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Perioperative hemodynamic monitoring: Still a place for cardiac filling pressures?



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The clinical usefulness of the so-called “static” cardiac filling pressures – central (CVP) and pulmonary-artery-occlusion-pressure (PAOP) – has come into question for guiding hemodynamic therapy due to their poor ability to predict fluid responsiveness in comparison with other monitoring modalities such as transpulmonary thermodilution-derived volumetric measurements, dynamic variables for assessing fluid responsiveness, and the potential risks associated with pulmonary artery catheterization.

This contrasts with observations in multiple patient populations showing a clear association between increased CVP and PAOP levels and poor outcomes, probably due to a reduction in effective perfusion pressure (mean arterial pressure minus CVP) and their role as effectiveness parameters of the cardiovascular system. Furthermore, clinical studies have revealed beneficial effects when interpreting CVP and PAOP dynamically and combining them with flow-related hemodynamic variables. Taking into account the additional information derived from bedside CVP and PAOP pulse curve interpretation, cardiac filling pressures remain an important hemodynamic monitoring tool.

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Background

The so-called “static” cardiac filling pressures - central (CVP) and pulmonary-artery-occlusion-pressure (PAOP) – have traditionally been used for titrating fluid therapy in the surgical and critically ill patient. Within the last decades, their usefulness has come into question due to their poor ability to predict fluid responsiveness in comparison with other monitoring modalities like transpulmonary thermodilution (TPTD) derived volumetric measurements [1] and the potential risks associated with pulmonary artery catheterization [2,3]. Based on these discussions, guidelines for fluid therapy – despite controversial even between the author's [4] – have formulated high grade recommendations prohibiting the use of CVP for diagnosing a fluid deficit in spontaneously breathing as well as ventilated patients and some clinicians even abstain from monitoring cardiac filling pressures at all. Nonetheless, CVP and PAOP are frequently measured variables in perioperative practice [5] and provide multiple information beyond the questions whether a patient may increase stroke volume upon a fluid challenge [6]. The present manuscript thus aims to give an overview about the current role of CVP and PAOP for monitoring in perioperative medicine.

The concept of using filling pressures for guiding fluid therapy

More than one hundred years ago, Ernest Starling described the relationship between right-atrial pressure (RAP), which is equivalent to central venous pressure (CVP), and cardiac output (CO), and showed convincingly that increasing venous pressure leads to an increase in CO [7]. Ignoring the fact that Starling's experiments were performed in an ex-vivo heart preparation and that CVP in an intact organism is influenced not only by the fluid status but also by multiple physiological factors such as right (and left) heart function, intrathoracic and abdominal pressures as well as the tone of venous capacitance vessels, Starling's concept invited clinicians to use the CVP primarily as a preload variable to predict fluid responsiveness [8]; this is though an oversimplification from the physiological point of view.

Focusing on the fact that cardiac output in addition to the cardiac function – graphically displayed as a cardiac response curve - is significantly influenced by the return of the blood to the heart, Guyton introduced the concept of mean circulatory filling pressure (MCFP) – the pressure in the circulatory system during a no-flow situation – and the venous return curve.

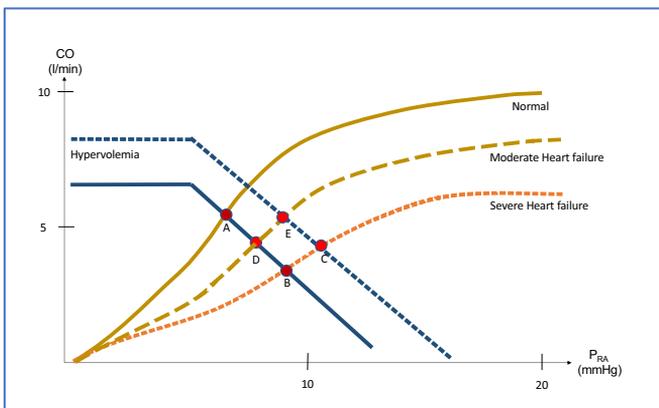


Fig. 1. Combined cardiac function curve and venous return curve under changing conditions. X-axis applies right atrial pressure (P_{RA}), y-axis shows cardiac output (CO). Cardiac function curve (yellow) crosses venous return curve (blue) and defines the working P_{RA} . A is the steady state operating point under physiological conditions. B is the operating point with reduced cardiac performance. C denotes a compensatory increase in vascular resistance and decreased vascular compliance in hypervolemia. A decrease in cardiac function due to heart failure results in a shift of the cardiac function curve downward and to the right. In terms of normovolemia, cardiac output decreases and P_{RA} rises (from control equilibrium (A)→D→B). Heart failure derived hypervolemia results in a right-shift of the venous function curve. Under these conditions, P_{RA} could be elevated with no reduction in CO (E) or with reduction in CO (C), which depends on the severity of the heart failure.

The latter describes the relationship between cardiac output, MSFP and right-atrial pressure (Fig. 1). As pointed out by many elegant reviews on this topic [9,10] and based on Guyton's concepts [11], CVP is a venous downstream variable affected by right ventricular function, reflecting right ventricular pressure during early diastole. Blood flow is generated by the pressure gradient between MCFP and right atrial pressure; with MCFP being the result of the fluid (= stressed) volume within the venous system, venous compliance, and venous resistance [9,12]. The lower right atrial pressure, the higher is cardiac output up to a plateaued maximum defined by the individual myocardial function (Fig. 1). Based on these assumptions, CVP may better be regarded as a variable reflecting the efficiency of the cardiovascular system.

However, despite the abovementioned misconception, derived from Starling's observation, that cardiac output will inevitably increase if the CVP is raised by giving fluids, the likelihood that a patient presenting with a low CVP (as a reflection of low right ventricular end diastolic pressure) subjected to a fluid bolus will increase CO is high [13]. In contrast, it is rather unlikely that a heart working on already increased right ventricular diastolic pressures will comparably respond to a fluid bolus. Thus, despite not correct from the pathophysiological perspective, it is not unlikely that a low CVP may be associated with fluid responsiveness, while high CVP levels are not [13].

In contrast to CVP that can easily be derived from a central venous line routinely inserted for other purposes, determination of the PAOP necessitates the use of a pulmonary artery catheter (PAC). The typical pressure tracings observed during insertion of a PAC are depicted in Fig. 2. Theoretically, assuming a healthy pulmonary arterial and venous bed, PAOP (and diastolic pulmonary arterial pressure) equalize with left atrial (LAP) and left ventricular end diastolic pressure (LVEDP) and may thus give an estimate of left ventricular end diastolic volume. However, because changes in left ventricular compliance (i.e., due to acute ischemia or hypertrophy) will change the relationship between left ventricular diastolic volume and LVEDP, conclusions on the filling status of the left ventricle by measuring PAOP are not necessarily correct [14].

But this does not question the reliability of the pressure measured. Because PAOP is also the backward pressure faced by the pulmonary vasculature, and a pulmonary arterial capillary pressure exceeding 25 mmHg may be associated with extravasation of fluid into the interstitial space and pulmonary edema, PAOP measurements give important information on the risk on this specific cardiopulmonary interaction as well as global left ventricular function. The lower the PAOP, the more likely is a good left ventricular systolic and/or diastolic function and a positive – i.e. increased stroke volume – reaction to a fluid bolus; the higher the PAOP, the more likely is a reduced systolic and/or diastolic function and a direct positive reaction to a fluid bolus will only be seen, even if the left ventricle is capable to adapt for this additional fluid by changing its compliance.

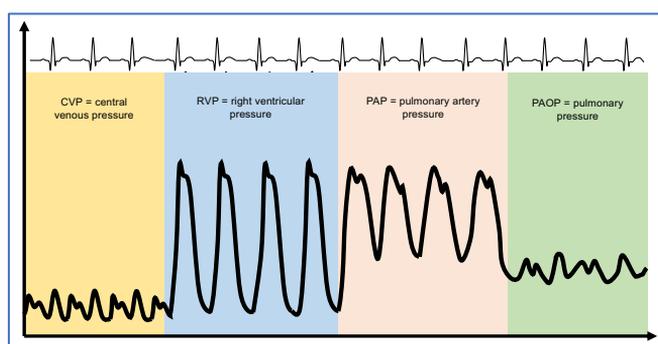


Fig. 2. Characteristic pressure curve changes while inserting a pulmonary artery catheter. A pulmonary artery catheter (PAC) is usually inserted via the superior vena cava. The first measurements correspond to the CVP (yellow). By inflating the balloon at the tip of the PAC and advancing the catheter further, the tricuspid valve is crossed and the catheter is positioned in the right ventricle, right ventricular pressure (RVP (blue) can be measured. Following the blood flow, the catheter can be further advanced through the pulmonary valve into the pulmonary artery (PAP (red)) and into the pulmonary artery occlusion pressure (PAOP (green)) position.

Of note, comparable to the right-sided CVP measurements, absolute PAOP levels are also influenced by intrathoracic and intraabdominal pressures, making it rather difficult to derive information from absolute levels. Thus it is by no means astonishing that several comparison studies revealed a poor predictive capacity of static filling pressures for predicting fluid responsiveness in the clinical setting. Despite this, there are also studies with contradictory results. A group from the Netherlands studied fluid responsiveness in patients undergoing cardiac and vascular surgery [15,16] and observed, that despite TPTD-derived global end diastolic volume (GEDVI) had a good to moderate predictive capacity in the overwhelming number of patients, PAOP had an at least comparable or even better predictive capacity in patients after valve surgery [17] and cardiovascular patients with reduced myocardial function [15]. Taking into account that the validity of TPTD-derived volumetric indices has recently been questioned due to an inability to differentiate patient groups with low and high left ventricular end diastolic volumes confirmed by echo- and angiography [18], and the still unsolved questions of mathematical coupling [19], in especially vulnerable population of patients with structural heart disease, the use of PAOP may still play a role for assessing fluid responsiveness.

Increased cardiac filling pressures and outcomes

If high cardiac filling pressures are reflective of myocardial dysfunction and/or hypervolemia, an association with poor outcomes is more than plausible. Indeed, various studies on patients with heart disease showed that PAOP levels during diagnostic right heart catheterization are associated with poor long-term survival [20]. Comparable observations have been made on the prognostic information derived from CVP measurements in patients with chronic heart failure [21] in an analysis of more than 2500 patients. Damman and coworkers found a significantly lower long-term survival in patients presenting with a CVP higher than 6 mmHg in comparison with patients with a normal CVP (0–3 mmHg). Interestingly, there was also an inverse relationship between CVP and glomerular filtration rate (GFR); the higher the CVP, the lower was the GFR [21].

In line with these findings, Williams and coworkers reported a direct relationship between CVP measured 6 h after ICU admission, mortality, and the need for dialysis in patients after cardiac surgery [22]. Of note is that this association was independent from cardiac function in so far that it was not changed if cardiac output was higher or lower than 2.0 l/min m^2 . These findings clearly show that increased cardiac filling pressures have adverse prognostic implications.

While the association between left heart dysfunction of either systolic or diastolic nature is obvious, it is presently not clear, if the poor outcomes observed with high CVP levels observed outside the catheter lab are primarily related to myocardial dysfunction or an effect of reduced organ perfusion secondary to a reduction of effective organ perfusion pressure ($\text{MAP} - \text{CVP}$). In support of this hypothesis (and the inverse relationship between CVP and GFR observed in medical patients), we have recently shown that a reduced effective (renal) perfusion pressure is an independent predictor of acute kidney injury in cardiac surgical patients after multivariate adjustments for the presence of myocardial dysfunction and treatment with vasopressors and inotropes [23], suggesting that other treatment-related factors such as fluid overload may be involved.

Protocolized use of cardiac filling pressures during goal-directed hemodynamic optimization

Despite the above mentioned claims that cardiac filling pressures should not be used for guiding fluid therapy [4] available evidence in perioperative and critically ill patients points in completely different direction as several randomized controlled studies support that the use of CVP and PAOP for guiding hemodynamic therapy is associated with improved outcomes, especially if they are integrated in an algorithm for goal-directed optimization.

In line with a relevant number of other studies outside the surgical setting, e.g., in patients with lung failure [24] or patients with severe sepsis or septic shock [8], Venn and coworkers were among the first to use a dynamic algorithm for CVP-guided fluid optimization in patients undergoing surgery with hip-fractures and showed a significant reduction in morbidity and mortality in comparison with a control group. Of note, the benefit was comparable to the reduction in complications observed in patients treated with stroke volume optimization [25]. Donati et al. and Osawa et al. showed significant reductions in complications in general as well as cardiac surgical patients, respectively, treated either by

optimization of systemic oxygen balance or by optimization of stroke volume [26] by using a CVP of 10 mmHg as a cutoff for giving a volume challenge or dobutamine [27] or as a stop signal (increase in CVP upon fluid loading of >4 mmHg [26]. Comparably, Polonen and coworkers used a PAOP of 18 mmHg as a stop signal during goal-directed hemodynamic optimization in cardiac surgical patients for a mixed venous oxygen saturation equal to or greater than 70% and a plasma lactate below 2 mmol/l and again showed a significant decrease in complications [28].

These positive findings using cardiac filling pressures in a protocol of goal-directed hemodynamic optimization contrast sharply with the notion that these variables should not be used for guiding fluid and hemodynamic therapy [1]; in particular, taking into account that almost all studies employing TPTD-derived volumetry in the operative and the critical care setting have failed to prove superiority in comparison with a standard, pressure based optimization concept [29]. However, it is important to note that – with the exception of the study by Venn [25] – beneficial effects were predominantly observed if CVP and PAOP were combined with flow or oxygen balance-related hemodynamic variables [30]. The simple use of only PAOP (without measuring cardiac output, oxygen delivery, and/or SvO₂) like in the ESCAPE heart failure trial may thus be not the optimal way to proceed [31].

CVP, RVP, PAP, and PAOP for assessing right heart function

Perioperative right heart dysfunction and pulmonary arterial hypertension are associated with adverse outcomes across many clinical entities and settings [32]. Unfortunately, the evaluation of right ventricular function by echocardiography is not trivial and may be associated with interobserver variability [33]. A direct comparison between right and left heart filling pressures by determination of the ratio between PAOP and CVP is an alternative and functional method to determine the severity of right heart failure. A PAOP/CVP ratio lower than 1 is diagnostic for right heart dysfunction, and a decreasing ratio is associated with disease progression [34].

Despite formally not being a filling pressure, rather than resembling the afterload of the right ventricle, pulmonary arterial pressure is a further important variable to continuously assess the interaction between the left and the right heart and the pulmonary circulation. Because diastolic pulmonary artery pressure (PAPD) is often closely related to the PAOP and LAP, continuous monitoring of PAPD, CVP, and their ratio allows continuous assessment of biventricular function. Additionally, direct right heart pressure (RVP) monitoring with a special PAC has gained increasing popularity within the last years [32]. The details of this monitoring technique are beyond the scope of this overview, however, by monitoring RVP and PAP continuously, acute changes in right heart function can be visualized and treatment effects easily be monitored [32].

Correct determination of CVP/PAOP and pulse-pressure analysis

The continuous recording of the cardiac filling pressures does not only generate a static value but a pulsatile pressure curve. The mechanical heart action consists of 5 phases that follow each other during one cardiac cycle: passive ventricular filling in late diastole followed by atrial systole, isovolumic ventricular contraction, ventricular ejection, and isovolumic ventricular relaxation.

Atrial contraction can be recognized as the a-wave. The c-wave follows when the atrial pressure increases as a consequence of ventricular contraction against the closed atrioventricular valve. In the absence of a tricuspid valve stenosis, the pressure at the end of the a-wave corresponds to right ventricular end diastolic pressure (RVEDP) [9]. Movement of the valve plane to the apex during the ejection phase is called x-descent. The v-wave – passive filling of the atrium during the late systole and early diastole – leads to the y descent after opening the valve.

In contrast to RAP that – with the rare exception of a stenosis of the superior vena cava due to a large aneurysm of the ascending aorta or tight suturing after bicaval cannulation for cardiopulmonary bypass – is identical to CVP, left atrial pressure (LAP) can only be indirectly assessed by PAOP. However, the PAOP curve is similar to the pulse pressure curve of RAP/CVP with three positive and two negative deflections (Fig. 3).

Pleural and intrathoracic pressure change physiologically during inspiration and expiration. During spontaneous breathing, the negative pressure during inspiration leads to a decrease in the cardiac

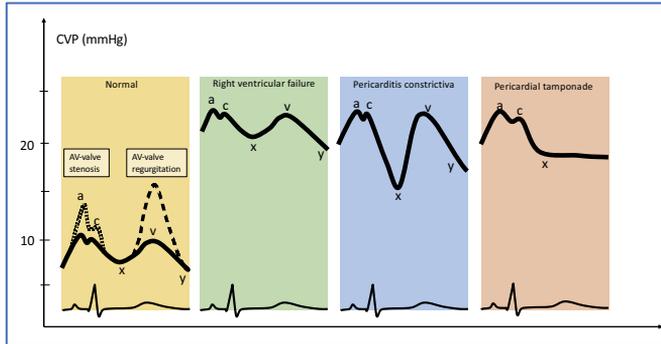


Fig. 3. Specific pressure curve of CVP/PAOP under changing conditions.

filling pressures, while upon expiration filling pressures increase. In contrast, positive pressure ventilation causes an increase in pressure during inspiration and a decrease during exhalation.

Absolute CVP and PAOP values should be measured at the so-called z-point - the end of the QRS complex between a and c wave – which best reflects RVEDP and should ideally be determined during end expiration when the difference between intrathoracic and atmospheric pressures is minimal [35]. This, however, may be challenging, especially in mechanically ventilated patients.

The visual analysis of the CVP and PAOP pulse curve provides important information on valve pathologies and rhythm disturbances. A high and sharp amplitude of the a-wave – which is also known as canon wave due to atrial contraction against a stenotic or closed AV - valve may indicate tricuspid or mitral valve stenosis or be an expression of atrio-ventricular desynchrony due to a nodal rhythm or inappropriate DDD pacing. The most commonly observed disorder is an excessive v-wave as a sign of tricuspid (CVP) or mitral valve (PAOP) regurgitation [36].

Additional diagnostic information

Apart from absolute values and typical changes in the respective pulse pressure curve, pronounced fluctuations of CVP and PAOP during inspiration in spontaneous breathing patients may be suggestive of increased breathing work due to pulmonary obstruction, reduced pulmonal compliance, or inadequate ventilator support [37].

Increased CVP may be an expression of disturbed right atrial inflow in the sense of congestion. “Kussmaul’s sign” describes a clinical situation with high right atrial pressure, which does not decline with inspiration but instead increases [38,39]. Therefore, neither the evaluation of one absolute value per se, but rather the development of the measured variables has to be considered. Of note, “Kussmaul’s sign” is not specific and may occur in any condition with increased right atrial pressure – like pericardial tamponade, severe right ventricular failure, and pericarditis constrictiva – and reflects a loss of pericardial compliance with dissociation between intracardiac and intrathoracic pressure and can again be detected by pulse-pressure-curve-analysis of the CVP. During constriction, the wave form can be described as an M or W, which is an expression of prominent x descent and – as a reflection of rapid diastolic emptying of the high-pressure right atrium – a striking rapid y descent. This is also called “Friedreichs-sign” [38]. This contrasts with the CVP pulse pressure curve during pericardial tamponade that is characterized by a reduction/loss of the y descent (Fig. 3).

While these are only a few and prominent examples of hemodynamic disturbances that may be detected by visual inspection of the CVP and PAOP pressure curve, they clearly show that it is important to visualize these pressure curves instead of only taking a note of the computer-generated mean value on the bedside-hemodynamic monitor. Continuous monitoring of the CVP trace has thus received a grade A recommendation in the recent revision of the German S3-guideline for the monitoring and hemodynamic therapy of cardiac surgical patients [40].

Conclusion

In conclusion, cardiac filling pressures are pivotal hemodynamic variables that give important information on left and right heart function, cardiovascular, and cardiopulmonary interactions. The association of high filling pressures with adverse outcomes, the successful integration of dynamic CVP and PAOP interpretation, and their use as “start and stop signals” during goal-directed therapy, as well as the additional information derived from visual analysis of the CVP and PAOP pressure curve challenge the frequently expressed skepticism regarding the use of cardiac filling pressures for guiding fluid therapy during hemodynamic optimization and beyond.

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Practice points

- Cardiac filling pressures give important information on left and right heart function, cardiovascular, and cardiopulmonary interactions.
- High cardiac filling pressures are associated with adverse outcomes.
- CVP and PAOP have been successfully integrated into goal-directed hemodynamic optimization protocols.
- Visual analysis of the CVP and PAOP pressure curves may additionally be used for bedside evaluation of tricuspid and mitral valve function, the hemodynamic effects of rhythm disturbances, and for the diagnosis of constrictive pericarditis and pericardial tamponade.

Research agenda

- Further research is warranted to determine whether CVP and PAOP may better be combined with blood flow (stroke volume index, cardiac index) or oxygen balance (oxygen extraction ratio, mixed venous oxygen saturation)-associated hemodynamic variables.

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