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Haemodynamic monitoring in acute heart failure

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Abstract Acute Heart Failure is a major cause of hospitalisation, with a rate of death and complications. New guidelines have been developed in order to diagnose and treat this disease. Despite these efforts pathophysiology and treatments options are still limited. There is agreement among the experts that increasing the cardiac output and the stroke volume without fluid overloading the patient should be the goal of every treatment. Despite this, there is no agreement on how to monitor the cardiac function and how to follow it after a therapeutic intervention. In other fields of critical care cardiovascular monitoring and application of early goal directed protocols showed benefits. This review explores the available possibilities of how to monitor the cardiac function in Acute Heart Failure. Standard and more advanced techniques are presented. Cardiac output monitors from the pulmonary artery catheter to the pulse pressure analysis and Doppler techniques are discussed, with focus on this specific clinical setting. Undoubtedly monitoring is valuable tool, but without a protocol of how to manipulate the haemodynamics, no monitor will prove alone to be beneficial. Haemodynamic driven early goal directed therapy are largely awaited in this field of medicine.

Keywords Heart failure · Haemodynamic monitoring · Preload · Cardiac Output · Pulmonary artery catheter

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Introduction

Acute heart failure (AHF) affects millions of people in the United States and Europe alone and is a leading cause of hospitalisation. Despite the size of the problem, neither the pathophysiology of AHF nor its ideal management have been definitively established. Guidelines published 1 year ago by the European Society of Cardiology define AHF as the rapid onset of symptoms and signs secondary to abnormal cardiac function, with or without any preexisting cardiac disease [1]. These guidelines represent a great step forward for the management of AHF, but more evidence is still required to assess whether the recommended protocols and therapies, including monitoring methods, may beneficially affect outcome. In this paper, we will discuss the rationale for monitoring patients with heart failure and the different methods available to the clinician.

The ESC guidelines recommend that monitoring begins as soon as the AHF patient arrives in the emergency department. Non-invasive and invasive methods exist. Non-invasive monitoring assembles a composite picture of the patient's situation using clinical examination, heart rate, respiratory rate, temperature and urine output as well as non-invasive blood pressure (NIBP) and electrocardiogram readings. Pulse oximetry is used to estimate arterial haemoglobin saturation with oxygen (SaO_2) and echocardiography informs estimates of both cardiac output and cardiac function.

Invasive methods include invasive arterial pressure (IAP) monitoring, central venous lines to measure central venous pressure (CVP) and central venous oxygen saturation, and pulmonary artery catheterisation (PAC) to measure pulmonary artery pressure (PAP), pulmonary artery occlusion pressure (PAOP), cardiac output (CO) and the mixed venous oxygen saturation.

The role of cardiac output monitoring in the management of AHF has not been fully established, although expert opinion suggests it should be performed more often. Until 15 years ago the PAC was the only way of continuously measuring cardiac output, but recently new, less invasive devices have been developed that allow the clinician more options in monitoring this variable [2, 3]. This article presents a review of non-invasive and invasive monitoring methods with particular attention to their use and their application under the AHF management guidelines.

History and aims of monitoring

Monitoring facilitates diagnosis in cardiovascular failure and tracks changes following the start of therapy. It is indicated when the information it provides may inform management decisions. In particular, knowledge of preload, contractility and afterload gives the clinician a better picture of cardiac function, and enables therapy to be fine-tuned accordingly. Such cardiovascular monitoring was first attempted using central venous pressure (CVP), and, more importantly, pulmonary artery catheterisation (PAC).

PAC was developed to answer clinicians' questions about heart failure [4, 5]. Although few studies have since assessed the role of such haemodynamic monitoring in the management of AHF, the PAC's ability to measure pulmonary artery occlusion pressure (PAOP) represented a revolution in cardiac monitoring [6]. CVP had been used as an indicator of preload, but PAOP, as an estimate of pulmonary venous pressure, gave a closer picture of left ventricular filling pressure and of the hydrostatic pressure responsible for pulmonary oedema formation, making it a more complete reference point. Additional features now allow the most sophisticated PACs to give continuous estimates of cardiac output (CCO) and volume indications such as right ventricular end-diastolic volume (RVEDV) [7–9]. Although in the last 10 years critics have questioned the difference the PAC makes to patient outcomes, its contribution to cardiovascular monitoring and to the understanding of the haemodynamically unstable patient is beyond debate.

Several newer devices have been developed to address the same clinical questions that the PAC began to answer 30 years ago. These have shifted their focus from pressure to volumes, and are less invasive [10]. Many of these newer devices can match or surpass the additional functions of the newest PACs, but the initial measurements the PAC was designed to gather, continuous monitoring of PAOP and pulmonary pressure, can still only be obtained with the original instrument. Because of this, PAC is still a valuable tool. It is the only device that can continuously monitor the response to treatment for pulmonary hypertension, and cardiac output measurement using intermittent thermodilution with a PAC is still considered the gold standard in clinical practice. The PAC is currently recommended for haemodynamically unstable patients who are not responding to treatment, and for patients with coexisting congestion and hypoperfusion, where PAC information can guide fluid loading and inotropic and vasoactive treatment [1].

Evidence for or against the PAC

The Pulmonary Artery Catheter has probably been the most debated tool in intensive care medicine in the last 10 years. A study by Connors et al. [11] opened the controversy in 1996. Their large prospective non-randomised study showed that even after correcting for treatment selection bias, patients treated with a PAC had a higher mortality rate. The device was not abandoned, but clinicians began thinking more critically about its use. Prior studies by Iberti and Trottier provide an interesting background to Connors et al.'s results [12, 13]. In a study investigating clinicians' understanding of the information supplied by the PAC, Iberti found almost 50% of doctors were unable to correctly identify a PAOP value to within 10 mmHg from a clear trace [12]. Trottier found in another study that clinicians failed to identify PAOP in 33% of the cases [13]. This provided an alternative explanation: that incorrect use of the PAC rather, than the device itself, was associated with the lack of improvement in patient outcomes. Also prior to the Connors study, Shoemaker published results from an optimisation study that randomised patients to three treatment groups: one protocol group in which therapy was guided with data from a PAC, one group in which a PAC was used by a clinician without a specific protocol, and one group in which CVP was used to guide therapy [14]. The protocol group had reduced mortality with respect to the others. Despite this evidence that protocol is important in determining outcome for patients for whom PAC is used, many subsequent studies have not used protocols. A small study (33 patients) published by Guyatt in 1991 had also found an increased mortality for patients monitored with a PAC, but here no protocol was used [15]. Several randomised trials were made after the Connors study to further investigate whether the use of a PAC itself was associated with an increased mortality. Rhodes et al [16] found in a 201-patient randomised controlled study that there were no differences in mortality between the two groups, although in this study there was also no protocol in the PAC group. And in 2005 Harvey et al. [17] published results from a large randomised controlled trial (PAC-MAN) that showed no clear evidence of either benefit or harm resulting from PAC use, although again this study also lacked a common treatment protocol.

Looking specifically at heart failure, the evidence is similarly equivocal. In 1978 Kovick et al. [18] reported the use of PAC to guide vasodilator therapy in chronic heart failure. Pierpont later confirmed that it was useful to use PAC in medical management of heart failure [19]. Gore found in 1987 in a retrospective observational study of patients with acute myocardial infarction (AMI) that PAC use was associated with increased mortality after adjustment for bias variables [20]. Another retrospective observational study of AMI patients by Zion also found no difference in mortality between patients treated with or without a PAC, after correcting for severity of illness [21]. In 2005 the ESCAPE trial examined whether PAC use improved outcome in patients with severe acute-on-chronic heart failure requiring hospitalisation. In this randomised controlled trial the main target of therapy was to reduce congestion by trying to reduce preload, guided by either PAC (targeting PAOP < 15 mmHg and CVP < 8 mmHg) or by clinical assessment [22]. The primary end point was the number of days alive after discharge from the hospital in the first 6 months, and secondary end points were exercise, quality of life, and biochemical and echocardiographic changes. PAC use was not associated with increased mortality but there was no clear benefit to patient outcomes. It is worth noting again that no standardised protocol was used in this study, even though the PAC group had target values for both PAOP and CVP.

Standard monitoring

A standard complete clinical examination should be performed for every patient. A full blood count and measurements of blood electrolytes, creatinine and glucose should also be made, as well as testing for markers of infection or other metabolic disorders. Although the physical examination is of vital importance in the assessment of patients with acute heart failure very few studies have been done to detect the sensitivity and specificity of clinical tests. Various studies have demonstrated the association of a third sound (S3 or gallop) with adverse outcome in patients with heart failure, but it has not been confirmed in all the studies done [22, 23]. This is similar to the SOLVD study for the assessment of the efficacy of ACE inhibitor in heart failure, where patients that at the baseline had higher JVP or an S3 had more advanced heart failure [24, 25]. There are not many data on the ability of clinical tests to assess hypoperfusion. In 1969 the temperature of the big toe was correlated to cardiac output ($r = 0.71$) and severity of shock [26] although not at a level acceptable enough for clinical practice. This has been confirmed by other authors who suggest that clinical examination is a very poor indicator of either perfusion or volaemic status [27, 28].

An electrocardiogram can help to determine the nature of heart failure, and should be performed in every patient with suspected AHF. Pulse Oximetry should be used to estimate the saturation of haemoglobin with oxygen (SaO_2). For patients not in cardiogenic shock, this non-invasive estimate of SaO_2 will be within 2% of the value measured by a CO-oximeter. Pulse oximetry can be useful in titrating the fraction of inspired oxygen (FiO_2) for a patient with an oxygen mask and is a valuable monitoring tool for patients treated with Continuous positive pressure ventilation (CPAP) and Non-invasive ventilation (NIV). Non-invasive blood pressure (NIBP) should also be routinely and regularly measured in patients with suspected or diagnosed AHF. An interval of 5 min is appropriate to monitor changes following treatment with vasodilators, diuretics and inotropes. Monitoring should continue until the readings stabilise. However, NIBP monitoring can lose its accuracy where there is intense vasoconstriction and we suggest that when hypotension calls for the use of inotropes or vasopressors, a step up to invasive arterial pressure monitoring should be considered. When monitoring blood pressure invasively it is important to monitor the mean value, and not only systolic or diastolic values. Mean arterial pressure is not affected by the site of cannulation, but this is not true for systolic pressure, which is higher in the periphery than in the proximal aorta. Where the patient is haemodynamically unstable or where multiple arterial blood samples are needed, the ESC guidelines recommend that an in-dwelling arterial catheter be inserted [1].

Advanced monitoring

Advanced monitoring using specialised devices can obtain more detailed information about cardiac function.

Preload indexes

The ESC guidelines for managing AHF recommend lowering the pulmonary capillary occlusion pressure to <18 mmHg and increasing cardiac output and/or stroke volume as the two foremost haemodynamic goals [1]. From the first of these it can be seen that fluid overloading must be avoided. Preload optimisation is thus mandatory, and as a result many measurement indices have been developed for this variable. Although newer and more representative indices of preload have shifted their focus from pressure to volume, the former is still the basis for the most widely used preload measurements in clinical practice [10]. Most of the new devices that measure volumes are also able to calculate cardiac output, which is important for the second of the above haemodynamic goals [29–33].

Central venous pressure

A central venous pressure line makes it possible to monitor CVP and central venous oxygen saturation ($ScvO_2$), as well as deliver drugs and fluids. Since Starling demonstrated a relationship between CVP and cardiac output and CVP and venous return, this measure has been used as a preload index in clinical practice, although debate over its value has become more intense in the last 10 years. CVP is fundamentally the result of two variables: the amount of blood in the central venous compartment and the compliance of that compartment. Starling's work on the relationship between venous return and ventricular function assumed that all the factors affecting the circulation stayed in equilibrium. However, this does not always hold true in clinical practice, especially for patients in whom the main site of pathology is the cardiovascular system itself. CVP's value as a preload index is further compromised by the effect of changes in intrathoracic pressure related to respiration (spontaneous breathing, PEEP, CPAP, etc.) on the vena cava. The most useful preload index would be the transmural pressure between the inside and the outside of the vessel, but this is clinically impossible to measure. Changes in lung compliance, such as caused by pulmonary oedema in AHF, can affect transmural pressure massively, but the intrathoracic pressure variation cannot be easily distinguished from a change in intravascular pressure by measuring the CVP. As a result of these limitations, it is clear that absolute values of CVP rarely predict ''good'' or ''bad'' right ventricular preload. Several studies have shown either very little or no agreement at all between CVP and cardiac output, and changes in CVP predict changes in cardiac output equally poorly [34, 35]. Even when a massively raised CVP in a patient with AHF clearly confirms a pump failure, the measure is rarely useful in titrating fluid management and therapy. Although there may be a close relationship between CVP and PAOP in healthy patients, raising the possibility that targeting CVP could minimise pulmonary oedema, the relationship is not true in disease and in clinical practice it is not possible to use CVP to predict the development of pulmonary oedema secondary to left ventricular failure [36, 37].

Pulmonary artery occlusion pressure

Pulmonary capillary wedge pressure is normally estimated in clinical practice by measuring the pulmonary artery occlusion pressure (PAOP). Ideally PAOP should reflect the left ventricular end-diastolic pressure (LVEDP) and so would be related to the left ventricular end-diastolic volume (LVEDV). In some pathological states this does not hold true. PAOP overestimates LVEDP in patients with mitral stenosis, for example, and mitral regurgitation and diastolic dysfunction also compromise the accuracy of these estimates. In addition, variations in intrathoracic pressure have the same effect on PAOP as on CVP. PAOP is measured intravascularly, but it is the transmural pressure that represents the real filling pressure. The transmural pressure also determines the movement of fluids across the capillary wall in the development of pulmonary oedema. Any force that changes intrathoracic pressure, such as mechanical ventilation or non-invasive ventilation, can thus lead to an estimation error when measuring the PAOP. PAOP has been examined as a preload index in different scenarios, but several studies have failed to show that it is a good index of preload. Other variables concentrating on volume, such as intrathoracic blood volume or right ventricular end-diastolic volume, or dynamic preload indices such as systolic pressure variation (SPV), pulse pressure variation (PPV) and stroke volume variation (SVV) have either proven to be better preload indexes or to better predict fluid responsiveness [10, 38–40].

So how can PAOP be used to guide fluid therapy to optimise preload? Although studies have failed to demonstrate an optimal PAOP value for maximising stroke volume, physiological consideration dictates that there should be a value below which fluid addition would be advisable, and a value above which additional fluid would be detrimental. These levels should be determined by the stroke volume response to fluid loading, with the optimal PAOP being the point where additional fluid produces only a minimum rise in stroke volume. Data from two studies showed that above 18 mmHg none of the patients benefited from fluid loading, while all showed a response at PAOP levels below 8 mmHg [41, 42]. Specific values for AHF patients are not available, but 18 mmHg is an accepted value above which the risk of pulmonary oedema is raised, and so efforts should be made to keep PAOP below 18 mmHg.

Volumetric monitoring

As noted above, measurement of pressure is of limited reliability in assessing preload. Volume estimation has been developed as way of bypassing this problem. Volumetric indexes aim to quantify the volume of a specific compartment of the cardiovascular system. Echocardiography can be used for volumetric monitoring, either completely non-invasively (transthoracic, TTE) or moderately invasively (transoesophageal, TOE). In the hands of an expert clinician this technique allows immediate assessment of several aspects of cardiac function. As well as visualising the action of the heart, echocardiography can be used to measure pulmonary pressures (if concomitant tricuspid regurgitation is present) and cardiac output, based on the Doppler principle. Echocardiography also allows the measurement of

left ventricular parameters. LV diameter, area and volume have all been studied and shown to be good indicators of preload. LV size decreases after volume depletion and increases after blood restitution in experimental studies. LV end diastolic area (LVEDA) is the best proxy measurement for LV size, and seems to best reflect changes in blood volume when measured through TOE [43–45].

Intrathoracic blood volume (ITBV), a measurement delivered by the PiCCO system (Pulsion, Munich) has also been studied as a preload index and has been shown to be superior to CVP and PAOP in several clinical settings. Although specific data for AHF patients are not available, studies in cardiac surgery, in lung transplantation and in liver transplantation have shown that intrathoracic blood volume index (ITBVI) is a better predictor of stoke volume than either CVP or PAOP [46–48]. Extravascular lung water (EVLW), another parameter calculated by the PiCCO system, reflects the volume of water outside of the vascular system, as an indicator of pulmonary oedema. In ITU settings high values of EVLW have been associated with worse outcome [49–51]. One study by Mitchell showed that targeting a low EVLW for ventilated patients reduced time spent on the ventilator, although unfortunately this study used a double indicator technique that is no longer available, and no study has since been carried out using PiCCO to confirm this result [52]. No data for EVLW in AHF are available. Right Ventricular End-Diastolic Volume (RVEDV) is a parameter available in some rapid response sensor PACs. RVEDV is more reliable than PAOP in reflecting the preload status of the heart, but still requires pulmonary artery catheterisation.

Contractility and afterload

The previous section concentrated on measures of preload. Contractility and afterload, the other two determinants of cardiac output, can also be monitored, but the same breadth of technology has not been developed to target these measures. As yet, contractility can only be assessed by echocardiography. Since afterload is the resistance to flow that the heart works against, some monitors have been developed to evaluate this parameter by estimating systemic vascular resistance (SVR). SVR is a calculated value equal to (mAP – CVP)/CO. This value is often calculated to give an idea of how vasoconstricted a patient is, but it is not measured. There are a number of theoretical reasons why this variable is not valid and there is no evidence that titrating therapy according to it is of any benefit.

Cardiac output

Cardiac output measurement options have recently expanded through innovations in monitoring technology. While 15 years ago the only way to measure CO was with a PAC, now the same monitoring function can be performed by many new devices, such as the PiCCOplus [47], LiDCOplus [32], PRAM [53], Vigileo, FloTrac [54], echocardiography [43], Doppler devices (CardioQ, Hemosonic, etc.) [33, 55] and impedance cardiography [56–58]. These methods have been validated against the intermittent thermodilution of the PAC. PiCCO, LiDCO and Doppler have been available for more than 5 years now and have been the most intensively studied of the new technologies. Their continuous tracking of CO allows the clinician to evaluate the progress of therapy during its administration. Specific data for AHF are not available, but many studies have been conducted in other clinical settings showing good agreement with the gold standard pulmonary thermodilution. Cardiac output monitoring with some of these machines has also been used in goal-directed therapy protocols that have been shown to improve patients' outcome [59, 60].

Conclusions

We know from other fields of critical care that monitoring cardiac output improves patient outcome when used to manipulate therapy at the right time (i.e. before irreversible oxygen debt is established). This can be done with several haemodynamic monitors. Perhaps the more important point to press home is that for monitoring to provide any benefit, the clinician must be using the information to guide management. Studies that have used protocols for early optimisation of patients' haemodynamic performance have been shown to improve outcome, while studies that have not used a protocol, or that have used one without a specific time focus, have not shown any benefit and in some cases have been harmful. We still await studies assessing the effectiveness of early haemodynamic optimisation protocols in the AHF setting. In the meantime, we reinforce the ESC guidelines' recommendation to focus on improving haemodynamics. The monitoring technology used to achieve this is probably of subsidiary importance compared to how the monitoring information is used to guide treatment, since monitoring itself will never cure anybody.

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