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Contemporary perioperative haemodynamic monitoring

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Abstract

Haemodynamic monitoring is the cornerstone in the optimization of tissue perfusion and the prevention of deteriorating metabolism. Haemodynamic alterations could be summarized in terms of cardiac dysfunction, changes of loading conditions (preload or/and afterload), and patient related issues. This review aims to present the clinical applications of different haemodynamic monitoring techniques, discuss advantages and disadvantages, and provide guidance to help the clinician select those techniques suitable to optimize haemodynamics in individual patients during the perioperative period.

Key words: haemodynamics, monitoring, noninvasive.

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Haemodynamic monitoring is the cornerstone in the optimization of tissue perfusion and the prevention of deteriorating metabolism, both in the perioperative period and in the intensive care unit (ICU) setting. Haemodynamic alterations could be summarized in terms of cardiac dysfunction, changes of loading conditions (preload or/and afterload), and patient related issues (stress, sedation level, positioning, disease). Monitoring of haemodynamics serves to optimize pump performance, adequacy of circulation and tissue perfusion, and hence, interferes directly with patient outcome [1].

For several years, a trend towards less invasive haemodynamic monitoring has been observed, but so far none of these can be considered the gold standard for patients in the perioperative period. The pulmonary artery catheter (PAC) has been the clinical standard for many years. Initially, the PAC was used as a monitoring tool of filling pressures; however, the focus has been moved to assessment of cardiac output (CO) and mixed venous oximetry (SvO_2). With the availability of calibrated and non-calibrated monitoring systems, stroke volume monitoring has become the primordial measured variable, with pressures being derived indirectly. The primary goal of these monitors is to assess fluid responsiveness and to trend haemodynamic variables. Each monitoring technique has advantages and shortcomings, and these should be strictly acknowledged. The inter-monitor variability of absolute CO differs significantly also in comparison with thermodilution. The clinician must therefore be very attentive and critical when interpreting monitoring data and should mainly rely on trend analysis rather than absolute values.

This review aims to present the clinical applications of different modern haemodynamic monitoring techniques, discuss advantages and disadvantages, and provide guidance to help the clinician select the most suitable technique for optimizing haemodynamics in individual patients during the perioperative period.

BLOOD FLOW AND TISSUE PERFUSION ASSESSMENT

Organ perfusion is the result of the heart pumping an oxygen-carrying fluid (blood) through a transport system (vessels), ensuring organ function. Delivery of O_2 and nutrients and removal of CO_2 and toxins are the tasks of tissue perfusion. Systemic blood pressure is the inflow pressure for organ perfusion and as such an indirect measure of blood flow, though a poor indicator of blood flow [2]. Low compliant vessels could result in high blood pressures that do not correlate with adequate perfusion.

When assessing the function of contracting cardiac chambers and propagating flow in vessels, it is important to realize that pressure remains an incomplete descriptor of volume and flow, as could be observed in the following equation:

$\Delta Pressure \times Compliance = \Delta Volume$

If volume is to be directly related to pressure, compliance must remain constant. However, both cardiac and vessel compliances are dynamic in nature. Drugs, sympathetic tonus, or a change in vol-

ume status all alter cardiac and vessel compliance. Cardiac compliance is influenced directly by e.g. filling, myocardial ischaemia or heart failure [3]. In addition, loading conditions must be estimated to allow assessment of cardiac function because improving loading conditions improve cardiac function in load-dependent hearts. Thus, optimization of cardiac compliance and loading conditions can improve blood flow. To accomplish this, haemodynamic monitoring is essential to achieve optimal perfusion and balance loading conditions and cardiac systolic function in such a manner that tissue perfusion is optimized [4, 5].

Traditional clinical assessment of tissue perfusion includes monitoring urine output, skin temperature, and capillary refill time, but all of these are characterized as insensitive and responsive with delay. Lactate is the most important indicator of hypoxic metabolism and low perfusion state [6, 7] but is a non-continuous parameter. In addition, lactate levels can be influenced by lactate-buffered fluids [8]. Mixed venous oxygen saturation (SvO_2) is an important indicator of tissue oxygenation that is altered by changes in SaO_2, VO_2, haemoglobin (Hb) and cardiac output (CO). However, it only reflects the O_2 supply/demand balance of the entire body oxygenation: a normal SvO_2 does not exclude tissue hypoxia. For example, in patients with hyperdynamic septic shock and arterial-venous microcirculatory shunting, SvO_2 may be high even though tissue hypoxia is present [8-10]. PCO_2 gap is another early marker of hypoperfusion. It depends on the global CO_2 production, on cardiac output and on the complex interplay between CO_2 tension and CO_2 content. It is also influenced by the dissociative curve of CO_2 and tissue blood flow [11]. When SvO_2 is normal/high, the presence of elevated PCO_2 gap is indicative of the persisting impaired perfusion, which can help to distinguish hypoperfused patients with "normal" SvO_2 from those with adequate perfusion [12].

To discriminate the etiology of cardiovascular dysfunction, the different constituents of haemodynamics, contractility, preload and afterload must be considered separately without losing sight of the fact that they are part of a larger, integrated system. There is a strong relationship between the three physiological entities, as suggested in the Frank-Starling curve (Figure 1). All three constituents will be discussed separately although their intimate relationship should always be kept in mind.

ASSESSMENT AND OPTIMIZATION OF PRELOAD

Assessment of fluid responsiveness

The Frank-Starling curve depicts the impact of volume administration on stroke volume in well-performing hearts versus those with decreased systolic function (Figure 1): the latter are characterized by a flat curve, without any potential to generate more pressure with additional administered volume. As a clinical correlate, both hypovolaemia and hypervolaemia can increase morbidity or mortality, especially in critically ill patients. Hypovolaemia has been associated with tissue hypoperfusion and subsequent hypoxia, insufficient perfusion and subsequent acute kidney injury, whereas hypervolaemia has been associated with tissue oedema, organ failure, increased ICU or ventilator days because of pulmonary oedema, secondary infection, and increased mortality [13, 14] (Figure 2). Recently, numerous studies showed that only ± 50% of patients are fluid responsive [15–17], which suggests that the patient's preloading condition and fluid responsiveness should be determined before fluid resuscitation is started. For surgical patients, careful management of perioperative fluids with goal-direct therapy can greatly improve outcomes [18].

Basically, two options are possible to estimate preloading conditions: a fluid challenge and mechanically ventilation induced intra-thoracic pres-

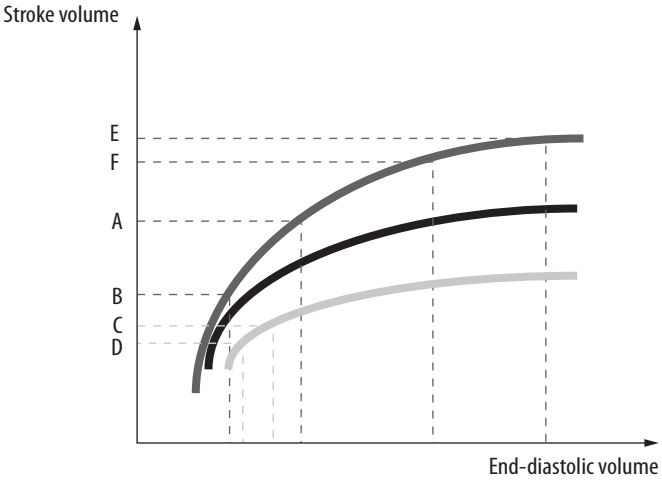


FIGURE 1. The relationship between contractility and preload (Frank-Starling curve)

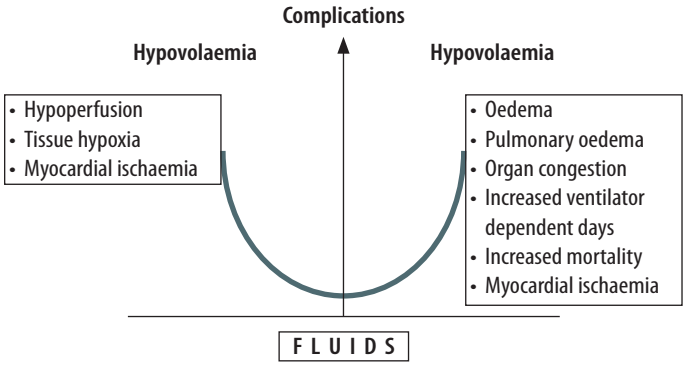


FIGURE 2. Relationship between complications and the optimal zone between hypovolaemia and hypervolaemia. Either too little or too much fluid will increase incidence of complications

sure alterations of blood inflow in the thoracic cage through both the superior and inferior caval vein.

Fluid challenge strategies

Fluid challenge strategies may include passive leg raising (PLR) [19], end-expiratory occlusion test (EEO) [20], a mini-bolus challenge [21], or a bolus infusion [22] in combination with measurement of SV or CO and related haemodynamic parameters with fast-response devices before and after the test, permitting instantaneous assessment of preload and thus prediction of fluid responsiveness. The PLR test is now supported by solid evidence, overcoming the risk of overfilling patients in cardiogenic or obstructive shock, though it appears cumbersome because of the specific positioning of the ICU patient [19, 23]. PLR is a reversible fluid challenge (bolus of 300 mL) and may therefore be the first choice to identify fluid responsiveness in a safe and reversible way. However, position changes limit its use in the operating theatre [23]. Another reversible fluid challenge strategy, the EEO test, prevents any variation in intrathoracic pressure, which leads to an increase in venous return, and can predict fluid responsiveness in patients with protective ventilation in the operating room or ICU [24, 25].

The cardiac chambers and surrounding vessels are in particular sensitive to compression when hypovolaemia is present, as is shown in the next examples. The superior caval vein and the right atrium are more prone to compression in hypovolaemia with increased intrathoracic pressures and PEEP. Cyclic insufflation during inspiration squeezes the pulmonary capillaries more in hypovolaemia, increasing right ventricular afterload. Finally, during expiration a fall in stroke volume is more pronounced when the ventricles are functioning on the steep part of the Frank-Starling curve (Figure 1). The latter is the physiological background of the delta down (see below). All these examples demonstrate that hypovolaemia should be corrected not only to improve circulation but also to optimize pumping function of the ventricles.

In a study by Covertino *et al.* [26], stroke volume (SV) appeared to be the more sensitive and fastest parameter to predict volume changes after fluid administration. In hypovolaemic patients, CO falls less than SV because heart rate interferes in an unpredictable manner because of age or drugs (such as β-blocker). CO monitoring is not sufficient to manage patients with complex haemodynamic disorders because absent knowledge of preloading and afterloading conditions. Besides SV, stroke volume variation (SVV) or arterial pulse pressure variation (PPV) induced by respiratory variation turned out to be a valid dynamic assessment of fluid responsiveness in mechanically ventilated patients [27, 28]. Both invasive arterial wave monitoring and non-invasive stroke volume monitoring permit monitoring of PPV or SVV. Nevertheless, several important issues should be recognized: 1) tidal volume should be at least 8 mL kg^{-1} ideal body weight [29], 2) though only applicable in passively mechanically ventilated patients, specific triggered ventilatory settings could still reveal PPV [30], 3) absent arrhythmia, 4) spontaneous breathing could hamper proper interpretation, 5) acute cor pulmonale or acute respiratory failure could reduce the impact of intrathoracic pressure changes, 6) open chest mechanical ventilation will greatly abolish intrathoracic pressure swings, 7) in children, as the ages varies, the cardiovascular physiology changes might thereafter affect the value of SVV [31].

With cardiac ultrasound, mechanical ventilation induced alterations of intrathoracic pressures induce both delta down and delta up of the transaortic flow velocity. In a graded haemorrhage and retransfusion study, Preisman *et al*. [32] showed delta down being correlated with a small left ventricular end-diastolic volume already from early blood loss, whereas delta up is related to inspiratory augmentation of left ventricular stroke volume.

Changes of the diameter of the superior (ΔSVC) and inferior caval vein diameter (ΔIVC) induced by respiratory variation (tidal volume > 7 mL kg^{-1}) are also predictors of fluid responsiveness with high specificity and sensitivity [33, 34]. Again, some caveats have to be recognized: right ventricular failure with pulmonary arterial hypertension will induce dilation of the afferent vessels, though hypovolaemia of the left ventricle could be present; significant tricuspid regurgitation following chest trauma will provoke dilated caval veins, while the left heart could be underfilled.

Nowadays, various dynamic parameters, derived from minimally invasive and non-invasive monitors, are used to evaluate and optimize a patient's preload condition and cardiac output, and some appear to be associated with reduced mortality, ICU length of stay and duration of mechanical ventilation [28, 35].

Invasive monitoring

Static variables such as central venous pressure (CVP) have been shown to be poor predictors of fluid responsiveness in critically ill patients because they can be affected by changes in venous return, cardiac compliance, vessel tone and intra-thoracic pressures [36]. CVP assessment should be combined with other haemodynamic monitoring and clinical assessment [37], in particular in patients with right ventricular failure and/or severe pulmonary hypertension. A dynamic approach of the CVP is possible with passive

leg raising, if the above-mentioned factors could be taken into account. The same rationale could be used in view of the value of pulmonary capillary wedge pressure (PCWP) to estimate left ventricular preload. However, numerous studies showed only a poor correlation of PCWP with filling status [38, 39].

For many years, invasive arterial pressure monitoring was combined with a PAC in haemodynamically unstable patients. A PAC can be used to obtain SV, CO, mixed venous oxygen saturation (SvO_2) and other pressure and flow variables to achieve a complete haemodynamic picture. Originally, in the 1980s the FICK principle was the gold standard. Pulmonary artery pressures (PAP) and PCWP measured with a PAC used to be the clinical standard to evaluate loading conditions and heart function. $ScVO_2$ became an interesting alternative in the management of patients with cardiac failure from what origin [40]. However, the clinical utility of PAC declined significantly from the end of the 90s because of the availability of alternative monitoring and the awareness of the potential of serious complications [41]. More recently, the PAC has been reserved for patients with hypoxia or acute pulmonary hypertension with overt right heart failure [42]. Moreover, recent reports show that use of the PAC does not improve outcome in critically ill patients [43, 44], a finding which is similar to other haemodynamic monitoring tools. Not the monitoring system itself, but the way it is integrated in haemodynamic monitoring strategies is an important feature in current practice.

With the development of minimally or non-invasive techniques, it is important to direct a guideline to use the specific monitoring in clinical practice.

Less invasive haemodynamic monitoring

For a few years, various monitors have been marketed and focus on step up systems offering the entire range from completely non-invasive over minimally invasive (with arterial pressure monitoring and derived variables) towards pulmonary artery catheter derived variables. The individual units can be utilized independently but can also be integrated into each other.

Calibrated systems

Thermodilution combined with arterial pulse contour analysis

PiCCO and VolumeView are monitoring tools combining arterial pulse contour analysis and transpulmonary thermodilution to derive a calculated CO, global end-diastolic volume (GEDV), cardiac function index (CFI), ejection fraction (EF), extravascular lung water (ELVW), pulmonary vascular permeability index (PVPI), SVV, PPV and $ScvO_2$ [45]. These devices provide information of global heart function, preload, afterload, as well as the balance between oxygen delivery and consumption. Compared to PAC, PiCCO and VolumeView are less invasive yet provide continuous, real-time calculation of SV, reliable and acceptable in haemodynamically unstable patients [46]. GEDV and CFI can help to determine volume responsiveness, while EVLW and PVPI can help in the diagnosis and differentiation of the causes of pulmonary oedema [45]. SVV and PPV derived by PiCCO have proven to be a reliable predictor of fluid responsiveness during surgery and in the ICU [28, 47, 48]. However, frequent recalibration is required. Some bias may exist in patients with cardiac arrhythmias, vascular abnormalities, aortic aneurysm or aortic valve stenosis.

Lithium dilution combined with pulse contour method

The LiDCOplus system combines lithium dilution and the arterial pulse contour method to generate continuous CO measurements. Close agreement with thermodilution derived CO has been reported in surgical patients with low output states [49, 50]. The LiDCOplus device always needs recalibration to optimize accuracy [51, 52] and does not provide advanced haemodynamic variables such as EVLW.

Non-calibrated systems

Arterial pressure waveform analysis

The Vigileo/FloTrac system provides real-time CO measurements by deriving SV from the arterial pressure waveform recorded from an arterial catheter without any calibration, a technique also called arterial pressure waveform analysis (APCO). On the basis of numerous data, age, gender and BSA, APCO provides information about SV, CI, SVV, PPV and systemic vascular resistance indexed for BSA (SVRI) [27, 53]. Goal-directed therapy based on Vigileo/FloTrac system could improve haemodynamics and reduce the duration of respiratory support after cardiac surgery. Hamed *et al.* showed that preload optimization with the Vigileo/FloTrac system after CABG increased cardiac performance compared with the PAC [54], including information on SV, PPV and SVV values, read on the Vigileo/FloTrac device. However, PPV values between 9% and 13% constitute a gray zone, which does not permit reliable prediction of fluid responsiveness [17]. This gray area constitutes up to 25% of all monitored anaesthetized patients. PPV interpretation is also limited by cardiac arrhythmias, vascular abnormalities, and severe aorta regurgitation [55]. With rapid haemodynamic changes, clinical assessment should be broadened and take into account other factors, such as changes of intra-thoracic pressures and vasoactive drugs administered. Furthermore, the

peripheral arterial pressure tracing is influenced by vascular tone, damping and reflection waves, much more than a central arterial pressure tracing. All these factors could induce deviations, though a comparison between a central aortic flow Doppler signal and a FloTrac obtained from a peripheral arterial catheter showed good correlation [56].

Transoesophageal cardiac ultrasound and Doppler (TOE)

TOE offers a complete range of haemodynamic variables, and goes much further than any other haemodynamic monitoring tool [3]. Haemodynamic parameters obtained by TOE can be used to assess and predict fluid responsiveness with parameters such as: 1) LVEDAI and right ventricular end-diastolic area indexed for body surface area (RVEDAI); 2) changes of diameter of the venous inlet into the thorax (including ΔIVC with transthoracic echo [vide infra] and ΔSVC with TOE); 3) SV derived from the area under the curve of a transaortic valvular Doppler signal (i.e. velocity-time integral (VTI), which reflects the distance one red blood cell is projected forward with each contraction; a constant aortic diameter is assumed); and 4) respiratory changes in peak velocity (ΔV_{peak}) and flow.

A left ventricular end-diastolic area indexed for body surface area (LVEDAI) < 5.5 cm^2 m^{-2} clearly indicates preload status is low [57], though the compliance of the left ventricle should be taken into account [58]. In the absence of severe tricuspid regurgitation, both ΔSVC and ΔIVC are good predictors of fluid responsiveness in mechanically ventilated patient. ΔSVC determined by means of TOE seems to be more accurate, but ΔIVC is more accessible with the transthoracic approach [33, 34, 59]. VTI variation > 20% predicts fluid responsiveness in mechanical ventilated patients, but cannot be used in patients with aortic valve disease [4, 60]. ΔV_{peak} > 12% predicts fluid responsiveness with a sensitivity of 100% and a specificity of 89% in mechanically ventilated patients in septic shock with preserved LV systolic function [61]. TOE allows monitoring changes of CO that are in high clinical agreement with those of the thermodilution method [62], but it cannot be used in awake patients or in those with oesophageal pathology. Continuous TOE monitoring became available recently with smaller probes and a dedicated machine in critically ill patients [63].

Non-invasive haemodynamic monitoring

Cardiac ultrasound and Doppler

Transthoracic echocardiography (TTE) allows intermittent assessment of haemodynamics at the bedside, and provides invaluable information about the anatomy and global function of the heart and the cardiac valves and about the status of the great vessels as well [64]. Furthermore, dynamic parameters obtained from echo-Doppler can be used to assess fluid responsiveness during the breathing circle or fluid challenges [65]. In critically ill, mechanically ventilated patients, TTE has been shown to be an accurate and precise method to estimate SV and CO [66]. The left ventricular outflow tract (LVOT) VTI SV estimation correlates with volume responsiveness [67, 68]. Both VTI variation > 20% and IVC variation > 12–18%, as well as VTI increases > 12% after PLR suggested fluid responsiveness to be present in mechanically ventilated patients [65]. Respiratory variation of the maximal Doppler velocity in LVOT ($V_{max}Ao$) was also used to assess fluid responsiveness, and has high sensitivity [50]. SVI obtained from carotid Doppler increasing > 12% after PLR is another method that can predict fluid responsiveness, with a sensitivity and specificity of 94% and 86%, respectively [69].

EVLW can be captured easily with lung ultrasound: the presence of B lines, comet-like signals moving to-and-fro with mechanical ventilation or spontaneous breathing, is a typical diagnostic tool to elucidate pulmonary oedema in a non-invasive manner.

Perioperative haemodynamic monitoring by TTE-Doppler is an inevitable and indispensable tool [70], but it does not allow continuous real-time monitoring. Furthermore, each change in the patient's condition requires repeated examination from different views, to eliminate ultrasound pitfalls and is strongly operator dependent.

Volume clamp and radial artery applanation tonometry

Devices using either the volume clamp method (such as Nexfin, CNAP, ClearSight) or radial artery applanation tonometry (such as T-Line) can provide continuous information of blood pressure, SV and PPV from analyzing the continuous arterial pressure waveform. These are uncalibrated, non-invasive, real-time techniques, which are very easy to perform. Many clinical studies have shown they reliably monitor perfusion pressure and CO in the perioperative period [71–73]. A moderate to normal relationship between CO estimations was obtained by this type of device in comparison with PiCCO or thermodilution [74–77]. As for fluid responsiveness prediction, PPV and SVV analysis from a Nexfin monitor have proven to generate reliable data in patients during mechanical ventilation in non-thoracic surgery or after CABG [78]. Stens *et al.* showed there is some age bias between SVV and PPV: SVV appears more sensitive in younger patients (age < 55 years), while PPV appears more sensitive in older patients (age > 55 years) [79]. Because haemodynamic

parameters are derived from a finger or radial artery pulse pressure waveform, significant peripheral vessel resistance changes (such as with oedema or during vasoconstriction using vasoactive drugs), or hand or finger movement [75–77] may limit its clinical use. Also, in patients in shock (with cold extremities) or in an extreme Trendelenburg position (with compression of the subclavian artery between the clavicle and the first rib) signals of these devices could be too poor to allow clinical use. With these limitations, some studies showed that the estimation of CO by Nexfin technology in critically ill patients was not reliable for tracking the effects of a fluid challenge, with a high level of disagreement compared with echocardiography or PICCO monitor [80, 81].

Another tool utilizes flow variation exemplified by plethysmography. The SaO_2 curve provides a non-invasive tool to monitor intra-thoracic pressure induced changes of stroke volume. This feature is used in the plethysmography variability index (PVI). From the SaO_2 curve, a continuous pulsatile to non-pulsatile flow ratio calculated during surgery [82] integrated in a goal-directed fluid management may reduce lactate levels and facilitates perioperative fluid management [83].

Partial CO_2 rebreathing

Partial CO_2 rebreathing is a non-invasive technique that estimates CO by using respiratory gas analysis and pulse oximetry. Many studies suggest moderate to good agreement with CO measurements obtained by the thermodilution method during surgery and in ICU mechanically ventilated patients [84–87], but there was a great bias during a period of rapidly changing shunt and dead space, as occurs at the start of one-lung ventilation [88]. Although many studies have shown that CO was underestimated by the partial CO_2 rebreathing method compared with the thermodilution method, it still can be used to guide haemodynamic management during surgery [87, 89]. In patients with acute lung injury, CO derived from the partial CO_2 rebreathing method was shown to be unreliable [90]. Moreover, it cannot be used with spontaneously breathing patients.

Pulse wave transit time (PWTT)

The estimated continuous cardiac output (esCCO) system is a novel non-invasive CO measurement based on the relationship between PWTT and SV. PWTT is calculated from the R wave interval of the ECG and peripheral (SpO_2) pulse wave arrival when ECG and SpO_2 are simultaneously recorded [91]. A large sample size multicenter study demonstrated that esCCO had a close correlation with thermodilution method [92]. However, most of the results showed low accuracy and sensitivity with a large bias between esCCO and with TTE or thermodilution derived CO during cardiac or non-cardiac surgery, and it failed to assess the preload and SVR [93, 94]. There are potential inaccuracies when vasopressors are used to treat hypotension associated with decreased SVR, and SVV measurement with esCCO may require further improvement [95, 96].

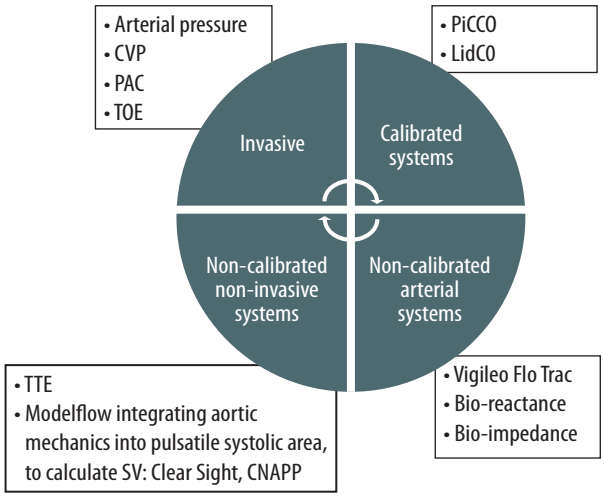
Bio-impedance and bio-reactance

Another non-invasive real-time CO monitor is called bio-impedance cardiography, which is based on the measurements of the impedance (or resistance) a small electrical current encounters when it travels through the body or chest area. Bio-impedance changes when fluid levels in the thorax change, e.g. as the left ventricle contracts and blood flows into the thoracic aorta. Bio-impedance measures the changes in amplitude of the voltage change across the thorax [97]. Both positive and negative studies have been published, with some suggesting bio-impedance to be a useful non-invasive method to assess extracellular volume changes in patients undergoing cardiac surgery, with a close relationship with thermodilution [98, 99], while others reported a negative correlation between the accuracy of bio-impedance and increased fluid accumulation within the thorax [100–102]. Bio-impedance systems are sensitive to intra-thoracic fluids, such as in patients with oedema or pleural fluid, often present in critically ill patients.

A newer method, named bio-reactance, accurately measures the phase shift of an oscillating current that occurs when a current traverses the thoracic cavity – the higher the cardiac stroke volume (and thus the blood flow), the more significant these shifts become. In contrast to the bio-impedance method, studies showed a good relationship between CO determined by bio-reactance and minimally invasive methods or thermodilution method [103–105]. Bio-reactance can be an accurate method to assess fluid responsiveness in critically ill patients and in patients undergoing major abdominal-pelvic surgery [97, 103–105]. Nevertheless, measurement of CO and other parameters can be disturbed by changes in tissue oedema, pleural effusions, arrhythmias, electrical interference, pacemakers or motion [105, 106]. Furthermore, the present software does not allow assessment of rapid fluid shifts because of time lags of 30 s. Future software updates should permit nearly beat-to-beat monitoring.

ASSESSMENT OF AFTERLOAD

Afterload could be described through a two-element Windkessel model, taking together the main pulsatile component of arterial load total (arterial



CVP – central venous pressure, PAC – pulmonary artery catheter, TOE – transoesophageal echo-Doppler, TTE – transthoracic cardiac ultrasound

FIGURE 3. Overview of the different monitoring techniques, including invasive, minimally invasive and non-invasive modalities

compliance or capacitance C) with C = SV/pulse pressure and a steady part, systemic vascular resistance (SVR = 80 × (MAP – CVP)/CO). Both are pressure and flow dependent. The effective arterial elastance (Ea) is a nowadays often forgotten measure, the ratio of end-systolic pressure (ESP) to SV. It lumps together the pulsatile and steady components of afterload and observes the arterial system as a single elastic volume. Ea therefore is dependent on SVR, heart rate and the elastic properties of the arterial system. ESP can be easily calculated as 0.9 × systolic blood pressure [107].

The dynamic Ea (Eadyn) is PPV/SVV and was thought to be a variable that could estimate the filling capacity of the circulation and therefore safely predict the response of the arterial pressure to preload optimization in preload-dependent patients with acute circulatory failure [108], but some conflicting results exist [109].

Minimally invasive monitors such as the Vigileo/FloTrac system, and oesophageal Doppler combined with minimally invasive arterial pressure monitor can be used to estimate PPV/SVV in mechanically ventilated patients [108, 110], while noninvasive monitoring (model flow based devices – Figure 3) can also be used in patients with spontaneous breathing [111]. Important is the fact that SVR should be normal to achieve correct data of SVV. If afterload changes, it is unknown to what extent mechanical ventilation could interfere with SVV [112].

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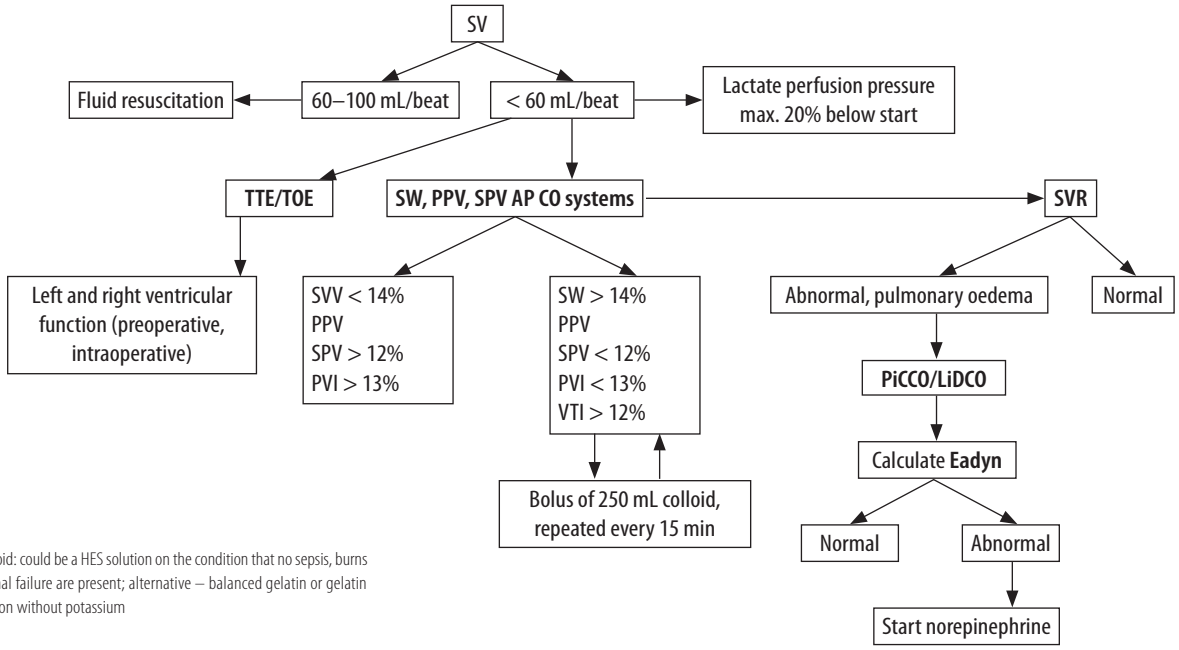
Afterload variables, such as left ventricular meridional wall stress (LV-ESWS), global longitudinal strain (GLS), SVR, total arterial compliance, aortic root size and descending aortic pressure/flow velocity, can be estimated with cardiac ultrasound [115–119]. Combined use of pulsatile changes of the aortic contour with TOE and invasively measured local aortic pressure at the same level approaches the arctangent model of Langewouters [116].



*Colloid: could be a HES solution on the condition that no sepsis, burns or renal failure are present; alternative – balanced gelatin or gelatin solution without potassium

FIGURE 4. Algorithm to summarize the initial approach of haemodynamic monitoring. Stroke volume and derivatives on their own are not enough; afterload and systolic cardiac function should also be taken into account

ASSESSMENT OF CARDIAC CONTRACTILITY

In patients who are "fluid responsive", fluid resuscitation can increase CO and optimize heart performance. In patients with cardiac failure, fluid resuscitation will aggravate heart function, but increasing ventricular contractility will improve it (at a constant afterload). Therefore, cardiac contractility should be assessed to identify the cause of shock. Echocardiography is able to rapidly identify the cause of hypotension, and it remains the mainstay diagnostic tool to assess cardiac pathology and haemodynamics. Both TOE and TTE can provide qualitative and quantitative information about cardiac systolic and diastolic function, structure and function of valves, absence of myocardial ischaemia, and intra-cardiac structures [3, 110].

Traditional variables such as SV, left ventricular ejection fraction (LVEF) and CO have some limitations if used to assess cardiac contractility, because they are affected by loading conditions [120]. Maximal LV power and preload-adjusted maximal ventricular power (PWR_{max}/D_2) have been shown to better reflect the LV contractile state. PWR_{max}/D_2 is more accurate and independent of preloading conditions, but less reliable in patients with severe hypovolaemia or severe hypertension [115, 121]. Nowadays, this parameter has been abandoned because of afterload interference.

Cardiac power index (CPI) could be obtained non-invasively via bio-reactance monitoring with the following formula:

$CPI = MAP \times CI/451$

with *CI* – cardiac index, *MAP* – mean arterial blood pressure. Cardiac power was found to be the strongest haemodynamic correlate with mortality after cardiogenic shock [122]. Further studies should determine the value of this parameter in critically ill monitoring.

Nowadays, researchers are trying to develop more sensitive, accurate and non-invasive methods to assess cardiac contractility. The slope of the regional stretch-strain relationship measured from tissue Doppler imaging (TDI) can be used as a non-invasive index of the myocardial inotropic state, but cannot be used in patients with atrial arrhythmias or high heart rates with fusion of mitral inflow E and A waves [120]. Recently, non-invasive speckle tracking echocardiography (STE) allowed combined regional and global myocardial function assessment [123, 124]. Global peak longitudinal strain (GLS), determined from STE, is a sensitive and feasible method, which overcomes many of the limitations of LVEF. It can be used to elucidate left ventricular dysfunction in septic shock patients in a discontinuous manner, with improved prediction of prognosis after myocardial infarction in comparison with LVEF [125]. However, optimal imaging is necessary, and GLS can be affected by heart rate variability, breathing translation, and choice of region of interest in the myocardium [120, 124].

CLINICAL PERSPECTIVE

Which monitoring tool should be utilized in specific situations? Actually, Figure 4 shows clearly there is a gradation in invasiveness of haemodynamic monitoring, which should be respected when considering optimal choice of monitoring in an ASA II patient versus a critically ill patient with multiple comorbidities. The choice of haemodynamic monitoring depends on the availability, acquaintance and experience of the team with the type of monitoring, the pathology presented and the degree of urgency. Furthermore, the time to implement one of the different monitoring tools also plays an important role in the choice. Hence, transoesophageal echocardiography and Doppler has a very short time to organize and implement, whereas a PAC necessitates a well-organized support by a nurse.

Still, a PAC, including SvO_2 monitoring, is nowadays still the clinical standard, particular in patients with right ventricular failure and pulmonary hypertension. Whereas these patients benefit from pulmonary artery pressure monitoring, achieved by a PAC, right ventricular systolic function follow-up could be more easily performed by means of cardiac ultrasound, in particular transoesophageal echocardiography. The latter allows valvular function, most importantly of the tricuspid and pulmonary valve, to monitor pulmonary artery pressures from regurgitant flow velocities.

Also, patients with severe left ventricular dysfunction need close follow-up, avoiding fluid overload and keeping these patients relatively dry. Intraoperative monitoring could be done combining transoesophageal echocardiography (short axis view of the left ventricle in conjunction with mitral valve regurgitation and transaortic valve flow assessment) with an arterial monitoring system, allowing monitoring of stroke volume, SVV and cardiac power, if available. The latter monitoring system could be continued in the postoperative phase.

Even less invasive haemodynamic monitoring could be utilized in patients in whom no real cardiac burden is present and absence of aggressive surgery, but fluid and insensible losses could be considerable following long-term and extensive procedures (e.g. extensive plastic surgery with DIEP flap and breast reconstruction, open laparotomy).

In all cases, anaesthesiologists should aim for early goal-directed haemodynamic monitoring, including low lactate levels and perfusion pressures max. 20% below normal life perfusion pressure,

aiming at preservation of homeostasis and body temperature.

CONCLUSIONS

There is an abundance of new minimally invasive or non-invasive, continuous and real-time monitors of cardiac function, perfusion and fluid responsiveness. Even though many studies have been and are being devoted to assessing the value of these monitors and their trending parameters, it is still far too early to confirm the effects these devices might have on outcome. It is important to master all levels of haemodynamic monitoring techniques – from completely non-invasive to minimally invasive and up to invasive monitoring – and acknowledge the limitations and shortcomings of all monitoring tools. Information from a haemodynamic monitor should be confirmed by data from another monitoring system to guarantee precise diagnosis (Figure 3), and thus correct management and optimization of the patient's loading conditions and heart contractility.

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