Perioperative hemodynamic monitoring

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Hemodynamic monitoring is the cornerstone of perioperative anesthetic monitoring. In the unconscious patient, hemodynamic monitoring not only provides information relating to cardiac output, volume status and ultimately tissue perfusion, but also indicates depth of anesthesia and adequacy of pain control. In the 21st century the anesthesiologist has an array of devices to choose from. No single device provides a complete assessment of hemodynamic status, and the use of all devices in every situation is neither practical nor appropriate. This article aims to provide the reader with an overview of the devices currently available, and the information they provide, to assist anesthesiologists in the selection of the most appropriate devices for any given situation.

Introduction

Hemodynamic monitoring describes the real time measurement of variables concerning the cardiovascular system. In the anesthetized patient, the goal of hemodynamic monitoring is to ensure adequate tissue perfusion and oxygen delivery, as well as predict instability and direct therapy when it occurs. Selection of rational, appropriate therapies requires accurate classification of developing instability; broadly distinguished as cardiac, distributive, hypovolemic or obstructive shock. Fundamentally, a provider must use hemodynamic variables to determine whether cardiac output (CO) is adequate, and if not, whether it will improve with fluid administration or if pharmacotherapy using vasoactive and inotropic agents is necessary. In addition, simple hemodynamic measures facilitate assessment of depth of anesthesia and adequacy of pain control.

Once therapy to restore circulation is initiated, hemodynamic monitoring measures therapeutic response. The combination of early detection, pre-emptive intervention and response assessment is...
frequently termed “functional or dynamic hemodynamic monitoring”. Coupling perioperative functional hemodynamic monitoring with protocols to optimize Oxygen Delivery reduces complications, postoperative length of stay and overall mortality; an effect that remains apparent 15 years after surgery. No single monitoring device provides a complete evaluation of hemodynamic status; as a result many devices are available each with their own benefits and limitations. Simple, non-invasive devices measure blood pressure, heart rate and cardiac output, but may be inaccurate in the setting of marked peripheral vasoconstriction. Minimally invasive (arterial catheterization) and more invasive (central venous and pulmonary artery catheterization) devices directly measure cardiac output, but may be time consuming to place and are more prone to complications. Between these extremes, there are an array of devices that indirectly determine cardiac output and assess pre-load responsiveness.

**Devices**

**Non-invasive blood pressure and heart rate measurement**

Routine, automated, non-invasive blood pressure measurement using the oscillometric technique is widely used in most anesthetic practice settings; replacing mercury sphygmomanometers, which were time consuming, prone to observer error, and carried environmental mercury toxicity risks. The oscillometric technique detects maximum pressure oscillations as the cuff deflates, the pressure at which this occurs corresponds to the mean arterial pressure. Algorithms are used to calculate systolic and diastolic blood pressure, and modern devices correlate well with old mercury devices. Oscillometric techniques are less effective in patients with arrhythmias and in the presence of external vibrations. Further, inaccuracies from improper cuff placement are magnified with this technique; cuffs that are too small, or tightly placed, over estimate pressure and large or loosely placed cuffs underestimate it. Chest electrodes, oscillometric methods or pulse oximetry can determine heart rate; frequently all three are used together.

Whilst relatively easy to obtain, the extent to which determination of blood pressure and heart rate reflects hemodynamic status is variable; although an exceptionally low pressure is almost always concerning, a normal pressure does not necessarily indicate hemodynamic stability and tachycardia is non-specific and may be blunted by medication. Development of hypotension represents a failure of the autonomic nervous system to compensate for reduced cardiac output and impaired oxygen delivery. The point at which hypotension develops varies with age, physical fitness, depth of anesthesia, cardiovascular side effects of anesthetic agent used, degree of pain control and co-morbid conditions. Under normal circumstances baroreceptors in the carotid body and aortic arch baroreceptors respond to reduced cardiac output by increasing sympathetic tone; causing an increase in heart rate and vascular tone to restore mean arterial pressure. Thus a patient may have imminent hemodynamic instability and an increasingly inadequate cardiac output prior to developing hypotension. Whilst blood pressure is a late marker of hemodynamic instability, in combination with heart rate it does provide a useful indication, albeit late, of hemodynamic status. In uncomplicated cases with minimal blood or fluid loss, heart rate and non-invasive blood pressure may be sufficient in isolation.

Non-invasive continuous measures of arterial pressure and calculation of CO based on the arterial pressure profile using finger plethysmographic analysis has been available for many years and are just now entering into the acute care environment. Only recently have clinical trials been performed in the operating room and suggest that these type of devices may be as accurate as invasive devices. Recent preliminary studies using a newer algorithm suggest even better performance and being completely non-invasive, potentially has advantages in selected patient populations.

**Fluid filled catheters**

Fluid filled central venous catheters, particularly those positioned in the pulmonary artery, represent the most invasive extreme of the monitoring spectrum but provide more hemodynamic information than any other single device (Table 1). Pulmonary artery catheters (PAC) are placed via a central vein, usually the right jugular or left subclavian vein, and advanced through the right heart chambers into a pulmonary artery. The catheter is carried in the direction of blood-flow by inflation of a small
balloon at the tip. Most complications result from attempts to puncture the vein and catheter passage through the heart (Table 2). Correct positioning in a pulmonary artery is confirmed when the waveform changes from that of a pulmonary artery to that of a pulmonary occlusion pressure (Ppao) (Fig. 1).

The use of pulmonary artery catheters (PAC) has reduced in recent years, largely because studies have demonstrated no benefit to placement. However, in these studies PAC use was not linked to an intervention and output variables were frequently misinterpreted. Clearly, no monitoring device will show benefit unless it directs appropriate interventions. Importantly, PAC placement did not increase mortality and it remains the only device that provides direct continuous CO monitoring, continuous mixed venous oxygen saturation (SvO2) and intra-thoracic vascular pressures (Table 1). An accurate CO measurement allows calculation of oxygen delivery (DO2) (Fig. 2). In the preoperative setting, optimizing the patient to ensure a DO2 of 600 ml/min/m²; by targeting a hemoglobin ≥ 11 g/dL, Ppao ≥ 12 mm Hg and then titrating inotrope therapy to achieve target DO2, improves surgical outcomes. However, it is only effective as a pre-optimization strategy; targeting supra-physiological DO2 once hemodynamic instability is established is not effective and may even be harmful.

Besides directing pre-optimization, PACs answer the most fundamental question of hemodynamic monitoring; is the cardiac output providing sufficient oxygen delivery to meet metabolic requirements? Inadequate oxygen delivery results in markedly increased oxygen extraction, manifested by a decrease in SvO2 (<70%). In the setting of reduced SvO2, interpretation of CO in the context of intrathoracic vascular pressures accurately classifies developing instability, directing appropriate therapy (Table 3). In this setting a high CO and low MAP indicates distributive shock, whereas a low cardiac output indicates the presence of one of the other three forms of shock; a low right atrial pressure (Pra) and Ppao indicates hypovolemic; a high Pra and Ppao indicates cardiogenic; an elevated mean pulmonary artery pressure (MPAP) and Pra > Ppao indicates obstructive (Table 3). It is important to point out that early distributive shock may present with an elevated SvO2 and increased CO.

When faced with overt or impending shock, determining whether fluid administration will improve the CO is key to successful management; i.e. is the patient preload responsive? Classification helps with this, but even patients with cardiogenic shock may be preload responsive and benefit from fluid administration in the absence of pulmonary edema. Ppao and Pra are often incorrectly used to predict preload responsiveness; low values indicating “the need for fluid”, and high values being used to indicate “adequate filling”. Yet, when studied, static measurements of Ppao and Pra have proven next to useless; failing to predict preload responsiveness whether low or high with no more reliability than the toss of a coin. However, Pra changes through the respiratory cycle are useful in spontaneously breathing patients. A reduction in Pra during inspiration accurately predicts fluid responsiveness, but is of limited value in ventilated surgical patients. Some authors argue that routine, isolated,

<table>
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<tr>
<th>Pulmonary catheter output</th>
<th>Normal range</th>
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<tr>
<td>Right atrial pressure</td>
<td>Pra</td>
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<tr>
<td>Pulmonary artery occlusion pressure</td>
<td>Ppao</td>
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<tr>
<td>Systolic pulmonary artery pressure</td>
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<tr>
<td>Diastolic pulmonary artery pressure</td>
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<tr>
<td>Mean pulmonary artery pressure</td>
<td>MPAP</td>
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<tr>
<td>Systemic vascular resistance</td>
<td>SVR</td>
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<tr>
<td>Pulmonary vascular resistance</td>
<td>PVR</td>
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<tr>
<td>Cardiac output</td>
<td>CO</td>
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### Table 2

Pulmonary artery catheter complications.

<table>
<thead>
<tr>
<th>Arterial puncture and bleeding</th>
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<tr>
<td>Pneumothorax</td>
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<tr>
<td>Non-sustained ventricular tachycardia</td>
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<tr>
<td>Device infection</td>
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<td>Valvular damage</td>
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monitoring of Pra in the operating room, or the intensive care unit for that matter, is a practice that should no longer be used.32

Therefore, although PACs can determine if the CO is adequate for metabolic needs and provide parameters that allow classification of developing instability, they do not effectively predict preload responsiveness. The most accurate method of predicting preload responsiveness in ventilated patients is stroke volume variation (SVV), defined as the ratio of maximal stroke volume difference over successive beats across several breaths and mean stroke volume over than same interval. Positive-pressure inspiration reduces venous return by phasically increasing intrathoracic pressure. This results in a transient decrease in right ventricular filling and right ventricular output, if the right ventricle is volume responsive. After a pulmonary transit time of 2–3 beats the decreased flow reaches the left ventricle decreasing left ventricular filling and, if the left ventricle is also volume responsive, decreased left ventricular stroke volume. Since arterial pulse pressure (diastolic to systolic arterial pressure) varies from beat-to-beat only by changes in stroke volume, arterial pulse pressure variation (PPV) co-varies with SVV.

The magnitude of SVV and PPV predicts the subsequent CO increase in response to a fixed intravascular volume challenge. We refer to the degree to which CO increases as preload responsiveness. An SVV >15% in a patient receiving a tidal volume >8 ml/kg predicts preload responsiveness,33–35 or >10% in patients receiving 6 ml/kg.36–38 PACs cannot quantify SVV, since CO is measured by thermodilution over a 15 s period. However two alternative methods, well studied in the operating room, are capable of providing this information; pulse contour analysis and esophageal Doppler.

Pulse contour analysis

Pulse contour analysis requires placement of an arterial catheter, usually in the radial or femoral artery. Four commercially available devices use pulse contour analysis to indirectly determine cardiac

Fig. 1. Changes in pulmonary artery catheter pressures, as measured from the distal port as the catheter is advanced to the correct position. Image courtesy of Edwards lifesciences (Irvine CA, USA).

Fig. 2. Oxygen delivery equation, DO₂ = oxygen delivery, CO = cardiac output L/min, Hb = hemoglobin concentration g/DL, SaO₂ = arterial oxygen saturation.
output, three of these use dilution methods for calibration. The premise of pulse contour analysis is that the pulse pressure depends upon the amount of blood ejected into the aorta, a fact first articulated in 1904.\textsuperscript{39} In essence, therefore, pulse pressure is related to stroke volume. However, the character of this relationship is complex, primarily because the magnitude of pulse pressure for a given stroke volume depends on the compliance of the aorta, which varies in a nonlinear fashion; becoming less compliant as pressure increases.

It was another 40 years before enough data was available to characterize the relationship between pressure and aortic compliance,\textsuperscript{40} but it is only recently that computer technology has permitted functional devices using complex algorithms to analyze the pulse pressure wave and derive a stroke volume. Such algorithms are required to account for reflection waves from the periphery, the magnitude of which are influenced by systemic vascular resistance.\textsuperscript{41} In addition, they need to adjust for, or be unaffected by, imperfect waves resulting from transducer system set-up and variations depending on whether a femoral or radial site is used.\textsuperscript{42} The PiCCO (Pulsion medical systems, Munich, Germany), LiDCO plus (LiDCO, Cambridge, UK), VolumeView/EV1000 (Edwards Lifesciences, Irvine CA, USA) and FloTrac (Edwards Lifesciences, Irvine CA, USA) utilize this technology.

**PiCCO**

PiCCO stands for pulse contour cardiac output, the “i” facilitates pronunciation. The device is calibrated using transpulmonary thermodilution, a 20 ml bolus of cold (\(<8\)°C) saline is given through a central vein and the thermal profile reported in a large artery. This method of calibration prevents the PiCCO from being used in the radial artery, and therefore PiCCO catheters must be placed in the femoral, axillary or brachial arteries. The PiCCO provides CO, Global end-diastolic volume (GEVD), SVV and PPV. Although some studies have demonstrated that GEDV may be superior to other static measures at predicting preload responsiveness,\textsuperscript{43,44} other studies have shown poor correlation, while demonstrating the effectiveness of SVV and PVV.\textsuperscript{45,46} To ensure accuracy of the PiCCO, it is important to calibrate every 8 h, or if the clinical status changes.

**LiDCOplus**

LiDCO stands for lithium dilution cardiac output, reflecting the use of lithium dilution to calibrate this device. Unlike transpulmonary thermodilution techniques, lithium dilution does not require a central line. Strictly speaking, LiDCOplus uses pulse power analysis (PulseCO) to provide continuous cardiac output data. Pulse power analysis converts the arterial waveform into a volume-time waveform and uses autocorrelation to determine stroke volume.\textsuperscript{47} Pulse power analysis is less influenced by reflection waves and transducer set-up variations since it is less dependent on the shape of the pulse wave. The LiDCO can be attached to a radial artery catheter, allowing monitoring from more conventional arterial access sites. Using lithium prevents calibration being affected by coadministration of “room temperature” or cold fluids. Like the PiCCO, this device provides CO, SVV and PPV and should be recalibrated after 8 h.\textsuperscript{48}

**VolumeView/EV1000**

The Volumeview is the latest calibrated pulse contour analysis device to enter the clinical arena. The VolumeView is an arterial catheter with a thermistor at the tip, it is placed in a femoral artery and the
pulse contour and thermodilution analysis occurs in the associated monitoring platform, the EV1000. Like the PiCCO it uses transpulmonary thermodilution for calibration, and provides the same output parameters. In validation testing it performs as well as the PICCO, and may calculate GEDV with greater precision.49

**FloTrac**

The FloTrac is sometimes called the Vigileo on account of the fact that the FloTrac probe also requires the Vigileo monitor. The FloTrac does not use a calibration technique to correct the CO and can be attached easily and quickly to any function arterial line. Instead, age, height, sex and weight are used to determine CO from the pulse contour analysis. Unsurprisingly, without calibration the CO is reported to be unreliable,50 and although third generation software demonstrates improved performance, it still underperforms when compared to PAC gold standards.51 In a recent review, Marik found that the FloTrac’s ability to track CO and its change was less accurate than PiCCO or LiDCO.52 However, SVV and PPV are not dependent on accurate calibration, and when used to direct fluid administration in the perioperative setting the FloTrac device reduces overall surgical complications.53,54

Overall, these three devices are very good at predicting preload responsiveness, and optimization protocols driven by SVV or PVV result in improved surgical outcomes.55,56 However, it must be noted that SVV and PPV require a closed chest to predict preload responsiveness, although deflation of a single lung in thoracic surgery does not impair the predictive capability of this technique.57 The presence of arrhythmias, specifically atrial fibrillation, introduce SVV and PPV as a result of different ventricular filling times rather than cyclic changes due to mechanical ventilation and in this setting fail to distinguish preload responsiveness. The PiCCO and LiDCO provide CO measurements that correlate well with those from a PAC.58,59

**Esophageal Doppler**

Esophageal Doppler is an alternative technique capable of providing SVV and CO. Doppler ultrasound is used to measure the velocity of blood in the descending aorta, which can be converted into a volume provided the diameter of the aorta is known. Some devices use normograms based on age, height and weight, whereas others use 2D ultrasound to measure the diameter. The ultrasound probe is advanced into the esophagus of the anesthetized patient until it sits at the mid thoracic level. Rotation of the probe to obtain a crisp waveform with each pulse will indicate optimum position and probe orientation. Variations in the waveform throughout the ventilation cycle can be used to determine SVV, which carries the same meaning as that derived from pulse contour analysis. This technique makes several assumptions; the angle of the ultrasound beam to the blood flow direction is the same as that of the transducer and probe, and a defined amount of blood has travels caudally and not into the descending aorta, usually set at 30%.60–63 Often, neither may be true, but SVV will still predict preload responsiveness.

Pre-optimization strategies using SVV derived from esophageal Doppler have resulted in improved surgical outcomes, in a variety of procedures.2,64 While this technique has been investigated more than pulse contour analysis, it is likely that optimization strategies driven by SVV/PVV from either source will be effective, provided the user is familiar enough with the device to ensure they are used properly.

**End tidal carbon dioxide**

An often forgotten adjunct to hemodynamic monitoring is measurement of end tidal carbon dioxide (ETCO2). While this is usually utilized in the operating room to ensure appropriate minute ventilation, a change in ETCO2 without a corresponding change in minute ventilation implies a change in carbon dioxide (CO2) delivery to the lungs, usually of vascular origin. An unexplained drop in ETCO2 may represent an increase in dead space as a result in PE.53 However, any cause of reduced CO will reduce CO2 delivery to the lung resulting in a lower ETCO2. Therefore a reduction in ETCO2 that is not explained by an increase in minute ventilation should prompt a complete evaluation of the hemodynamic status.

Conversely, an unexplained increase in ETCO2 implies enhanced CO2 delivery to the lungs. This could occur as a result of developing distributive shock in early sepsis, where metabolic rate and CO
both increase, although in a spontaneously breathing patient a reduction in ETCO2 is more likely to be seen as a result of respiratory compensation for the underlying metabolic acidosis of sepsis.

**Electrical impedance cardiography**

This technology is not currently used beyond the research setting, however it holds much promise for a non-invasive method of cardiac output and SVV monitoring. The basic principle is that the impedance (or resistance) to a current flowing through a conductor is related to the volume of the conductor. Changes in volume result in changes in impedance, and by using Ohm’s law we know that if a constant current is applied through a conductor, then changes in voltage will equal change in impedance. Hence by applying a constant current across the thorax we can determine the change in volume overtime, but since current chooses the path of least resistance, we can assume that the current mostly passes through the large vessels in the thorax. Therefore a change of impedance represents a change of volume in the thoracic aorta, which can be converted into a stroke volume by analysis of the impedance waveform, using a similar algorithmic method to pulse contour analysis. However, significant challenges exist to make the technology robust enough for the operating room. For example, a method of handling interference from diathermy devices is required, and a strategy for overcoming limitations in pulmonary edema, where the aorta ceases to be the path of least resistance through the thorax is needed.

**Bioreactance cardiography**

To overcome the limitations of bioimpedance systems, alternative approaches have been developed. Bioreactance uses the frequency phase shift of an oscillating electrical current instead of its amplitude detects pulsatile flow, rather than just the volume of fluid in the thorax, resulting in lower artifact and better signal to noise ratios. The NICOM system (Cheetah Medical, Portland, OR) is a commercially available device using bioreactance to measure CO. Several recent studies have documented that it is as accurate as PAC in monitoring CO, its change over time, and sudden changes in CO induced by a passive leg raising maneuver. It has recently been validated in the operating room too.

**Conclusions**

Several devices are available for the anesthesiologist. For simple, short cases without anticipated fluid losses a combination of non-invasive blood pressure monitoring, heart rate and ETCO2 will provide a vigilant practitioner sufficient means to diagnose unexpected instability. In high risk patients, or surgical cases invading a body cavity, use of SVV to guide preoptimization will suffice; using either the PiCCO, LiDCO, FloTrac or esophageal Doppler. In long complex surgery, where a mixed shock picture may develop, for example emergency abdominal surgery in a patient with pre-existing cardiomyopathy, valvulopathy or pulmonary hypertension, one should consider more complete hemodynamic monitoring using a calibrated CO device, or a PAC combined with a method to measure SVV.

**Practice points**

- Hypotension is a late marker of hemodynamic instability.
- Right atrial pressure and pulmonary artery occlusion pressure are no more effective than random chance at predicting preload responsiveness.
- Right atrial pressure, in isolation should not be routinely measured.
- Stroke volume variation and pulse pressure variation predict fluid responsiveness in patient receiving invasive positive pressure ventilation.
- Pulmonary artery catheters are helpful for classification of shock, but minimal invasive methods (pulse contour analysis and esophageal Doppler) are better at predicting preload responsiveness.
Future research directions

- Consensus statements and guidelines on pre-optimization strategies are urgently needed to provide a framework which would support perioperative evaluation of new hemodynamic monitoring devices.
- Comparative effectiveness research studies of new devices compared to PAC for achieving pre-optimization in the surgical patient.
- Standardized testing of new CO monitoring devices and SVV/PPV, a system much like the European society of hypertension international protocol for the validation of blood pressure monitors.
- Future studies of new hemodynamic monitoring devices should ensure the parameters are connected to a protocollled action, otherwise many new devices will suffer the same unnecessary fate of the PAC.

Conflict of interest

MEC has no competing interests to disclose. MRP is a consultant to Edwards LifeSciences and LiDCO Ltd.

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