Review

Clinical relevance of data from the pulmonary artery catheter

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Abstract

The usefulness of parameters measured using the pulmonary artery catheter has been challenged because no benefit in patient outcome has been observed in clinical trials. However, technological advances have been made, including continuous measurement of cardiac output (CO), mixed venous saturation (SvO_2), and right ventricle end-diastolic volume (CEDV) have been made. Pulmonary artery occlusion pressure (PAOP), CEDV and right atrial pressure (RAP) are not good predictors of fluid load responsiveness except when very low. Despite this methodological limitation, variation of these parameters during fluid loading remains a good indicator of fluid challenge tolerance. Accuracy of continuous thermodilution and SvO_2 measurement has been demonstrated in vitro and at bedside. A decrease in SvO_2 is a global index of an inadequate oxygen delivery (DO_2)/oxygen requirement relationship. In this setting, a therapeutic decision to improve determinants of SvO_2 should be considered with the help of all other PAC parameters. Technological improvement transforms PAC in a real time integrated physiological device and allows one to observe the impact of therapeutic intervention. What we need now is a clinical trial with a PAC-guided treatment algorithm taking into account the above integrated PAC parameters.

Introduction

More than 35 years ago, Harold James Swan (who died on 7 February 2005, just after writing his last contribution, published in *Anesthesiology* [1]), along with William Ganz and collaborators, reported an article entitled 'Catheterization of the heart in man with the use of a flow-directed balloon-tipped catheter' [2]. The first catheter was intended exclusively to measure right heart and pulmonary artery (either occluded or not) pressures. Following an idea by Ganz, a thermistor was incorporated into the catheter and a reliable method to measure cardiac output (CO) by thermodilution was developed. The first report on the pulmonary artery catheter (PAC) was centred on myocardial ischaemia, but application of the PAC was quickly extended to other pathologies and settings including cardiac failure, septic shock, acute respiratory distress syndrome (ARDS), cardiac surgery and high-risk surgery.

The general design of the PAC did not change for more than three decades; however, technological progress has recently been achieved. The incorporation of a rapid response thermistor permitted continuous measurements of CO, right ventricular ejection fraction (RVEF) and right ventricular end-diastolic volume (RVEDV). Also, the addition of an fibreoptic canal permitted continuous spectrophotometric measurement of mixed venous oxygen saturation (SvO_2).

Are the classic and new parameters reliable at the bedside? When using PAC, how may we better understand the various pathologies and how may we devise a better therapeutic strategy in critical care and anaesthesiology? These are some of the questions we address in the present review.

Parameters from the pulmonary artery catheter

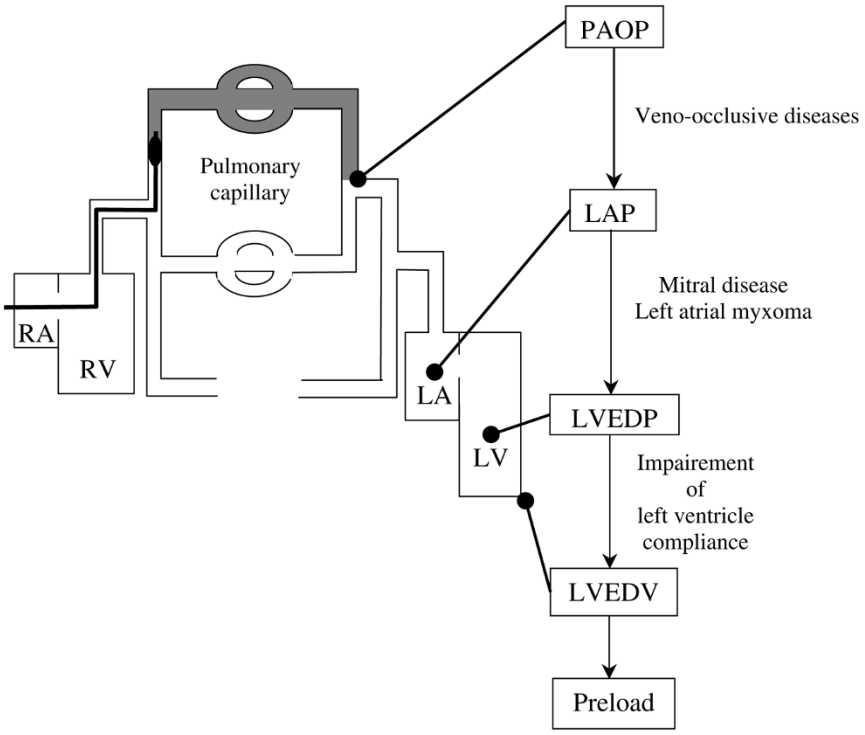
Pressure in the pulmonary circulation

Pulmonary artery occlusion pressure

The pulmonary artery occlusion pressure (PAOP) is obtained following inflation of the balloon at the tip of the PAC. In theory, after inflation of the balloon there is a continuous column of blood from the pulmonary artery to the left ventricle during diastole. The end of the diastole can be identified by the 'a' wave of the PAOP curve, which coincides with the 'p' wave on the electrocardiogram. Consequently, PAOP is considered an approximation of the left ventricular end-diastolic pressure (LVEDP) [3]. For a given left ventricular compliance, LEVDP is proportional to the left ventricular end-diastolic volume (LVEDV). As described by the Frank–Starling relationship, the force of ventricular contraction is proportional to the length of the myocardial fibres, as determined by LVEDV. Therefore, PAOP can be considered an indicator of preload.

ARDS = acute respiratory distress syndrome; CEDV = continuous RVEDV; CCO = continuous CO; CO = cardiac output; DO_2 = oxygen delivery; LVEDP = left ventricular end-diastolic pressure; LVEDV = left ventricular end-diastolic volume; PAC = pulmonary artery catheter; PAOP = pulmonary artery occlusion pressure; PAP = pulmonary artery pressure; Pcp = pulmonary capillary pressure; RVEDV = right ventricular end-diastolic volume; RVEF = right ventricular ejection fraction; SaO_2 = arterial oxygen saturation; $ScvO_2$ = central venous oxygen saturation; SvO_2 = mixed venous oxygen saturation; VO_2 = oxygen consumption.

Figure 1



Schematic representation of PAOP. The grey area represents the area without flow. Examples are given of pathologies in which PAOP is not equivalent to left ventricular preload. LA, left auricle; LAP, left atrial pressure; LV, left ventricle; LVEDP, left ventricular end-diastolic pressure; LVEDV, left ventricular end-diastolic volume; PAOP, pulmonary artery occlusion pressure; RA, right auricle; RV, right ventricle.

Knowledge of preload is fundamental to clinical practice in intensive care or during surgery. For example, when other haemodynamic parameters (e.g. Svo_2, serum lactate concentration, or renal function) suggest to the clinician that tissue perfusion must be improved via increased oxygen delivery (Do_2), preload determination allows the clinician to choose between fluid loading and inotropic drugs. In other pathologies, such as left ventricular failure or ARDS, preload must be controlled to avoid worsening of pulmonary oedema.

However, the assumption that pulmonary artery occlusion always induces a continuous blood column may not be valid in some cases (Figure 1). First, when the catheter tip is in West zone 1 or 2, the increase in alveolar pressure interrupts the blood column. Consequently, PAOP is higher than end-diastolic pulmonary pressure and pulmonary venous pressure. To address this problem, the catheter tip must be in West zone 3. If this is the case, then the following relationship will be present during the respiratory cycle in mechanically ventilated patients [4]: end-diastolic pulmonary pressure > PAOP, and $\Delta PAOP/\Delta PAP > 1.5$ (where PAP is the pulmonary artery pressure). Second, in mitral valve disease PAOP reflects the increase in left atrial pressure and not the LVEDP. In mitral stenosis the PAOP trace has a large 'a' wave and in mitral regurgitation a large 'v' wave. Finally, all changes in ventricular compliance (the LVEDP/LVEDV relationship) induce overestimation of preload by PAOP. Modification to left ventricular compliance may result from numerous pathologies, including myocardial ischaemia and failure, myocardial hypertrophy or dilatation, septic chock, aortic disease and pericardial disease.

Despite these practical limitations, PAOP provides some useful information. When dynamic preload dependency indicators (see below in this paragraph) are unreliable (i.e. in the presence of arrhythmia or spontaneous ventilation, among other conditions), it is always possible to establish a Frank–Starling relationship between PAOP and stroke volume variation (or CO) following successive fluid challenges. The stroke volume does not increase any further after additional fluid challenge when the flat part of the Frank–Starling curve is reached, indicating preload independency. In pulmonary arterial hypertension a difference (>7–8 mmHg) between diastolic PAP and PAOP indicates an increase in pulmonary artery (or capillary) resistance, and primary pulmonary hypertension is diagnosed. In contrast, pulmonary arterial hypertension without any gradient is secondary to increased pulmonary venous resistance, and causes of altered left ventricular compliance (e.g. myocardial ischaemia or left ventricular failure) or mitral disease must be explored.

Preload dependency

Right atrial pressure and central venous pressure have largely been used as static preload indicators. During the past decade, however, several dynamic indicators for preload dependency such as pulse pressure variation or Δdown have been studied. In most of these studies, these parameters were compared with the classic static parameters such as PAOP or central venous pressure [5]. Compared with dynamic indicators, static parameters have poor ability to predict responsiveness to fluid challenge, except when they are very low (less than 5 mmHg) [6,7]. Static parameters cannot therefore be recommended as indicators of preload dependency, and PAOP cannot be used as a first-line tool to make fluid loading decisions if dynamic parameters are available.

Dynamic parameters also have limitations in settings such as cardiac arrhythmia, spontaneous ventilation, high-dose vasopressors and right ventricular failure. Dynamic indices are unable to predict volume of fluid challenge and tolerance to a subsequent fluid challenge when the patient's volume status is on the upper part of the slope of the Frank–Starling relationship. In this setting the PAOP remains a good indicator of fluid challenge tolerance; a large increase in PAOP (5–10 mmHg) after one fluid challenge indicates that further fluid loading should be considered with caution. Fluid responsiveness is a better basis for decisions regarding fluid loading; however, it is not equivalent to fluid loading tolerance. Therefore, static and dynamic preload parameters provide complementary information.

Pulmonary capillary pressure

As described above, increased gradient between PAOP and diastolic PAP indicates increased pulmonary resistance or increased pulmonary blood flow, or both. In these settings, pulmonary capillary pressure (Pcp) may exceed PAOP [8]. Therefore, an increased gradient between diastolic PAP and PAOP is considered a valuable indicator of increased Pcp. The resistance between pulmonary artery and left atrium can be simply modelled as one artery resistance and one venous resistance in series, with a capacitance located in the capillary bed [9,10]. Because of this series resistance with a capillary capacitance, the Pcp can be measured from the pressure decay profile after occlusion of the balloon (Figure 2). After occlusion of the pulmonary artery, the downstream blood is discharged into the capillary across arterial resistance and then into the pulmonary veins across venous resistance [8]. The initial rapid drop in pressure reflects the Pcp as the downstream blood is trapped in the capillary bed and equilibrates with the Pcp. The following slower drop in pressure is determined by the discharge of blood across the pulmonary venous resistance and tends toward the PAOP (Figure 2).

The more sophisticated approach to determining the Pcp includes the average smoothing of the pressure signal and mathematical curve fitting of the signal. With an approach that is more realistic at the bedside, the Pcp can be measured using a graphical method; the Pcp is estimated as the point at which the pressure curve deviates from the slope of the first rapid decay (Figure 2).

Because Pcp is the main determinant of efflux between capillary lumen and alveolar space, whether the integrity of alveolar–capillary barrier is impaired or not, its measurement may be of interest in pathologies such as ARDS to guide fluid loading [8]. A Pcp threshold value must be determined above which pulmonary oedema develops, and pulmonary compliance and gas exchange are impaired. Fluid loading should be then limited to this threshold value as much as possible, taking into consideration the perfusion of other organs. Further studies are evidently necessary to explore the utility of such a strategy and to develop new tools for automatic measurement of Pcp.

Continuous measurement of end-diastolic volume and ejection fraction

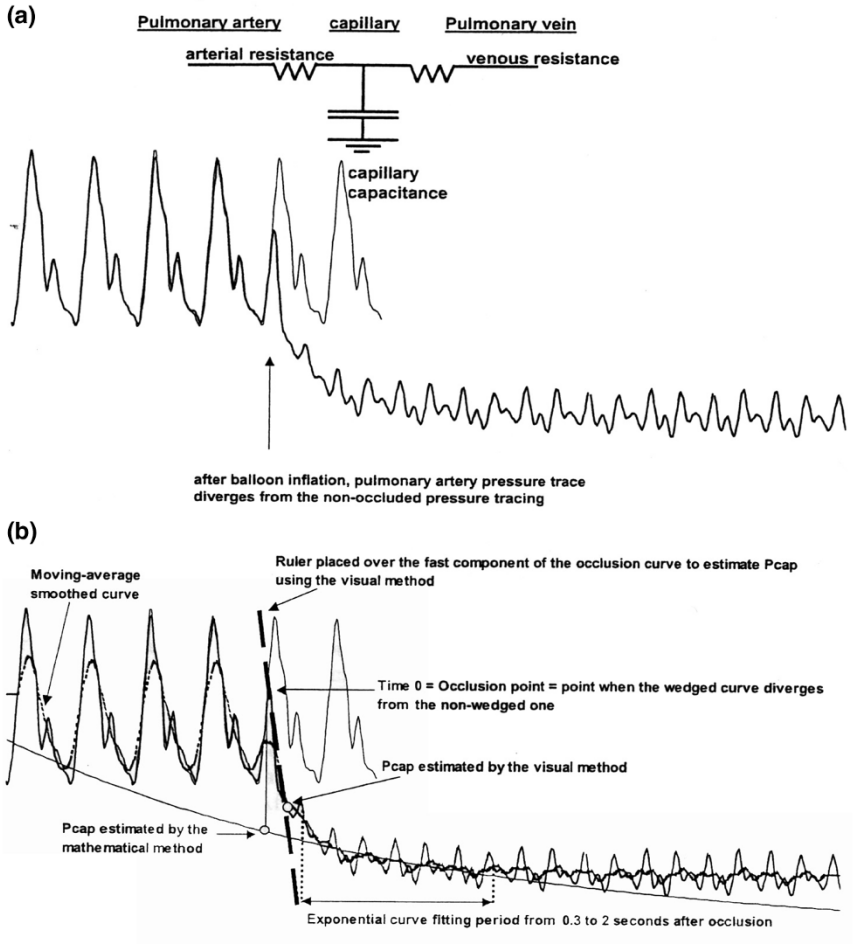
In the 1980s, technological improvements led to the introduction of the 'volumetric' pulmonary catheter. These catheters differ from the previous version in three different ways [11]. They have two intracardiac electrodes that allow continuous measurement of the patient's electrocardiogram and of the R-R interval (or a connection with cardiac monitoring in more recent devices); they have a rapid response thermistor (response time between 50 and 70 ms); and they have a special injection port that allows more complete distribution throughout the right ventricle.

The 'volumetric' PAC thermodilution curve is processed and the logarithmic decay portion of the curve is calculated on a beat-to-beat analysis, as determined by the intracardiac electrodes. By calculating the residual temperature change between beats, the computer determines the RVEF. The RVEF is then used to calculate the RVEDV, as described in the following equation:

$$\text{RVEDV} = \text{CO}/(\text{heart rate [beats/min]} \times \text{RVEF})$$

RVEDV should represent a left ventricle preload indicator, but some major problems were identified in the earliest studies. RVEDV was compared with PAOP, which is acknowledged to be a poor indicator of preload dependency. There is mathematical coupling between CO and RVEDV, because the RVEDV index is calculated from stroke volume. This mathematical coupling has been proposed to account for the significant correlation between these two parameters. To overcome this methodological problem, several authors measured CO independently using indirect calorimetry, two different thermodilution technologies, or transoesophageal echocardiography [12,13]. In those studies, RVEDV remained correlated with CO, and RVEF measured using a 'volumetric' PAC was equivalent to RVEF measured using another method.

Figure 2



A schematic representation of the electric circuit analogue of the pulmonary circulation. **(a)** Pulmonary artery pressure decay after occlusion of the balloon during a respiratory hold in the presence of mechanical ventilation. **(b)** An additional trace using 20 data point moving average smoothing of the original trace (collected at 100 Hz) is superimposed on the curves. This further facilitates the visual estimation of the capillary pressure by defining more precisely the point of divergence of the occluded and nonoccluded curves. In addition, an exponential curve has been fitted onto the curve 0.3-2 s after occlusion. This fitted curve has been extrapolated to the time of occlusion to provide the capillary pressure. Pcap, pulmonary capillary pressure. Reproduced with permission from Takala [8].

That the new, volumetric PAC can measure both RVEF and RVEDV is an important feature. Continuous measurement of RVEF and of RVEDV (CEDV) should be useful in guiding haemodynamic treatment. RVEF reflects the right ventricular contractility and afterload, whereas RVEDV provides information on right ventricular preload. Initial studies found a good correlation between RVED and CO, but in most of them the RVEDV was no different before and after fluid loading, challenging the ability of RVEDV to predict fluid responsiveness [14-17]. However, in two studies indexed RVEDV was significantly lower before than after fluid challenge [18,19]. Above a value of 138 ml/m^2 patients did not respond to further fluid loading with an increase in CO, and under 90 ml/m^2 a high percentage of patients were responders to fluid loading. However, between these two values RVEDV index was unable to predict fluid responsiveness. This lack of ability to predict preload dependency was recently confirmed during cardiac surgery in which CEDV was used [20]. The concept of an optimal value of RVEDV was probably an oversimplification of a complex relationship between preload, contractility and afterload. Because of contractility and afterload, RVEF should probably be taken into consideration when interpreting RVEDV. However, if a single value of RVEDV is unable to predict fluid responsiveness, then trends over time should be of interest if they are combined with other parameters from the PAC, particularly continuous CO (CCO) and continuous measurement of Svo_2. Right ventricular failure could be another field of interest for CEDV, particularly in guiding treatment.

Cardiac output determination

Thermodilution: the bolus method

Measurement of CO using a PAC is based on the injection of tracer into the right atrium and analysis of the change in its concentration in the pulmonary artery. If it is assumed that the mass (M) of tracer is constant, then it has been shown that M is equal to the product of the blood flow (Q) and its concentration over time (C), as expressed in the Stewart–Hamilton equation.

$$M = Q \times \int C(t)dt$$

Currently, the tracer usually used is an injection of cold solution in the right atrium. The temperature variation is monitored in the pulmonary artery. The above equation can be expressed as follows:

$$V_i (T_b - T_i) (\rho_i C_i/\rho_b C_b) = Q \times \int T dt$$

$$Q = V_i (T_b - T_i) \times (\rho_i C_i/\rho_b C_b)/\int T dt$$

Where V_i is the volume of injectate, T_i is the temperature of injectate, T_b is the blood temperature, T is the variation in temperature over time, ρ_i and ρ_b are the specific gravities of injectate and blood, and C_i and C_b and the specific heats of injectate and blood.

The final Stewart–Hamilton equation includes a correction factor that depends on the type of catheter used [21]:

$$Q = V_i (T_b - T_i)/S \times (\rho_i C_i/\rho_b C_b) \times k$$

Where S is the area under the thermodilution curve and k is the catheter constant.

Despite these correction factors, several methodological limitations persist [3,22]. First, heat transfer to right atrium blood, wall and surrounding tissue lead to overestimation of CO. Intracardiac shunts, baseline temperature variation in pulmonary artery blood, abnormal haematocrit and cardiac arrhythmia are other sources of errors. Second, conditions surrounding the injectate infusion may represent a further source of error. A 10 ml injectate at room temperature seems adequate in most circumstances (2.6–4.2 l/min per m^2), but cold injectate is recommended in low flow and hyperdynamic states. Variations in volume and speed of the cold tracer infusion can induce differences between measurements. Mechanical ventilation induces complex variations in CO, which depend on the clinical situation. For all of these reasons, variations between two single measurements of up to 25% can occur, and it is therefore recommended that a minimum of three bolus measurements throughout the respiratory cycle be averaged. The variation between two series of three measurements is reduced to 15%. A third methodological limitation is that rapid change in temperature induced by rapid fluid administration (>1 l/hour), use of an upper body warming blanket and extracorporeal oxygenation decrease the accuracy of CO measurement. Finally, in tricuspid regurgitation the transit time of the tracer is increased and the temperature is modified by regurgitation of blood into the right atrium. Therefore, tricuspid regurgitation can induce overestimation or underestimation of CO.

Continuous cardiac output

In contrast to intermittent thermodilution, the tracer used for CCO is not cold but warm. A 10 cm thermal filament is inserted into the catheter at the level of the right ventricle. The surface temperature of the filament is always below 44°C. Low levels of heat energy are transferred to the blood according a pseudo-random binary sequence. A cross-correlation based on the input sequence and the downstream signal measured by the thermistor is performed. The heat signal is processed over time and the classical thermodilution curve is rebuilt. CO is determined using a modified Stewart–Hamilton equation. The CO value is an average over a 3 min period (minimum) [22] and not a beat-to-beat measurement. It is an 'almost' continuous CO measurement.

In vitro studies found good accuracy but with a systematic trend toward overestimation [23]. The degree of over-estimation is nevertheless lower with CCO than with the bolus method [23]. CCO has been evaluated in humans in comparison with reference methods such as the Fick method, dye dilution (indocyanin green) and electromagnetic measurement of aortic blood flow (often considered the 'gold standard' in the cardiac laboratory) [24-26]. In all studies the bias was acceptable (–0.48 to +0.35 l/min, with precision of 0.56–0.74 l/min) [24-26]. Accuracy of the bolus method is lower with very high and very low CO. The classical limitations of thermodilution also apply to CCO monitoring, but because the CCO value represents an average of measurements made over a period of time, it might be expected that increasing the integration period of the signal could decrease the influence of the usual factors that limit the thermodilution technique, such as tricuspid regurgitation, cardiac arrhythmia and baseline variation of pulmonary artery blood, among others. When compared with the bolus method, CCO determination has negligible bias but exhibits better reproducibility, probably because CCO monitoring avoids interindividual variations in volume and speed of infusion of the tracer bolus [27].

Mixed venous oxygen saturation

In several studies a drop in SvO_2 has been associated with a poorer prognosis after cardiac surgery [28], in severe cardiopulmonary disease [29], and in cardiogenic and septic shock [30,31]. Therefore, SvO_2 monitoring should be of interest in critically ill patients. SvO_2 is considered an index of global oxygenation, reflecting the balance between DO_2 and oxygen consumption (VO_2). The main determinants of SvO_2 are VO_2, haemoglobin, arterial oxygen saturation (SaO_2) and CO. At constant VO_2 and with haemoglobin and CO within

the normal ranges, there is good correlation between SvO_2 and CO. Because the mathematical relationships between SvO_2 and its determinants are linear (VO_2 and SaO_2) or curvilinear (haemoglobin and CO), the weight of these determinants is not the same; the influence of VO_2 and SaO_2 is independent of their absolute values, but a small decrease in CO in a hyperdynamic state does not induce any change in SvO_2. Impaired microcirculation, as occurs in severe sepsis or septic shock, induces a deficit in oxygen extraction so that the SvO_2 will not necessary decrease, even in the presence of an inadequate VO_2/DO_2 relationship. In this situation a normal SvO_2 is not equivalent to adequate organ perfusion. Finally, the SvO_2 is a global index of oxygenation, and does not provide information on regional perfusion.

For all of the reasons given above, SvO_2 must be interpreted with caution. It is not an index of inadequate CO, and each of the four determinants must be considered. However, whether SvO_2 does or does not correlate with CO is not of major importance. In the majority of cases a decrease in SvO_2 represents an alert that the global VO_2/DO_2 relationship is inadequate, regardless of the source of this decrease. For example, even if the drop in SvO_2 is secondary to an increase in VO_2 (and not to a decrease in CO or DO_2) during weaning from mechanical ventilation, this variation in SvO_2 must be taken into consideration because it reflects the fact that the cardiorespiratory status of the patient does not fit the new situation. In this example one should consider whether there is a need for transfusion, whether left ventricular failure is present and the respiratory load; postponement of weaning may be necessary [32]. Hence, even when CO is not directly involved in a SvO_2 variation, reconsideration of therapeutic approach can ensue.

Continuous measurement of mixed venous oxygen saturation

In several studies, an unexpected drop in SvO_2 has been observed following cardiac surgery, emphasizing the potential value of continuous SvO_2 monitoring [33,34]. Measurement of SvO_2 has been available since fibreoptics were incorporated into the PAC. Spectrophotometry is the reference technique for measuring oxygen saturation. The absorption spectrum of red blood cells depends on the relative concentrations of oxyhaemoglobin and haemoglobin. Using the continuous method, the emitted light illuminates the blood within the vessel lumen, and is backscattered and refracted by the different blood cells and the vessel wall. The reflected light is retransmitted to a photodetector by one or two bundles of fibreoptics. Depending of the device, two or three wavelengths between the red and infrared domains are used. Oxygen saturation is assumed to be a function of the ratio of reflected light at the various selected wavelengths.

The accuracy of continuous measurement has been tested *in vitro* and *in vivo* [35-38]. In both the correlation is good and bias – in those studies that reported it – is small. Ideally, continuous measurement of SvO_2 should be done over 24 hours; this avoids significant drift and the need for recalibration. However, several factors have been found to influence the accuracy of the method, namely blood flow velocity, distance between the catheter and the vessel wall, red blood cell shape and refractive index of the plasma [22]. The use of three wavelengths, which is theoretically better at avoiding such disturbances, was not found to be more accurate than using two wavelengths in a clinical study [39].

Taking into account the ratio of benefit to risk with pulmonary artery catheterization, a central venous catheter with continuous monitoring of oxygen saturation (central venous oxygen saturation [$ScvO_2$]) has been developed. Several studies have compared the accuracy of the two methods [40,41]. In critically ill patients there is a systematic positive shift of between 5 and 8 mmHg in $ScvO_2$ compared with SvO_2 [40]. This shift may be explained by a relative increase in cardiac and cerebral blood flow in circulatory failure and a redistribution of blood flow in sepsis associated with a proportionally reduced blood flow in hepatic, splenic, mesenteric and renal territories. In most studies there is a wide range of 95% limits of agreement (when available) in intensive care as well as in anaesthesiology, indicating variability between the two methods [40,41]. Therefore, individual values of SvO_2 and $ScvO_2$ are not equivalent. However, in the same studies [40,41] the bias was low (<2–3%), indicating that the trends of SvO_2 and $ScvO_2$ are similar (Figure 3). Clinical decisions are rarely based on a single measurement of a single parameter. Trends in these parameters and their variations following therapeutic decisions are often more interesting.

If we are to use SvO_2 as a parameter for haemodynamic monitoring, then we must define a threshold value. A few years ago, Rivers and coworkers [42] used continuous monitoring of $ScvO_2$ in an early goal-directed therapy algorithm in severe sepsis and septic shock patients. The threshold value of $ScvO_2$ was 70%. That approach was effective; the mortality rate in the early goal-directed therapy group was decreased by 16.7% in comparison with the control group. If we consider the shift of 7 mmHg between SvO_2 and $ScvO_2$ in critically ill patients [40], a SvO_2 threshold of around 65% should be used in further studies.

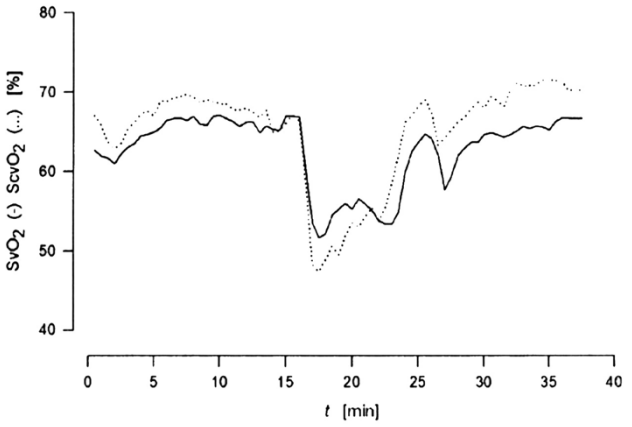
Pulmonary artery catheter as an integrated physiological tool in clinical practice

To this day data on PAC use does not indicate benefit for unstable patients in the intensive care unit [43-46]. Some results are more promising in high-risk surgical patients but remain contradictory [45,47,48]. In a recent report a large database of patients with severe trauma was developed; patients with severe trauma, severe shock and older patients had better survival when treated with the help of a PAC. Therefore, data from PAC use are rather confusing and

somewhat contradictory. The lack of benefit reflects more the lack of controlled trials with treatment algorithms including all PAC parameters than the ineffectiveness of PAC guidance. Some of them compared PAOP with central venous pressure in predicting response to fluid loading. However, neither PAOP nor central venous pressure is a good indicator of preload dependency [5]. Other trials used CO measurement by PAC to achieve 'supranormal' oxygen transport in critically ill patients [49,50]. In this latter setting, it is not the accuracy of CO measurement by PAC that is challenged but the pathophysiological hypothesis that there is any benefit from a 'supranormal' oxygen transport [49-52].

It does not make sense to use a PAC, with the attendant potential adverse events of right cardiac catheterization, without using all of its capabilities. CO is probably the main parameter used in most studies, but CO is a regulated variable and it is necessarily arbitrary to predetermine a CO threshold value. There is no good or bad CO value, but there is CO that permits or does not permit an adequate Do_2. As a global index of adequacy between Vo_2 and Do_2, Svo_2 is the target of choice for therapeutic decisions. Svo_2 should be kept above a threshold value and all other PAC parameters should be used to choose how to maintain Svo_2 above this threshold value. As described above, a threshold value between 65 and 70% should be used in future studies. There are few studies using Svo_2 as one of the main therapeutic targets. In a study conducted in cardiac surgery patients [28], the first objective in the goal-oriented haemodynamic group was to keep the Svo_2 above 70% and lactate serum concentration below 2 mmol/l. This Svo_2 goal was achieved by giving more fluid loading, more inotropic drugs and less vassopressor medication. The result was a decrease in morbidity and in-hospital stay. A more impressive example was reported by Rivers and coworkers [42]. The very early timing of the protocol and the quick transfer to intensive care certainly played some role in the success of this study. Clearly, $Scvo_2$ is the therapeutic target that explained most of the difference between the control and the optimized group, prompting faster fluid loading, more red blood cell transfusions and – in a few patients – administration of an inotropic drug (dobutamine). Therefore, in future studies using a PAC, Svo_2 should be at the centre of the haemodynamic algorithm. Preload indicators (PAOP, right atrial pressure, CEDV), CO and other parameters such as hemoglobin concentration and arterial oxyhaemoglobin fraction must be used to choose the optimal way to achieve the desired objective of an Svo_2 above 70%. Of course, such a strategy should not be applied in too dogmatic a way. The clinical context should always be taken into consideration. For example, a patient with a severe systolic dysfunction following extensive myocardial infarction would certainly have an Svo_2 below 70%. However, if the function of the other organs is not compromised, then optimization of CO by inotropic drugs might impair the myocardial dysfunction further and have an affect opposite to that expected.

Figure 3



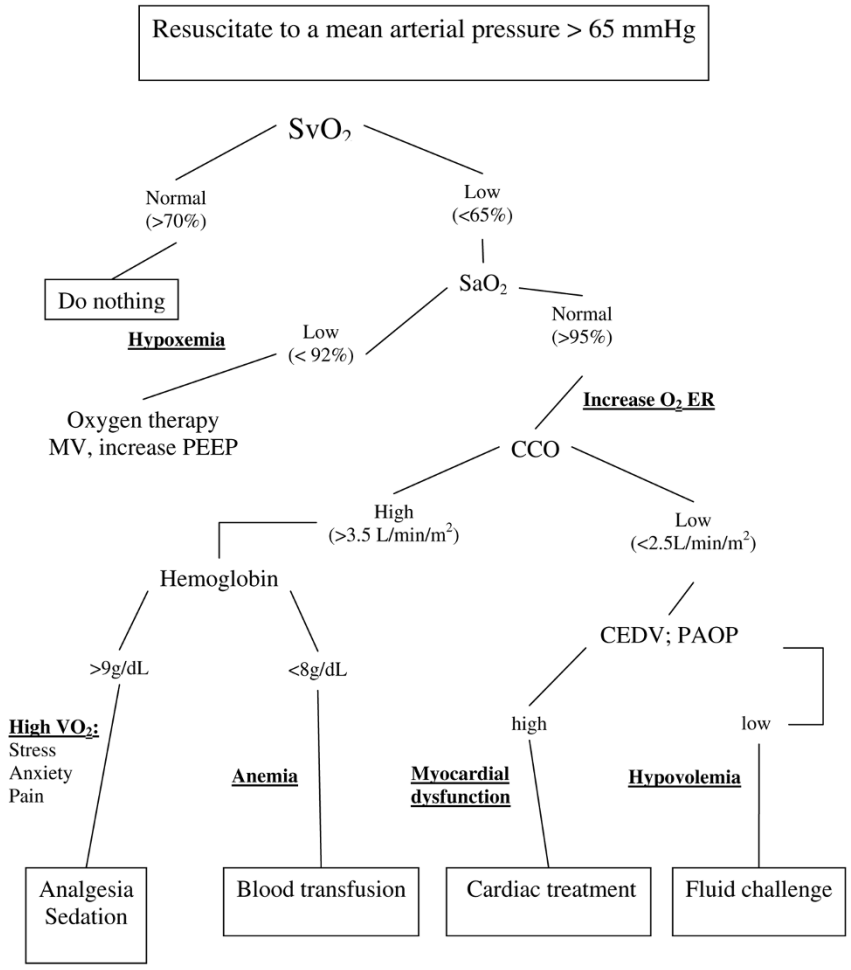
Continuous measurement of Svo_2 and $Scvo_2$. Shown is the time course of continuous measurement of Svo_2 and $Scvo_2$ in a patient with acute respiratory distress syndrome who developed tension pneumothorax, which was treated by insertion of a chest tube. Svo_2, mixed venous oxygen saturation; $Scvo_2$, central venous oxygen saturation. Reproduced with permission from Kasnitz and coworkers [29].

Conclusion

Since the beginning of the 1970s, PAC has been the reference method for haemodynamic monitoring in critically ill patients. Despite the arrival of new devices (invasive or not), thermodilution is the reference method for measuring CO at bedside. The possibility to measure continuously some parameters such as CO, Svo_2 and CEDV provides further insight when monitoring. With former PAC technology (as opposed to the continuous PAC), the haemodynamic status of patients was assessed every 4–6 hours in the best case. Many haemodynamic events were missed by intermittent measurements. Several studies, most of them conducted in patients who had undergone cardiac surgery, revealed unpredictable variations in CO or Svo_2 without necessarily any change in other haemodynamic parameters [33,34,53]. More importantly, continuous monitoring allows physicians to observe the impact of therapeutic interventions, such as fluid loading, inotropic agents, vasopressor and blood transfusion. Continuous monitoring provides a useful guide to therapeutic intervention, and the initial diagnosis may be challenged in case of therapeutic failure.

Numerous devices have been introduced during the past decade for haemodynamic monitoring, some of which are less invasive than PAC placement. Echocardiography is an impressive diagnostic tool, but it can not be repeated indefinitely over time and is operator dependent. Pulse contour methodology has the same limitations as thermodilution (or dye dilution) with respect to calibration, and it is largely influenced by variation in arterial compliance. Oesophageal Doppler must be replaced frequently if patients are not deeply sedated. All of these devices are less invasive

Figure 4



PAC-guided treatment protocol. Therapeutic options to be considered are given in rectangles. CCO, continuous cardiac output; CEDV, continuous end-diastolic volume; O_2ER, oxygen extraction ratio; PAOP, pulmonary artery occlusion pressure; PEEP, positive end-expiratory pressure; RVEF, right ventricular ejection fraction; Sa_{O_2}, arterial oxygen saturation; Sv_{O_2}, mixed venous oxygen saturation; V_{O_2}, oxygen consumption. Reproduced with permission from Pinsky and Vincent [54].

than PAC, but at the same time they have their own limitations. In our view, the most relevant difference between PAC and other haemodynamic monitoring is that the PAC – a single device – permits assessment CO, several preload and fluid challenge tolerances (CEDV and PAOP) and provides a global index of whether oxygen transport is adequate for demand (Sv_{O_2}). Continuous measurement of all of this information transforms the PAC into an integrated physiological device.

Insertion of a PAC is rational if, and only if, all parameters are considered as a whole. The issue is not whether the insertion of a PAC *per se* will improve the prognosis of critically ill patients or high-risk surgical patients, but whether PAC parameters taken together as an integrated component of treatment decision making can improve patient outcomes. Indeed, no monitoring device, regardless of how accurate, invasive, or sophisticated it is, will improve outcome if it is not associated with a specific treatment, taking into account the device's specificity and with an appreciation of the underlying physiopathology. No benefit in terms of outcome is associated with the mere use of echocardiography, blood gas sampling, or determination of blood lactate concentration, but will we cease to use them? Of course not!

It is surprising that no controlled trial of a goal-directed therapy using PAC is actually being conducted. We present an example of an algorithm, adapted from that proposed by Pinsky and Vincent [54] (Figure 4), which could be used in the design of such a trial. Of course, this simple algorithm is not universal and may require modification based on the specificity and the severity of the pathology. Other parameters from different monitoring devices (such as variation in arterial pressure during mechanical ventilation to

assess preload dependency) could be integrated into this strategy. We now need a clinical trial using PAC parameters in a treatment algorithm to determine whether PAC use can improve outcomes in critically ill and high-risk surgical patients.

Competing interests

BV has received consulting fees from Edwards Lifesciences. ER, MC and GL declare that they have no competing interests.

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