2 with percentage errors of 13.5 and 18.0%, respectively for TPCI and pulse-contour CI with PAC-CI).

Supplementary to CO measurements for the assessment of left ventricular (LV) contractility, via TP a cardiac function index (CFI) is calculated. That is the ratio of CO over the blood volume contained in the four chambers of the heart at end-diastole (global end-diastolic volume-GEDV and index-GEDI) [16]. CFI was recently compared with echocardiographic LV ejection fraction (LVEF) in critically ill patients with circulatory failure. A CFI <3.2 min^{-1} predicted LVEF of $\leq$35% with a sensitivity of 81% and specificity of 88%, caution should be exercised in patients with right ventricular (RV) dilation [17].

An alternative method to derive continuous CO from the properties of the arterial waveform is the one used by FloTrac/Vigileo (Edwards LifeSciences, Irvine, CA) [18]. This is a self-calibrating algorithm (does not require thermodilution) that incorporates a proprietary multivariate equation taking into account biometric variables related to arterial compliance (e.g., age and sex) and geometric variables describing the arterial pressure waveform [19, 20]. This equation was developed from and validated in a human database of arterial pressure tracings and thermodilution CO reference values. FloTrac has undergone modifications, and recently De Baker et al. performed a multicenter validation of the third-generation software in a cohort of septic patients [21, 22]. There has been concern, with the earlier version, of underestimating cardiac output in pathophysiologic states of increased arterial compliance and vasodilatory shock [23–25].

Volume Status and Fluid Responsiveness

Preload assessment and fluid responsiveness is a routine exercise in critically ill patients, and it becomes of vital importance during hemodynamic management. It can often be challenging in the ICU patient since there is no single clinical gold standard. Furthermore, the decision to administer fluids should not be taken lightly in view of accruing evidence on the harmful consequences of positive fluid balance in both the neurologic [26] and general ICU patients [27, 28]. The frequently employed static preload filling-pressure markers like central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP) have been shown to correlate poorly with ventricular filling volumes and fluid responsiveness in healthy volunteers [4] and critically ill patients [29], leading investigators to discourage their use for the purpose of fluid management [30, 31]. Another common practice is the one following daily fluid balances (DFB) to ascertain euvolemia. Specifically in patients suffering from SAH, current guidelines advise maintenance of circulating blood volume and avoidance of hypervolemia [32]. Different authors have documented poor correlation of DFB with actual circulating blood volume, in patients with SAH, as measured by integrated pulse spectrophotometry and pulse dye densitometry, making DFB an unreliable indicator [33, 34].

The ultimate goal of volume expansion is augmentation of stroke volume requiring biventricular preload dependence. Stated on Frank-Starling terms, it requires both the ventricles to be operating on the ascending part of their performance curves. Prediction of the performance of the ventricles on the Frank-Starling curve, hence the ability of volume expansion to enhance stroke volume in the individual patient, has dramatically improved by the use of dynamic variations in the arterial-waveform due to heart–lung interactions during positive pressure mechanical ventilation [35–37]. These interactions result from the effects of pleural and transpulmonary pressures on vascular and cardiac chambers. The main components, on the right side of the heart, include decreased venous return accompanied by increased RV afterload leading to reduced LV preload [38]. Opposite for the left side, the increase in pleural pressure leads to a reduction of afterload [38, 39].

The larger these effects are, directly dependent on intravascular volume, chamber compliance and contractility, the more pronounced the variation in LV stroke volume. It is exactly this stroke volume variation (SVV) that has been shown to be the most accurate predictor of fluid responsiveness [40]. Systolic pressure variation (SPV), pulse pressure variation (PPV), and SVV can all be derived by analysis of the arterial blood pressure waveform as obtained by routine invasive arterial monitoring. They can be quantified, continuously monitored, and displayed. Pulse pressure variation has been shown to perform superiorly to SPV reflecting the property of pulse pressure as a surrogate of stroke volume. Michard et al. [35] found that a PPV $>13\%$ predicted a response (defined as a 15% increase in CI) to a 500 ml fluid bolus with a positive predictive value of 94% and for PPV $<13\%$ a negative predictive value of 96%. In this same study and from the receiver operating characteristic curves comparing the predictive ability of the different markers, it can be seen that CVP and PAOP are no more predictive than a coin toss.

To properly use these dynamic markers of fluid responsiveness, some important limitations should be kept in mind. Michard [40] summarized them under the acronym "SOS". The first S, stands for small tidal volume (TV) and spontaneous breathing activity and the "O" for open chest conditions. Small TV, as the ones used under lung protective strategies, may not induce stroke volume variation in an otherwise fluid responsive patient leading to a false negative result. Most studies evaluating these markers were validated in mechanically ventilated, heavily sedated if not chemically paralyzed, patients with 8–10 ml/kg tidal volumes and shown reliable only with a TV of at least 8 ml/kg [41]. Huang et al. [42] studied patients with ARDS, ventilated with a mean TV of 6.4 ml/kg and found that a PPV cut-off of 12% discriminated fluid-responders with a specificity of a 100%; sensitivity was only 68% and the number of subjects in the study small. In order to overcome these shortcomings and for only brief physiologic testing, the clinician may elect to temporarily increase the TV to 8 ml/kg and/or use a short acting paralytic agent to eliminate spontaneous breathing and improve patient-ventilator synchrony. This would increase the accuracy of dynamic preload variables and provide an opportunity to further investigate and optimize ventilator settings, using, for example, the airway pressure–time curve profile (stress index [43]) to detect tidal recruitment/hyperinflation in patients with lung injury. The last "S" stands for sustained cardiac arrhythmia as in the case of atrial fibrillation where the observed beat-to-beat stroke volume variation is secondary to altered cardiac filling times. In addition, caution with a potentially false positive result of the PPV can be seen in patients with RV dysfunction (e.g., patients with ARDS ventilated with high airway pressures). These patients, despite a PPV $>13\%$ may not respond to a fluid challenge [44, 45].

A simple clinical bedside test that overcomes some of these limitations, like spontaneous breathing, is the passive leg raise (PLR) test. This involves elevation of the patient's legs to 45° leading to an autotransfusion of volume pooled in the lower extremities and pelvic veins. Detection of an increased CI with this maneuver has been shown to be an accurate predictor of fluid responsiveness [46–48]. PLR has not been tested in a neurocritical care population and caution should be exercised in patients with compromised intracranial compliance where a sudden change in body position could lead to an increase in intracranial pressure. This precaution would only exclude a minority of extremely position-sensitive patients since PLR takes only few minutes to be performed and yield information.

Bedside, ultrasound is an extremely valuable tool in hemodynamic assessments and complements the techniques mentioned above; it is becoming an essential skill and requirement for all intensivists. Using transthoracic echocardiography (TTE) and via the parasternal short axis view, the finding of systolic obliteration of the left ventricular cross sectional area ("kissing" papillary muscles sign) is considered to indicate severe hypovolemia [49]. In addition, Feissel et al. and Barbier et al. demonstrated that the quantification of respiratory variation in the diameter of the inferior vena cava (dIVC) by the use of bedside ultrasonography is a simple and accurate marker of fluid responsiveness in mechanically ventilated patients. In both studies, with only slightly different cutoffs for dIVC (Feissel 12% and Barbier 18%) fluid responders and non-responders could be discriminated with sensitivity and specificity $\geq 90\%$ [50, 51]. Recently the use of this technique was reproduced in a population of 29 patients receiving mechanical ventilation and advanced hemodynamic monitoring for the management of SAH. Moretti and Pizzi [52] found a cutoff dIVC $>16\%$ to be 70% sensitive and 100% specific as a predictor of fluid responsiveness. It should be understood that similar limitations as the ones reported earlier like spontaneous breathing and TV could also decrease the accuracy of dIVC as a marker.

Goal-Directed Hemodynamic Management in the Neuro-ICU

Pressure-flow augmentation and volume status manipulation is commonly employed in the setting of prevention and treatment of delayed cerebral ischemia (DCI) after SAH and during CPP/ICP guided therapy in TBI. In SAH, this particularly becomes relevant in patients who develop neurogenic stress cardiomyopathy or neurogenic pulmonary

edema (NPE) independently or concurrently with hemodynamic augmentation for DCI [53–55]. Continuous measurement of MAP and CI becomes imperative and indirect reconstruction of Frank-Starling curves (see PPV discussion earlier) can further assist in titration of inotropes, vasopressors, diuretics, or fluids.

Furthermore and via TP, filling volumes (GEDI) and quantification of pulmonary edema or extra-vascular lung water (EVLW) are available to the clinician (calculation principles of filling volumes and EVLW are graphically explained in the Fig. 1, for in depth discussion the reader is referred to Isakow and Schuster [6]). The EVLW measurement by single TP has been validated against the gold-standard gravimetric method of pulmonary edema quantification, previously in animal models [56] and recently in a human autopsy study [57]. An increased EVLWI has been independently associated with poor prognosis and increased mortality in ICU patients [58, 59]. The pathophysiology of NPE is not fully elucidated, and mechanisms of both hydrostatic and high permeability edema formation have been implicated [60]. Deciphering the predominant mechanism in the individual patient could lead to specific interventions. Monnet et al. [61] investigated two indices of pulmonary permeability in 48 patients mechanically ventilated for acute respiratory failure. Via TP, they calculated the ratios EVLW/Pulmonary blood volume (PVPI) and EVLWI/GEDI. A PVPI $\geq$3 and an EVLWI/GEDI $\geq 1.8 \times 10^{-2}$ allowed the diagnosis of ALI/ARDS (vs. hydrostatic pulmonary edema) with a sensitivity of 85% and specificity of 100%.

Bedside, basic critical care echocardiography to assess global LV/RV size and systolic function, can be taught to and be performed by non-cardiologists [62]. TTE by the neurointensivist in the hemodynamically unstable patient with SAH can be extremely useful in diagnosing severely reduced CO in association with neurogenic stress cardiomyopathy or apical ballooning syndrome. Complementary information can be obtained by bedside lung ultrasound to detect interstitial edema as a precursor to alveolar edema as shown by Lichtenstein et al. The authors describe a high association between the "B-predominance" and pulmonary edema. The B-line is a comet-tail ultrasound artifact generated by edematous subpleural interlobular septa surrounded by air filled alveoli [63].

Delayed cerebral ischemia is a major complication after SAH, traditionally treated with the so-called "triple-H" therapy (Hypertension, Hypervolemia, Hemodilution) [64, 65]. This regimen has never been prospectively tested in a randomized controlled fashion, and the individual components risk/benefit ratios are not well documented [66–68].

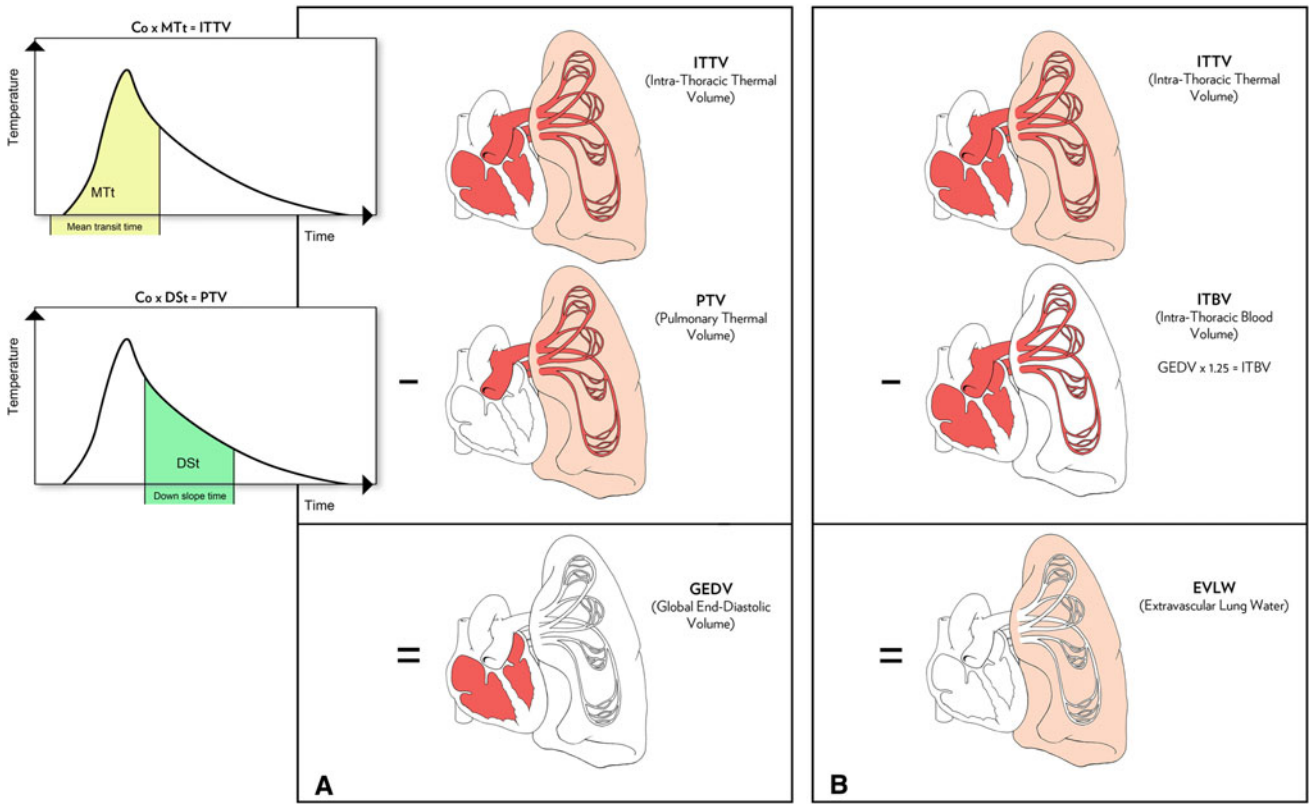

Fig. 1 Transpulmonary thermodilution—calculation of filling volumes and extravascular lung water

Table 1 Advanced hemodynamic monitoring—general goals and hemodynamic augmentation

General goals–normal ranges
LV performance
CI = 3.0–5.0 l/min/m^2
CFI = 4.5–6.5 min^{-1} (<3.2 predictive of EF <35%)
TTE = Visual inspection of LV/RV size and contractility
Preload
GEDI = 680–800 ml/m^2
TTE = LV CSA obliteration indicates hyperdynamic-hypovolemic ventricle
Fluid responsiveness (responders)
SVV >10%
PPV >13%
dIVC >16%
Accurate in controlled MV settings, synchronous with the ventilator, caution with small TV, no afib/flutter. PLR if limitations
Pulmonary edema
ELWI = 3.0–10.0 ml/kg
Lung US = A-line predominance
Hemodynamic augmentation
Normovolemic hypertension (Norepi/Neo/Dopa high-dose)
BP increments of 20%—titrate to exam, tolerance and indices of pressure autoregulation, brain oxygenation and/or microdialysis data
Preload + fluid responsiveness markers (general goals)
Primary CO augmentation (Dobutamine/Milrinone)
CI >3.5—titrate as above
Preload + fluid responsiveness markers (general goals)
Mild hypervolemia allowed under monitoring of ELWI and B-lines, fluid administration guided by dynamic markers

It appears that normovolemic hypertension may be superior in achieving CBF augmentation without compromising systemic and brain oxygenation [67, 69]. Also, isolated augmentation of the CI has been shown effective in reversing delayed ischemic deficits, independently of blood pressure [70, 71]. Use of advanced hemodynamic monitoring (AHM) could assist in optimizing these hemodynamic variables to specific clinical goals and/or direct measures of CBF and tissue parameters like brain tissue oxygenation ($PbtO_2$) and metabolic-microdialysis data. Importantly, AHM could be employed to limit the harmful effects of vasopressors or hypervolemia [69, 72]. Normal ranges for various AHM modalities and a suggested hemodynamic protocol can be found in the Table 1.

Management of severe TBI is centered in optimizing CPP and controlling ICP. Individualizing CPP targets taking into account the state of pressure autoregulation, brain oxygenation, and tissue metabolic parameters is encouraged [73]. On the contrary, indiscriminate, aggressive CPP augmentation is considered potentially harmful leading to ALI/ARDS [73, 74]. Fluid-volume goals, in these patients are complicated and often competing in the presence of multi-organ dysfunction; both hypovolemia and hypervolemia have been linked with unwanted effects [74–76]. Studies investigating brain oxygenation by Contant et al. [74] and Martini et al. [77] in patients with severe TBI, serve as caution to the side effects of management targeted to individual physiologic end-points.

Conclusion

Management of neurocritically ill patients requires understanding and monitoring of multiple organ systems and the interplay among them. Physiologic goals can be competing and interventions have to be chosen based on a consideration of their risk/benefit profile. Advanced hemodynamic monitoring, invasive, non-invasive, and in multiple forms including the PAC, transpulmonary thermodilution, arterial pulse contour/waveform analysis, and ultrasound, constitute a powerful instrument. Monitoring, by itself cannot be expected to improve outcomes. Interpretation of data and the clinical decisions that come as a result, necessitate thorough understanding of the physiologic principles and "mechanics" of the available modalities. Prospective studies assessing clinical protocols employing neurologic and systemic physiologic monitoring variables are needed to establish their safety and value. The addition of goal-directed regiments targeting tissue oxygenation and metabolism makes AHM an important tool in an effort to select potentially beneficial from harmful interventions and materialize better patient outcomes.

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