Review

Clinical review: Hemodynamic monitoring in the intensive care unit

Joachim Boldt

Director, Department of Anesthesiology and Intensive Care Medicine, Klinikum der Stadt Ludwigshafen, Ludwigshafen, Germany

Correspondence: Joachim Boldt, BoldtJ@gmx.net

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Abstract

Since the beginning of modern anesthesia, in 1846, the anesthetist has relied on his natural senses to monitor the patient, aided more recently by simple technical devices such as the stethoscope. There has been a tremendous increase in the availability of monitoring devices in the past 30 years. Modern technology has provided a large number of sophisticated monitors and therapeutic instruments, particularly in the past decade. Most of these techniques have enhanced our understanding of the mechanism of the patients' decompensation and have helped to guide appropriate therapeutic interventions. As surgery and critical care medicine have developed rapidly, patient monitoring capability has become increasingly complex. The most important aspect in monitoring the critically ill patient is the detection of life-threatening derangements of vital functions. Aggressive marketing strategies have been promoted to monitor almost every aspect of the patient's status. However, these strategies are only telling us what is possible; they do not tell us whether they enhance patient safety, improve our therapy, or even improve patient outcome.

Keywords costs, critical illness, monitoring, organ function, outcome

> "Not everything that counts can be counted and not everything that can be counted counts" (Albert Einstein)

Improvements in surgical techniques and perioperative anesthetic management have led to enhancement of surgery and intensive care therapy for patients who would never before have been acceptable candidates. Ongoing developments in monitoring techniques have shed new light on our knowledge of pathophysiological processes associated with critical illness.

Since the first public demonstration of modern anesthesia in Boston in 1846 there has been a tremendous increase in monitoring devices, especially in the past 30 years. Modern technology has provided a large number of sophisticated monitors. Most of these newly developed techniques have enhanced our understanding of the mechanism of patient decompensation and have helped to guide appropriate therapeutic interventions. Aggressive marketing strategies have been promoted to monitor a variety of functions. However, it still remains unclear whether they are able to enhance patient safety or even improve patient outcome.

What is so specific in monitoring the intensive care unit patient?

Multiple organ dysfunction syndrome accounts for most deaths in the intensive care unit (ICU). Patients who develop this complication place an enormous burden on all hospital services, especially intensive care. Although the exact pathophysiology of multiple organ dysfunction syndrome is not yet definitely known, alterations in systemic hemodynamics, organ perfusion and tissue microcirculation resulting in tissue hypoxia appear to play a key role in the onset and maintenance of this syndrome. Pflueger stated in 1872 that "arterial oxygen content, arterial pressures, velocity of blood stream,

CO = cardiac output; CVP = central venous pressure; DO_2 = oxygen delivery; d*p*/d*t* max = maximum rate of rise of ventricular pressure; EF = ejection fraction; EVLW = extravascular lung water; Hb = hemoglobin concentration; ICU = intensive care unit; NIRS = near-infrared spectroscopy; PAC = pulmonary artery catheter; PAOP = pulmonary artery occlusion pressure; $ptiO_2$ = oxygen partial pressure in tissues; RF = residual fraction; RVEDV = right ventricular end-diastolic volume; RVEF = right ventricular ejection fraction; SaO_2 = arterial oxygen saturation; SV = stroke volume; SvO_2 = venous oxygen saturation; TEE = transesophageal echocardiography; TTE = transthoracal echocardiography; VO_2 = oxygen consumption.

mode of cardiac work, mode of respiration are all incidental and subordinate; they all combine to service the cell" [1].

Optimal monitoring of the critically ill ICU patient remains a challenge. Controversy continues as to whether the patient will profit from a more aggressive monitoring. Unfortunately, our current understanding of this area is poor. Time appears to be crucial for an early diagnosis of hemodynamic catastrophe and earlier therapy appears to improve outcome in this situation.

Possibilities for systemic hemodynamic monitoring

Monitoring of cardiac function

The assessment of ventricular function is based on the measurement of both volumes and pressures. By relating changes in left ventricular volume or pressure to time during phases of the cardiac cycle, indices of contractility can be generated. These indices include fractional shortening, mean circumferential shortening, the maximum rate of rise of ventricular pressure (d*p*/d*t* max), and ejection fraction (EF).

The difficulty of evaluating cardiac performance is reflected by the number of hemodynamic variables, which are thought to be indicators of myocardial function [2]. Some variables can be measured only in experimental settings and not in clinical practice. Left ventricular d*p*/d*t* max has been widely accepted as an index of the contractile performance of the left ventricle. Despite theoretical objections, it is accepted that measurement of left ventricular d*p*/d*t* max is a satisfactory index of ventricular contractility.

The most widely accepted technique for measuring cardiac output (CO) is the thermodilution method using a pulmonary artery catheter (PAC) and a bedside microprocessor. This technique is easy to perform without any risk of indicator accumulation, and can thus be carried out sequentially and multiple times even in the critically ill.

Singer promotes CO measurement non-invasively by the transthoracic electrical impedance technique or by transesophageal Doppler sonography [3]. However, results are very controversial [4–7]: some investigators state that the transthoracic electrical impedance method compares well with the CO estimates by thermodilution; others, however, find this technique unreliable when compared with other methods, particularly in those patients with altered lung function.

PAC monitoring

The introduction of flow-directed PAC has not only revolutionized monitoring in intensive care medicine, but has also contributed to improving our knowledge of cardiovascular function. It is a relatively simple and safe procedure, which produces information relevant to certain aspects of cardiopulmonary function in a variety of circumstances [8]. The PAC monitoring instrument enables us to obtain direct information on pressure variables such as pulmonary artery pressure, pulmonary capillary wedge pressure (better named pulmonary artery occlusion pressure [PAOP]) and right atrial pressure, as well as flow variables such as (continuous) CO. Common formulae allow us to calculate further determinants of the cardiovascular system (e.g. systemic vascular resistance, pulmonary vascular resistance, right ventricular stroke work, and left ventricular stroke work).

Since the introduction of the PAC by Swan in the 1970s, more than 2 million PACs have been inserted. The value, limitations, and indications of the PAC are still controversially discussed. Some authors have demonstrated that a PAC can be helpful in the early diagnosis of subendocardial ischemia [9], and others have shown that even the experienced intensivist failed to diagnose significant hemodynamic alteration in the absence of a PAC [10]. The value of elevated PAOP or abnormal wave patterns in the wedge tracing as indicators of ischemia remains controversial. Gore *et al*. [11] have shown that patients who suffered from acute myocardial infarction and who were monitored with a PAC had a worse outcome than patients treated without a PAC. Other workers have concluded that, in patients who are at high risk of suffering myocardial ischemia, there was no significant difference regarding outcome or postoperative complications whether they were managed with a central venous pressure (CVP) catheter or a PAC [12].

Measurement of PAOP does not always reflect end-diastolic volume. The CVP and PAOP parallel each other with a high degree of correlation in the perioperative period in patients with EF $>50\%$ [13]. In patients with severely impaired myocardial function (EF $<40\%$), no more correlation between the CVP and PAOP could be demonstrated, most probably due to changes in myocardial compliance caused by myocardial hypertrophy or a stiff left ventricle secondary to ischemia or cardiac surgery procedures.

The list of potential complications when using a PAC for monitoring the critically ill is large. Infections are one of the most important risks, particularly in enhanced periods of PAC monitoring. Sise *et al*. [14] found an increase in infection and an increase in the incidence of catheter fault with approximately 20% of catheters after 6–7 days. However, complications are few in skilled, experienced hands and can be readily anticipated.

Further developments of PAC-based hemodynamic monitoring

Mixed venous oxygen saturation

PACs do not only help to measure pressure and flow variables, but there have been a lot of developments in the PAC equipment that provide further information on the patients' state. Continuous monitoring of venous oxygen saturation (SvO_2) by fiberoptic reflectometry is another adjunct to hemodynamic monitoring of the critically ill patient. The advantage of this technique is the ability to realize immediate indications

of both trends and abrupt changes in the oxygen-supply-to-demand ratio at the bedside [15]. SvO_2 has been promoted as an indicator of changes in CO. Normal values for SvO_2 range from 70 to 75%. A linear correlation has been demonstrated between these CO and SvO_2. This correlation, however, is not actually linear but curvilinear, and only if oxygen consumption (VO_2) and arterial oxygen content do not change is this relationship true. SvO_2 only reflects the overall oxygen reserve of the whole body. A normal SvO_2 value, for instance, does not rule out an impaired oxygen supply to individual organs [16].

Monitoring of the right ventricle

The focus of interest in hemodynamic monitoring has been on the 'dominant' left side of the heart. The tendency to 'overlook' the right ventricle as an important part of the circulatory system occurred because it was traditionally regarded as a passive conduit, responsible for accepting venous blood and pumping it through the pulmonary circulation to the left ventricle [17]. Maintenance of normal circulatory homeostasis depends on an adequate function of both ventricles. Changes in dimension and performance of one ventricle influence the geometry of the other.

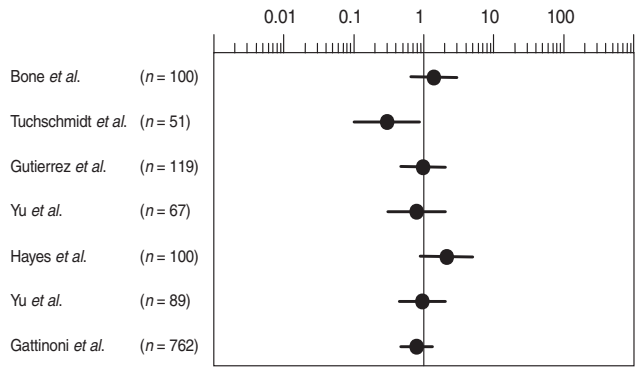
There is growing interest in the importance of the neglected right side of the heart, particularly in patients suffering from sepsis and acute respiratory dysfunction syndrome, and in heart-transplanted patients [18]. Commonly monitored parameters such as CVP, right atrial pressure or right ventricular pressure have been demonstrated to be invalid for judging right ventricular function or right ventricular loading conditions [19]. Hoffman *et al*. [20] demonstrated no correlation between CVP and right ventricular end-diastolic volume (RVEDV), and they emphasized that the preload factor in the original Frank–Starling hypothesis had nothing to do with pressure but concerned volume. Moreover, the use of RVEDV and right ventricular ejection fraction (RVEF) is unaffected by arbitrary and poorly reproducible zero points for pressure transducers. Right ventricular performance is difficult to measure by other conventional monitoring techniques because of the functional anatomy and complex geometry of the right ventricle [21]. Measurement of RVEF by thermodilution is an easy technique to perform with no accumulation of toxic indicators based on the use of a fast response thermistor that allows an accurate detection of a rapid step change of the staircase curve of the downstream temperature change. The principle of this method is based on the thermodilution technique. The catheter is equipped with a fast response thermistor and electrodes for intracardiac ECG recording. The typical downslope thermodilution washout curve follows an exponential decay, interrupted by the diastolic plateaux. The ratio between the temperature change of two successive diastolic plateaux represents the fraction of blood remaining in the right ventricle (the residual fraction [RF]). The RVEF is calculated from the equation EF = 1 – RF. The thermistor of the new catheter is able to measure beat-to-beat temperature variations of the downstream temperature changes after injection of 10 ml ice-cold dextrose. As the CO and stroke volume (SV) are also calculated by the microprocessor, RVEDV can be derived from SV/RVEF and the right ventricular end-systolic volume from RVEDV – SV.

The accuracy and validity of this technique have been shown using radionuclear methods in humans as well as in animal experiments, and it has been proved to be valid and accurate for measuring right ventricular volumes (and RVEF) in comparison with radiographic, radionuclide, and echocardiographic methods [22]. Further development of computer techniques allows continuous monitoring of right ventricular hemodynamics. Monitoring of right ventricular data may contribute to the evaluation of the patients' prognosis [18]. However, no large clinical trials are available showing a beneficial impact of RVEF monitoring on patient outcome.

To Swan or not to Swan?

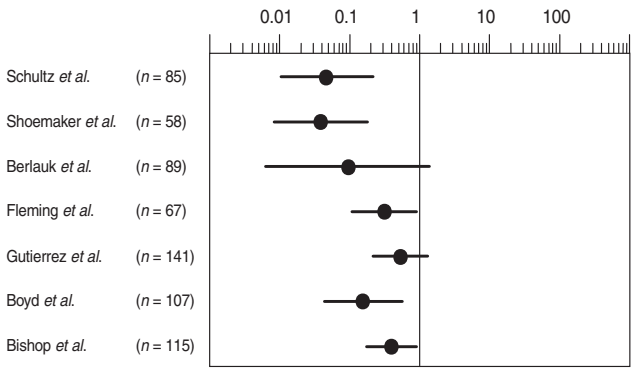
Hemodynamic monitoring using the invasive pulmonary artery balloon-tip thermodilation catheter has been the gold standard for evaluation of circulatory function for several years. The impact of the PAC on patient outcome has been questioned in a variety of studies. The question "to catheterize or not to catheterize" is not new [23,24]. The controversy culminated with the Connors *et al*. study published in 1996 [25], in which a higher mortality rate was reported in patients in whom a PAC was inserted than in those patients treated without a PAC. Unfortunately, this study was retrospective not randomized, and the controls were case-matched using a controversial propensity score. The Connors *et al*. study [25] has influenced several reviews on the worth of pulmonary artery catheterization and calls for a moratorium on the use of PACs. In a Medline-based meta-analysis reviewing 12 randomized, controlled trials including 1610 patients, a significant reduction in morbidity was shown when a PAC-guided therapeutic strategy was used [26]. Although PAC-based hemodynamic management has been shown to be successful in improving outcome in planned major surgery [27], this benefit has not been translated to the ICU patient. In a study in surgical patients admitted to the ICU after developing organ failure, Gattinoni *et al*. [28] failed to show improved outcome with therapies aimed at maintaining either oxygen delivery or SvO_2 at supranormal values. In a meta-analysis, Heyland *et al*. [29] demonstrated that a therapy targeted at supraphysiologic endpoints (oxygen delivery, VO_2) was not associated with decreased mortality. Boyd and Bennett [30] demonstrated with the help of a meta-analysis that outcome was not improved when therapy attempting to improve tissue perfusion was started in patients in whom sepsis and organ failure had already occurred (Fig. 1). When PAC-guided management was carried out earlier, however, a significant outcome improvement was seen (Fig. 2). Finally, in a consensus conference on the PAC [31], it was found that there is insufficient evidence to fully determine whether PAC-guided therapy significantly alters outcome; more prospective, ran-

Figure 1



Odds ratio and 95% confidence interval for studies (total of 1031 patients) attempting to improve tissue perfusion after onset of tissue hypoxic can be expected. No beneficial effect on mortality was seen. (Modified from [30].)

Figure 2



Odds ratio and 95% confidence interval for studies (total of 662 patients) attempting to improve tissue perfusion before onset of tissue hypoxic can be expected. Beneficial effect on mortality were seen. (Modified from [30].)

domized clinical trials are therefore recommended. Although the PAC-based monitoring has its limitations and pitfalls, it has proven to still be an established technique for monitoring the hemodynamics of the ICU patient.

Measurement of extravascular lung water and intrathoracic blood volume

The most common method for evaluating lung water content is based on the double-indicator dilution technique using indocyanine green as the non-diffusible indicator prepared in ice-cold dextrose (a diffusible indicator) [32]. Absolute values of extravascular lung water (EVLW) appear to be less important than intra-individual changes. Other techniques for assessing pulmonary fluids are either invalid or too bulky and expensive (e.g. computerized tomography and nuclear magnetic resonance imaging). One of the fundamental lesions in inflammation is an altered capillary integrity resulting in an increase in (pulmonary) endothelial permeability. Depressed left ventricular performance increases hydrostatic pressure in the pulmonary circulation, synergistically influencing fluid flux across a damaged pulmonary microvascular membrane. EVLW can be rapidly and safely measured at the bedside, although femoral arterial cannulation is believed to be dangerous in patients receiving high doses of vasopressors [33]. The double-dye technique needs indocyanine green for the measurement process, which adds considerable costs.

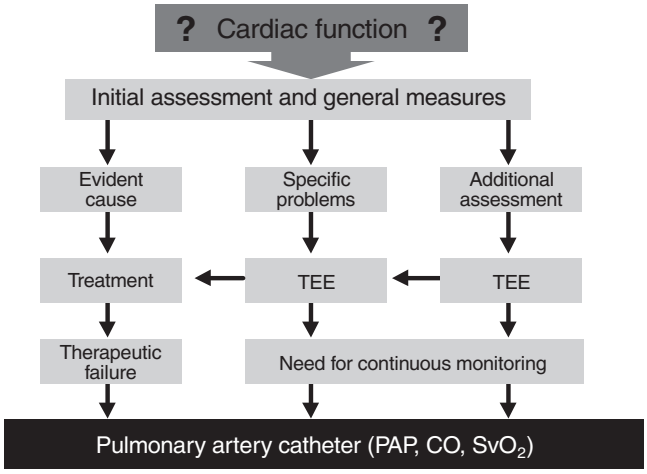
Blood volume is mostly indirectly inferred from measurement of arterial pressure, heart rate, CVP, and PAOP. However, these variables appear to be unreliable indicators of hypovolemia. Correct determination of cardiac filling pressures by PAOP may be difficult under various circumstances (e.g. in ventilated patients with large excursions in intrathoracic pressure) [34], and intrathoracic blood volume appears to be a more reliable indicator of preload than the cardiac filling pressures are [35]. Using EVLW and intrathoracic blood volume monitoring, a reduction in ICU stay and hospital stay was shown [36,37], and even mortality was demonstrated to be reduced [38].

Echocardiography

Our monitoring armamentarium has been enhanced significantly by the introduction of imaging techniques [39]. Esophageal Doppler has been reported to be an alternative to the PAC [40]. Assessing global and regional left ventricular function is the domain of echocardiography (either transthoracal echocardiography [TTE] or transesophageal echocardiography [TEE]). Two-dimensional echocardiography provides important information about cardiac function and structure including left ventricular cavity size, fractional shortening and regional wall motion abnormalities [41]. Information on the presence and extent of ischemic heart disease is possible by monitoring segmental wall motion abnormalities. These abnormalities, however, are only indirect markers of myocardial perfusion that can persist for prolonged periods in the absence of infarction. TEE seems to provide more accurate information on ventricular size than standard monitoring instruments. End diastolic volume was a better predictor of myocardial performance than PAOP. Two-dimensional colored echocardiography allows quantification of shunts, CO and non-invasive assessment of concomitant valvular disease. Data obtained by echocardiography may have significant impact on a patient's treatment [42]. Echocardiography is the first diagnostic method used on suspicion of aortic dissection, endocarditis, and pulmonary embolism with hemodynamic instability. Hypovolemia, left ventricular failure, global systolic function and size of both ventricles can rapidly be diagnosed using TTE/TEE. Finally, valvular abnormalities and functionally important heart disease can be readily determined [43].

TTE/TEE is unlikely to replace already established monitoring technologies in the ICU. Echocardiography provides different information to the PAC. TTE/TEE has the disadvantage that no continuous monitoring of cardiac function is possible. This monitoring technique requires a high standard of training and a lot of experience, and the costs are tremendous in comparison with other monitoring devices. This monitoring instrument thus cannot be considered as a standard 'screening' device, and it is too early to postulate that it should be used *in lieu* of the PAC. Both monitoring methods, TTE/TEE and PAC, are therefore more complementary than competitive, and both techniques can be recommended for monitoring of hemodynamics in the critically ill ICU patient (Fig. 3).

Figure 3



Use of the pulmonary artery catheter and transesophageal echocardiography (TEE) in the intensive care unit patient. PAP, pulmonary artery pressure; CO, cardiac output; SvO_2, mixed venous oxygen saturation. (Modified from [43].)

Monitoring of organ perfusion and microcirculation

Inadequate tissue perfusion and oxygenation are likely to contribute to the development of organ failures and increased mortality in critically ill patients [44]. For this reason, assessment of the adequacy of oxygen supply to organs and tissues is essential. Monitoring of tissue oxygenation and organ function in the clinical setting is largely based on measuring traditional variables of resuscitation, such as global hemodynamics, pulse oximetry, capillary refill, urine output, or indirect biochemical markers. These parameters remain insensitive indicators of dysoxia and are considered to be poor surrogates for the oxygen availability at tissue levels, since tissue oxygenation is determined by the net balance between cellular oxygen supply and oxygen demand. Furthermore, the fact that continuing regional tissue dysoxia can persist despite the presence of an apparently adequate systemic blood flow, pressure, and arterial oxygen content highlights the need for specific indices of oxygenation at tissue level.

Methods to detect tissue dysoxia and oxygen debt can grossly be subdivided into two groups; namely, techniques directed at the assessment of oxygenation at the systemic level, and monitoring techniques for measurements at the organ level [45] (Table 1).

Indirect methods to measure tissue perfusion/oxygenation

Oxygen delivery and oxygen consumption

Total body perfusion and oxygenation relies on adequate arterial oxygen saturation (SaO_2), hemoglobin concentration (Hb) and CO. The total amount of convective oxygen delivered to the peripheral tissue per minute (DO_2) can be calculated as $DO_2 = CO \times CaO_2$, with $CaO_2 = (Hb \times 1.39 \times SaO_2)$. Under steady state conditions, uptake of oxygen from the arterial blood (VO_2) represents the sum of all oxidative metabolic reactions in the body. VO_2 can be measured directly by the use of metabolic carts and the analysis from expired gas, or calculated from CO and arterial and mixed venous blood samples. The ratio VO_2/DO_2 is the oxygen extraction ratio. VO_2/DO_2 dependency occurs when the increase in oxygen extraction can no longer fully compensate for the fall in DO_2. The relationship between DO_2 and VO_2 can therefore be used to assess the adequacy of tissue oxygenation. However, determination of DO_2 and VO_2 requires right heart catheterization to measure CO or is expensive if metabolic carts are used to measure VO_2. Moreover, the interpretation of DO_2/VO_2 relationships has been criticized because of mathematical coupling of data [46].

Mixed venous oxygen saturation

SvO_2 can be readily measured from blood gas analysis derived at the bedside either intermittently or continuously with a fiberoptic PAC. Since the pulmonary artery carries blood from all vascular beds of the organism, mixed venous blood may represent the amount of oxygen in systemic circulation that is left after passage through the tissues. SvO_2 might thus serve as a parameter of global oxygenation. The determinants of SvO_2 are SaO_2, systemic VO_2, CO, and Hb, with $SvO_2 = (SaO_2 - VO_2) / (1.39 \times Hb \times CO)$. Accordingly, an increase in VO_2 and a decrease in Hb, CO and arterial oxygenation will result in a decrease of SvO_2. Interpretation of SvO_2 values might be difficult in conditions where DO_2/VO_2 relationships are altered. For example, arterial–venous microcirculatory shunting in sepsis may increase SvO_2, thus pretending adequate tissue oxygenation, while regional tissue dysoxia is present [47].

Blood lactate

The importance of monitoring arterial lactate levels in critically ill patients has been advocated [48]. Lactate is formed from pyruvate by the cytosolic enzyme lactate dehydrogenase. Lactate concentrations >2 mmol/l are generally considered a biochemical indicator of inadequate oxygenation [49]. Circulatory failure with impaired tissue perfusion is the most common

Table 1

Measuring tissue oxygenation

Monitor	Method	Variables	Global/regional	Invasive/non-invasive
Systemic oxygenation	Pulmonary artery catheter	$VO_2/DO_2/ERO_2$	Global	Invasive
Mixed venous O_2 saturation	Pulmonary artery catheter–blood gas analyses	SVO_2	Global	Invasive
Lactate	Laboratory–enzymatic testing	Lactate	Global	Invasive
Gastrointestinal tonometry	Measurement of pCO_2 in an air-filled or saline-filled balloon	$prCO_2/pCO_2$ gap, pHi	Regional	Minimally invasive
Near-infrared spectroscopy	Absorbance analysis of near-infrared light	Hb/O_2Hb, cytochrome aa_3	Regional	Non-invasive
Oxygen electrodes	Polarographic probes	pO_2	Regional	Minimally invasive

DO_2, oxygen delivery; ERO_2, oxygen extraction ratio; Hb/O_2Hb, deoxygenated/oxygenated hemoglobin; pCO_2 gap, arterial-to-intramucosal partial pressure of carbon dioxide difference; pHi, gastric intramucosal pH; pO_2, partial pressure of oxygen; $prCO_2$, regional gastric carbon dioxide tension; SvO_2, mixed venous oxygen saturation; VO_2, oxygen consumption.

cause of lactic acidosis in intensive care patients. A number of mechanisms other than impaired tissue oxygenation may cause an increase in blood lactate, including an activation in glycolysis, reduced pyruvate dehydrogenase activity, or liver failure. Understanding the complex process of tissue lactate production and utilization is therefore important to understand the usefulness and potential limitations of monitoring blood lactate levels. The presence of elevated lactate levels should nevertheless prompt the clinician to initiate diagnostic procedures for assessment of the circulatory status.

Tonometry

The hypovolemic patient is at risk of experiencing splanchnic hypoperfusion with subsequent development of bacterial translocation and systemic inflammatory response syndrome [50]. Abnormalities of splanchnic perfusion may coexist with normal systemic hemodynamic and metabolic parameters [51]. Measurement of gastric intramucosal pH has emerged as an attractive option for diagnosis and monitoring of splanchnic hypoperfusion, and it appears to have more relevance for predicting postoperative complications [52]. The introduction of gastric or sigmoid mucosal tonometry for the measurement of intraluminal carbon dioxide has enabled the clinician to change focus from global oxygen transport to regional tissue oxygenation. Tonometry in the present context refers to the measurement of partial pressure of a gas. Measuring the regional gastric carbon dioxide tension photometrically with infrared spectrometry via a special gastric tube and calculating the arterial-to-intramucosal partial pressure of carbon dioxide difference and gastric intramucosal pH provide valuable information about splanchnic perfusion [52]. Tonometer measurements might provide an insight in a region of the body that is among the first to develop an inadequacy of tissue oxygenation in circulatory shock and is the last to be restored by resuscitation [53]. Gastrointestinal tonometry has been evaluated in various situations during surgery and intensive care [54]. As a result, it has been shown that prolonged acidosis in the gastric mucosa might be a sensitive, but not specific, predictor of outcome in critically ill patients [55]. Whether measurement of gastric mucosal pH or the mucosal/venous carbon dioxide gradient reflects total splanchnic perfusion is in doubt [56]. The prognostic value of tonometry in the critically ill is not definitely clear. Only few studies have demonstrated benefit from applying tonometry [57]. Nevertheless, other measurements of hepatosplanchnic perfusion (e.g. indocyanine green extraction, mucosal laser Doppler flowmetry, remission spectrophotometry, or liver vein oxygen saturation) are not widely accepted methods and are still mostly used for research reasons.

Near-infrared spectroscopy

Near-infrared spectroscopy (NIRS) is a continuous non-invasive method applying the principles of light transmission and absorption to determine tissue oxygen saturation. NIRS measures oxygenated and deoxygenated Hb as well as the redox state of cytochrome aa3 as an average value of arterial, venous and capillary blood according to the law of Lambert–Beer. Cytochrome aa3, the terminal cytochrome of the respiratory chain, is responsible for approximately 90% of cellular oxygen consumption through oxidative phosphorylation [58]. Since the redox state of cytochrome aa3 is primarily determined by available oxygen, a decrease in cellular oxygen delivery results in a reduction of oxidative phosphorylation and a decreased oxidation level of cytochrome aa3. Monitoring the redox state of cytochrome aa3 might therefore be a key indicator of an impaired cellular oxidative metabolism and tissue dysoxia. Although NIRS may be applied to almost any organ, it has mainly been used in studies investigating cerebral or muscle oxygenation after different types of hypoxic injuries [59]. The main limitation of NIRS in the clinical setting is the inability to make quantitative measurements because of the contamination of light by scatter and absorption [60].

Tissue oxygen tension

Monitoring tissue oxygen tension has become feasible for clinical use by the development of miniaturized implantable

Clark electrodes. The polarographic oxygen sensors enable us to measure oxygen partial pressure in tissues ($ptiO_2$), organs, and body fluids directly and continuously. The $ptiO_2$ values correspond to oxygen availability on a cellular level and provide information about oxygen supply and utilization in specific tissue beds. Tissue oxygen tension has been measured successfully in intensive care as well as during neurosurgical procedures [61,62]. Studies on the critical threshold of $ptiO_2$ after traumatic brain injury showed that the absolute level of oxygenation in the cerebral white matter was a reliable predictor of neurological outcome [61]. Organs like the brain are not readily accessible, however, and thus are not suitable for clinical routine monitoring. Monitoring muscle partial pressure of oxygen might provide an early and reliable indicator of stagnant blood flow and tissue dysoxia. It is easily accessible and reacts to hemorrhage, resuscitation, and shock on a similar time scale to that of the gastrointestinal tract. Limiting factors in the use of polarographic oxygen probes are the dependence of electrode currents on tissue temperature, errors in $ptiO_2$ readings due to tissue trauma and edema by electrode insertion, or intravascular misplacement of the oxygen sensors.

Conclusions

There are many monitoring instruments available and there have been some outstanding developments in the past. One major question rises whenever a new system is floating the market: What is the 'gold-standard' for our monitoring instrument? There is no doubt that the use of hemodynamic monitoring devices may yield additional information, and there is no doubt that some of this information may be useful. However, many of these devices may confuse and mislead the responsible physician who has to care about his patient. It is not always obvious that the more sophisticated the monitoring, the 'better' it is for the patients. The dictum is still 'first, do no harm'.

One of the burning problems in managing the ICU patient is thus whether a specific monitoring technique has an impact on patient outcome. Interestingly, it has never been shown that a specific hemodynamic monitoring technique improves outcome: neither ECG, oximetry or other well established monitoring techniques have been proven in large trials to have an impact on patient outcome. There is the risk that all these techniques may delay or prolong what could otherwise have been a quick, simple, and safe procedure, and they may render the procedure much more expensive. The malfunctioning monitor and the wrongly trained intensivist may be a great risk for the patient! Nevertheless, in the critically ill ICU patient there is a need for something more than the senses to monitor the patient's hemodynamics, although some opponents of invasive hemodynamic monitoring wish to turn back the clock to the old days of a 'finger on the pulse'. The properly trained intensivist is a *conditio sine qua non* for managing the critically ill patient because it is widely accepted that 'a fool with a tool is still a fool'.

Competing interests

None declared.

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