Postoperative hemodynamic instability and monitoring

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Purpose of review
The purpose of the review is to identify the recently validated minimally invasive or noninvasive monitoring devices used to both monitor and guide resuscitation in the critically ill patients.

Recent findings
Recent advances in noninvasive measures of blood pressure, blood flow, and vascular tone have been validated and complement existing minimally invasive and invasive monitoring techniques. These monitoring approaches should be used within the context of a focused physical examination and static vital sign analysis. When available, measurement of urinary output is often included. All studies show that minimally invasive and noninvasive measure of arterial pressure and cardiac output are possible and often remain as accurate as invasive measures. The noninvasive techniques degrade in severe circulatory failure and the use of vasopressor therapy. Importantly, these output parameters form the treatment goals for many goal-directed therapies protocols.

Summary
When coupled with a focused physical examination and functional hemodynamic monitoring analyses, these measures become even more specific at defining volume responsiveness and vasomotor tone and can be used to drive resuscitation strategies.

Keywords
arterial tone, functional hemodynamics, goal-directed therapy, minimally invasive monitoring, shock, volume responsiveness

INTRODUCTION

An estimated 230 million surgical procedures are performed each year around the world [1]. Approximately 18% of patients undergoing surgery will develop a major postoperative complication [1–4] and these complications remain an important factor in determining functional recovery and long-term survival [5]. For this reason, appropriate management and proactive evaluation will be very important for the patients as well as the healthcare providers.

Hypovolemia and cardiac dysfunction, leading to insufficient tissue perfusion and oxygenation, are the leading causes of perioperative complications and poor outcomes [6–9]. Effective fluid management to prevent and treat hypovolemia and administration of vasoactive medications for cardiac and vascular dysfunction are crucial to maintain oxygen delivery and prevent intravascular volume disturbances [10–12]. Therefore, placing the most appropriate hemodynamic monitoring devices to guide perioperative hemodynamic optimization is an important first step in reducing the risk of complications. Importantly, the host's baseline physiologic status and the seriousness of the surgery are primary determinates of outcome. Although less than 15% of the procedures are performed in high-risk patients, these patients account for 80% of in-hospital deaths [13–15]. Relevant to this reality, a recent 'consensus of 12' study on perioperative cardiovascular monitoring of high-risk patients [16▪▪] concluded that adequate and focused hemodynamic monitoring and early appropriate therapy can improve outcome in these high-risk surgical patients.

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Curr Opin Crit Care 2016, 22:393–400
DOI:10.1097/MCC.0000000000000320

KEY POINTS

- Monitoring is only one modality of the overall process of patient assessment.
- Recently, technical advances make the measure of CO at the bedside commonplace and accurate.
- In the absence of severe vasoplegia and increased vasopressor use, noninvasive–invasive monitoring has similar accuracy to minimally invasive techniques.
- All monitoring needs to be linked to treatments.

Clinical examination continues to be an important initial step in the hemodynamic evaluation of the high-risk patient. The primary survey can quickly identify any concern for cardiorespiratory insufficiency and is helped by the simple mnemonic A-B-C-D-E, representing airway, breathing, circulation, disability (functional status), and exposure (direct visualization of the patient) (Table 1). A primary goal of hemodynamic monitoring, within the concept of initial evaluation and management, is to evaluate cardiopulmonary function, cardiovascular reserve, and the adequacy of blood flow and oxygen delivery to the tissues and, if deemed inadequate, monitor the impact of therapies directed at restoring cardiopulmonary sufficiency. Hemodynamic monitoring can range from basic to advanced, noninvasive to invasive, intermittent to continuous, and static to dynamic measurements. Table 2 lists the various monitoring devices by their invasiveness, sample frequency, and quality of the physiologic parameter they monitor.

Table 1. Components of the primary survey

Step	System	Description
A	Airway patency/ maintenance	Ensure patency (breath sounds and capnometry) with application of oxygen
B	Breathing	Verify adequate oxygenation (SpO_2 and ABG) and ventilation ($etCO_2$ and ABG) and auscultation to reveal pneumothorax, bronchospasm, or edema
C	Circulation (with hemorrhage)	Blood pressure, ECG, heart rate, urine output as well as looking for internal or external sources of bleeding (i.e., drains)
D	Disability (neurologic evaluation)	Neurologic examination to rule out stroke or seizure if hypoxia, hypovolemia, hypoglycemia, and residual anesthetic ruled out
E	Exposure	Direct and thorough head-to-toe examination

ABG, arterial blood gas; $etCO_2$, end-tidal CO_2; SpO_2, oxyhemoglobin saturation.

BASIC HEMODYNAMIC MONITORING

Basic hemodynamic monitoring in the ICU for identification and treatment of overall cardiopulmonary sufficiency includes a focused history, physical examination, and the noninvasive assessment of primary hemodynamic variables, such as vital signs [i.e., heart rate, mean arterial pressure (MAP), respiratory rate, temperature, and pulse oximetry O_2 saturation] and, if available, urine output [17]. However, these primary variables and the physical examination have repeatedly proven insufficient and inaccurate for hemodynamic evaluation, rapid assessment, and identification of occult or compensated shock, especially in the previously healthy patient and when cardiopulmonary status is changing quickly [7,18–22]. Biochemical markers of tissue hypoperfusion (e.g., lactate, metabolic acidosis, and central venous oxygen saturation) because of cardiovascular insufficiency may be abnormal indicating occult tissue hypoperfusion even without systemic hypotension or other overt clinical signs of shock [23,24]. Recently, Casserly *et al.* [25▪] have again demonstrated markedly increased mortality in septic patients when lactate was greater than 4 mmol/l, even in the absence of hypotension. Whether hyperlacticemia reflects tissue hypoperfusion or overwhelming inflammation in the setting of sepsis is unknown, but hyperlacticemia is universally a poor prognostic sign, even if not being useful in guiding resuscitation.

ADVANCED HEMODYNAMIC MONITORING

If an initial intervention (i.e., fluid bolus) does not rapidly reverse the shock state, restore arterial pressure, and organ perfusion, it is imperative to collect more focused physiologic variables. Continuous measures of arterial pressure, cardiac output (CO), and blood oxygenation are used to better monitor the critically ill patient [26▪▪]. The continuous monitoring of these advanced variables has allowed for the development of hemodynamic goal-directed resuscitation and treatment of shock.

However, the transition from basic to advanced hemodynamic monitoring is artificial at best. As many very important hemodynamic values, like continuous measuring of arterial waveforms and CO, are now potentially accurately estimated using completely noninvasive methodologies, separating

Table 2. List of various hemodynamic monitoring devices and their associated physiologic measures

Monitor (brand names)	Invasiveness	Sample frequency	Physiologic measures
Pulse oximeter (various)	Noninvasive	Continuous	SpO_2 and hemoglobin
Capnometry (various)	Noninvasive	Continuous	Capnometry and $etCO_2$
Plethysmography variability (Masimo)	Noninvasive	Continuous	PVI
Noninvasive blood pressure (Dynamat)	Noninvasive	Intermittent (maximum every minute)	Systolic, diastolic, and mean systemic pressure
Electrocardiogram (various)	Noninvasive	Continuous	Rate, rhythm, ischemia, and dynamic changes in stroke volume
TTE	Noninvasive	Intermittent	Contractility, volume responsiveness (inferior vena caval diameter changes), volume status (kissing papillary muscles), valve function, fractional area of contraction, and VTI
Continuous noninvasive arterial pressure (CNAP and ClearSight)	Noninvasive	Continuous	SBP, DBP, mean arterial blood pressure, PPV, CO, and SVV
Arterial catheterization	Invasive	Continuous	SBP, DBP, mean arterial blood pressure, and PPV
Arterial pulse contour devices (PiCCO, LiDCO, FloTrac, and MostCare)	Invasive	Continuous	PPV and SBP SPV, CO, and SVV
Indicator dilution cardiac output (PiCCOplus and Virgilio)	Invasive	Continuous	CO, extravascular lung water, and global cardiac volume
Central venous catheterization	Invasive	Continuous, intermittent, or continuous	CVP, $ScvO_2$
Pulmonary artery catheter (Swan-Ganz catheter)	Invasive	Continuous; intermittent	PAP, CVP, CO, SVR, SvO_2, and pulmonary artery occlusion pressure
Esophageal Doppler (CardiAQ)	Invasive	Continuous	VTI, stroke distance, and FTc
Transesophageal echocardiography (various)	Invasive	Intermittent	Contractility, volume status (superior vena caval diameter changes), volume status (kissing papillary muscles), valve function, fractional area of contraction, and CO

CO, cardiac output; CVP, central venous pressure; $etCO_2$, end-tidal CO_2; FTc, flow time corrected; PAP, pulmonary artery pressure; PPV, pulse pressure variation; PVI, plethysmography variability index; $ScvO_2$, central venous oximetry; SpO_2, oxyhemoglobin saturation; SPV, SBP variation; SvO_2, mixed venous oximetry; SVR, systemic vascular resistance; SVV, stroke volume variation; TTE, transthoracic echocardiography; VTI, velocity time interval.

basic from advanced monitoring based solely on invasiveness is misleading. Certain caveats continue to hold true. First, in the setting of profound circulatory shock, noninvasive measures of hemodynamics may be less accurate or may not trend dynamic changes as well as these same hemodynamic variables when measured invasively. Still, the ability to rapidly know real-time arterial pressure and its waveform, and calculate CO and its derived variables greatly increases the diagnostic and therapeutic options for the bedside clinician.

Although the pulmonary artery catheter is a pleuripotential monitoring device [17], its use in the management of critically ill patients is problematic [27]. If not used to identify specific treatable causes of hemodynamic insufficiency whose treatment improves outcome, the potential benefit of such a device will be minimal at best [28,29]. Indeed, a recent survey of over 2 million postoperative cardiac surgery patients revealed that pulmonary artery catheter use did not improve outcome when compared with large propensity-matched controls [30▪▪].

As circulatory shock is the inadequate delivery of oxygen to the tissues, it is dependent upon perfusion pressure (MAP) and flow. Except for the kidneys and heart, most organs and tissues autoregulate blood flow and local oxygen delivery (DO_2) using local adjustment of vasomotor tone. However, below a critical MAP threshold, autoregulation fails. Although minimal MAP thresholds for all patients and all organ systems are unknown and

controversial, MAP values less than 60 mmHg are below most patients' autoregulation thresholds and result in insufficient perfusion to the heart and other organs [17,31▪]. The duration and degree of hypotension below a MAP of 60–65 mmHg is well correlated with mortality and organ failure [32,33]. Consequently, most studies and guidelines target a minimum MAP of 65 mmHg during initial resuscitation of shock [34,35]. Except in patients with chronic hypertension or severe atherosclerosis, further augmentation of the MAP above this threshold provides no further benefit [36,37] and artificially increased vasomotor tone may actually decrease blood flow by constricting arterioles [38] and increased arrhythmias [37].

Initial resuscitation is often with intravenous crystalloid infusion (except in some cases of hemorrhagic or cardiogenic shock). However, only about half the hospitalized patients presenting circulatory shock are volume responsive [39]. Targeting a specific threshold central venous pressure (CVP) is not effective either unless CVP is very low (i.e., <2 mmHg) [40–42]. Marik and Cavallazzi [43] performed a meta-analysis of studies comparing CVP and ventricular stroke volume, CO, and fluid responsiveness showing no relationship [44]. If anything, a rising CVP in response to fluid infusion should be used as a stopping rule to further fluid infusion [45].

Consequently, intravenous crystalloid infusion, although the nearly universal initial therapy for hypotension and hypoperfusion, is particularly difficult to manage. Additionally, increasing observational and correlational data have associated positive fluid balance with mortality and organ failure, particularly acute lung injury/acute respiratory distress syndrome [46–50]. Furthermore, it is unclear from retrospective data whether administration of early inotropic/vasopressor support in place of or concurrent with volume expansion improves outcomes or harms patients [51–53]. Although enthusiasm for early goal-directed therapy has waned as physicians have become more aggressive with initial resuscitation, interest in postoptimization to sustain CO postoperatively remains a useful therapy. Importantly, Pearse *et al.* [54] performed a meta-analysis as part of their large multicenter prospective clinical trial of postoperative high DO_2 therapy in high-risk surgery patients. They showed that although their trial just missed significance, when coupled with all other published clinical trials, significant survival benefit was achieved.

Transthoracic echocardiography (TTE) is considered an important step in examining a patient in shock to evaluate the type of shock and the cardiac function [55▪,56]. The left ventricular (LV) ejection fraction obtained by TTE depends on LV contractility and afterload so it must be interpreted with respect of the MAP. Poor contractility may indicate the need for inotropic support. Stroke volume can be approximated by 'kissing of the papillary muscles' and estimated by the product of the velocity-time integral of the subaortic flow and the area of the LV outflow tract. Measurement of the abdominal inferior vena caval diameter can give the clinician additional estimates of volume status. Finally, TTE is the gold standard to detect acute cor pulmonale from an acute increase in pulmonary vascular resistance by evaluating the right ventricular function and right-to-left size ratio [16▪▪,57].

FUNCTIONAL HEMODYNAMIC MONITORING

Functional hemodynamic monitoring is the measurement of the hemodynamic response to a predetermined intervention and the use of the result to define the pathophysiologic state of the patient and predict response to potential therapies [26▪▪,58,59▪▪]. Recent research has allowed the use of functional hemodynamic monitoring to predict: volume responsiveness; arterial vasomotor tone reactivity (elasticity); and microvascular tissue hypoxia because of cardiovascular insufficiency, even in the setting of compensated shock as measured by advanced hemodynamic monitoring of macrovascular indices.

VOLUME RESPONSIVENESS

Michard *et al.*'s [43] defined 'volume responsiveness': an increase in CO at least 15% in response to a 500 ml intravenous fluid infusion. Volume responders and nonresponders were distinguished by respiratory variation in arterial pulse pressure variation (PPV) of at least 13% on mechanical ventilation of at least 8 ml/kg tidal volumes. Accurately measuring PPV requires continuous hemodynamic monitoring and display of arterial pressure or the use of commercially available devices that do these calculations automatically. Since Michard *et al.* [43] original publication in 2000, literature supporting the reliability and reproducibility of PPV at least 13–15% to predict volume responsiveness has exploded [60,61]. Bedside use of PPV is now both well supported and easily accessible. Multiple commercial devices are now available to calculate and continuously display PPV, stroke volume variation (SVV), and cardiac index based on these principles [62].

Noninvasive measures of stroke volume variability have included echocardiographic measurement

of the velocity-time integral of aortic blood flow, analysis of the plethysmographic waveform variability, ultrasonographic assessment of inferior vena cava [63,64], superior vena cava [65], and internal jugular [66] diameter respiratory variations, and noninvasive measurement of carotid arterial blood flow and bioreactance by noninvasive CO monitoring [42,67]. However, PPV appears to be the most specific and sensitive predictor of volume responsiveness, even slightly better than SVV [68]. In a systematic review and analysis of pooled data, Marik *et al.* [61] calculated the correlation coefficient between PPV and increased cardiac index to be 0.78, whereas the same coefficient was 0.72 for SVV. Both were superior to estimates of volume responsiveness using static measures of 'volume status' (e.g., CVP, left end-diastolic volume index), which were no better than random chance.

Although robust and useful, the predictive value of PPV is restricted by confounding disease and therapies. Intra-abdominal hypertension, cardiac arrhythmia (e.g., atrial fibrillation), spontaneous breathing, decreased chest wall compliance, and a rapid relative risk relative to hazard ratio all may result in inaccurate PPV assessments [26▪▪]. When Richard and colleagues [69] performed a randomized control trial using PPV-guided fluid therapy in patients with septic shock, they could only apply this protocol in 9% of their cases because of the use of low tidal volume ventilation. In these settings, one can use the dynamic change in CO in response to a passive leg raising maneuver [70–72] or the increase in systolic arterial pressure during an end-expiratory hold maneuver [73]. Passive leg raising was found to be a highly robust indicator of volume responsiveness [74,75].

DYNAMIC ARTERIAL VASOMOTOR TONE, COMPLIANCE, AND ELASTANCE

Arterial compliance and elastance are reciprocal measures of the relationship between the change in volume and the change in pressure. Dynamic arterial compliance is defined as the ratio of SVV to PPV, and the dynamic elastance, or 'instantaneous stiffness,' is defined by the reciprocal ratio [76]. Therefore, one would anticipate that increased CO (i.e., increased SV with unchanged heart rate) would have a predictable effect on MAP response based on arterial elastance. Very low dynamic elastance would be associated with minimal changes in MAP as CO increased, and vice versa.

Monge García *et al.* [77] studied a population of hypotensive patients in acute circulatory shock who were all determined to be volume responsive (SVV $\geq$ 10%) before and after infusion of 500 ml hydroxyethyl starch, the patients who 'responded' by increasing their MAP at least 15% could only be distinguished from 'MAP nonresponders' by dynamic elastance (PPV/SVV). The area under the receiver operator curve for this prediction based on dynamic elastance before volume expansion was 0.986 ± 0.02 (95% confidence interval 0.84–1).

Although Pierrakos *et al.* [78] found an increased MAP after fluid challenge was given to 'responders' (defined by increased CO) but not in 'nonresponders' (no increased CO after fluid challenge), they were not able to correlate increased CO or SVV with MAP, as dynamic elastance was not used as a distinguishing factor. Similarly, when Monnet *et al.* [79] studied patients in septic shock treated with norepinephrine at baseline, the contribution of dynamic elastance may explain why the decreased rate of norepinephrine infusion alone (i.e., without intravenous fluid bolus) was shown to decrease static markers of preload (e.g., CVP and LV end-diastolic volume) and MAP, although CO (surrogate for volume responsiveness/preload dependency) did not decrease. The decreased static markers of preload increased by norepinephrine reduction may also be because of peripheral vasodilation, which would both increase an unstressed blood volume and decrease the resistance to venous return [80,81].

METABOLIC MEASURES OF TISSUE PERFUSION

Measures of oxygenation and CO_2 flux have traditionally been used to assess tissue perfusion. Similarly, hyperlacticemia and metabolic acidosis are excellent markers of shock severity, at least during the initial evaluation prior to therapy [82]. Although central venous oxygen saturation values less than 70% are predictive of circulatory stress, higher values do not exclude that diagnosis because venous blood is quite heterogeneous in its saturation levels [83]. However, measuring the difference between central venous to arterial CO_2 levels is very sensitive because CO_2 is highly diffusible [84,85]. CO_2 gaps more than 6 mmHg suggest inadequate blood flow for metabolic demand and can be used to guide resuscitation. Similarly, measuring the rate of tissue oxygen desaturation and recovery in response to a vascular occlusion test has been shown to identify underresuscitated trauma patients [86–88], predict survival from septic shock [89–91]. However, these and other measures of regional blood flow do not correlate well with macrocirculatory measures like changes in MAP and CO [92]. So at the present time, these measures and indices are best placed in the realm of clinical research.

CONCLUSION

The sole use of hemodynamic monitoring devices in the postoperative setting has not been linked to improved outcomes; however, appropriate interpretation of cardiovascular variables may help guide the best indicated interventions [93]. Perioperative goal-directed protocols, using the above-described monitors, have shown improved outcome in high-risk surgical patients by focusing on early and adequate DO_2 to the tissues [6]. Echocardiography is increasingly used as an early tool to identify a problem once the initial therapy does not result in restoration of cardiopulmonary function. Macrocirculatory targets are becoming clear and research is now focused on localized tissue perfusion, the balance between perfusion pressures at the levels of arterioles and venules within organs and tissues, microcirculatory dysfunction, endothelial disturbance, mitochondrial dysoxia, and capillary flow. Understanding the utility and the limitations of the various devices allows providers to optimally care for the high-risk surgical patient [94].

Acknowledgements

None.

Financial support and sponsorship

None.

Conflicts of interest

M.R.P. is consultant to Edwards Lifesciences, LiDCO, and Masimo. He has stock options with LiDCO and Cheetah Medical. A.M. has no conflicts of interest.

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