Cardiac Preload Evaluation Using Echocardiographic Techniques

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Introduction

For many decades, central venous (CVP) pulmonary artery occlusion pressures (PAOP), assumed to reflect of right and left filling pressures, respectively, have been used to assess right and left cardiac preload. Although they are obtained from invasive catheterization, they are still used by a lot of physicians in their fluid infusion decision making process [1]. Many approaches have been proposed to assess preload using non-invasive techniques. Echocardiography and cardiac Doppler have been extensively used in the cardiologic field but have taken time to be widely used in the intensive care unit (ICU). However, echocardiography is now considered by most European ICU physicians as the first line method to evaluate cardiac function in patients with hemodynamic instability, not only in terms of diagnosis but also in terms of the therapeutic decision making process [2–3]. Regarding cardiac preload and cardiac preload reserve, cardiac echo-Doppler can provide important information.

Echocardiographic Indices

Vena Cava Size and Size Changes

The inferior vena cava is a highly compliant vessel that changes its size with changes in CVP. The inferior vena cava can be visualized using transthoracic echocardiography. Short axis or long axis views from a sub costal view are used to measure the diameter or the area of this vessel [4]. For a long time, attempts were made to estimate CVP from measurements of inferior vena caval dimensions. Because of the complex relationship between CVP, right heart function, blood volume, and intrathoracic pressures, divergent results were reported depending on the disease category of patients, the timing in measurement in the respiratory cycle, the presence of significant tricuspid regurgitation, etc. While Mintz et al. [5] found a good positive correlation ($r = 0.72$) between the end diastolic inferior vena cava diameter normalized for body surface area and the right atrial pressure, others found poor correlations between absolute values of inferior vena cava diameters and right atrial pressure [4, 6, 7]. In patients receiving mechanical ventilation, three studies have evaluated the correlation between inferior vena

cava size and right atrial pressure [7–9]; Lichtenstein et al. found a good correlation whereas Nagueh et al. and Jue et al. observed unsatisfactory correlation. This may be due to different techniques used to measure the diameter of the inferior vena cava [10]. When inferior vena cava size is measured using a two-dimensional method, correlation with right atrial pressure is poor. Using M-mode measurements, correlation was demonstrated to be good. To summarize all these findings, a small inferior vena cava size corresponds to normal right atrial pressure. An inferior vena cava diameter equal or inferior to 12 mm seems to predict a right atrial pressure of 10 mmHg or less 100% of the time. In contrast, an increased inferior vena cava size may correspond either to a normal or increased right atrial pressure. Importantly, inferior vena cava size depends on end-expiratory pressure in mechanically ventilated patients [11]. Therefore, inferior vena cava diameter increases when end-expiratory pressure increases. So, in patients with a high end-expiratory pressure, an increased inferior vena cava size may be present in patients with a low or normal right atrial pressure.

In the same way, the transverse diameter of the left hepatic vein was measured to assess right atrial pressure. Luca et al. demonstrated a good correlation between expiratory or inspiratory diameters and right atrial pressure. Moreover, percentage increments of left hepatic vein diameter correlated well with percent changes of mean right atrial pressure during the rapid infusion of 250–5000 ml of saline [12].

Right atrial pressure was also assessed by recording inferior vena caval flow using pulsed Doppler and analyzing tricuspid annulus movement using Doppler tissue imaging (DTI).

More interestingly, in spontaneously breathing patients, the collapsibility index, defined as the inspiratory percent decrease in inferior vena cava diameter was demonstrated to be well correlated with the value of right atrial pressure [4, 6, 7]. In spontaneously breathing patients, a collapsibility index > 50% would indicate a right atrial pressure < 10 mmHg with a good predictive accuracy [6] in terms of sensitivity and specificity. Nevertheless, although respiratory variation of inferior vena cava diameter can indicate the level of right atrial pressure, the knowledge of right atrial pressure is of little value for managing patients with cardiovascular compromise, first, because by nature, filling pressures do not fully reflect preload and second, because a given value of filling pressure does not provide relevant information on volume responsiveness in a given patient. In patients receiving mechanical ventilation, while the collapsibility index was reported to fail to reflect CVP [7], the respiratory changes of the inferior vena cava diameter were shown to be highly correlated with the percent increase in cardiac output induced by a 500 ml fluid infusion (Feissel M, unpublished data).

The superior vena cava (SVC) was also analyzed. Vieillard-Baron et al. demonstrated a collapse of this vessel during insufflations in mechanically ventilated patients. A collapsibility index > 60% was described as an excellent predictor of a positive hemodynamic response to fluid challenge (unpublished data).

Interatrial Septal Shape and Movement

The shape and movements of the interatrial septum depend on pressure as well as the size and contraction of left and right atrium during apnea. As with pressure variations, the temporal sequence of right and left atrial contraction is different over a cardiac cycle [13]. Therefore, the interatrial septum has cyclic oscillations depending on the pressure gradient between the left and right atrium. During atrial contraction, the septum bulges into the left atrium. In contrast, during systole the interatrial septum moves into the right atrium and at end-systole into the left atrium. During diastole, the septum bows toward the right atrium (Fig. 1). The amplitude of these movements is less than 1 cm in normovolemia and may be more than 1.5 cm in hypovolemia.

In spontaneously breathing patients, the interatrial septum moves during inspiratory and expiratory phases. During the inspiratory phase, right preload increases and the septum moves toward the left atrium. During mechanical ventilation, movement of the interatrial septum is also observed. Insufflations decrease right preload and increase left prelaod and as a consequence, the interatrial septum is curved towards the right atrium. During the end-expiratory phase, left preload decreases and interatrial septal reverse (right to left) movement is observed [14]. In the same way, pulmonary arterial hypertension changes these movements by increasing right atrial pressure.

PAOP may be assessed using transthoracic echocardiography or transesophageal echocardiography (TEE) by observing curvature and movement of the interatrial septum. The interatrial septum is usually curved toward the right atrium when PAOP > 14–15 mmHg. Mid-systolic reversal (right to left) was demonstrated

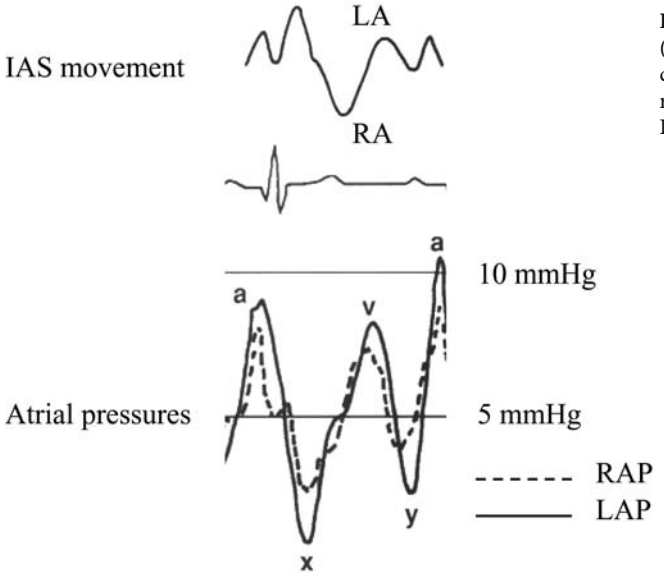

Fig. 1. Interatrial septal (IAS) movement over a cardiac cycle. RAP: right atrial pressure; LAP: left atrial pressure.

when PAOP << 14–15 mmHg. This movement was minimal when PAOP was between 12–14 mmHg and buckling of the septum was noted when PAOP was < 10 mmHg [15].

Therefore, movements of the interatrial septum are complex with variations throughout the cardiac and ventilation cycles. Nevertheless, these movements give information concerning left and right atrial pressures, but should be interpreted with caution particularly in mechanically ventilated patients.

Left Ventricular Dimensions

The end-diastolic size of the left ventricle (LV) determines the strain of myocardial fiber before systolic contraction, which represents the LV preload. In many studies, LV diameter, area, or volumes have been demonstrated to be good indicators of preload. In experimental and clinical studies the LV size has been demonstrated to decrease during provoked volume depletion and to increase after blood restitution [16–19]. Moreover, during provoked hypovolemia induced by stepwise blood withdrawal, the LV size was found to correlate with the amount of blood withdrawn [19]. In many clinical situations, volume depletion is associated with a decreased LV size, particularly during general anesthesia. The best way to quantify the LV size in ICU patients, is to measure the LV area using TEE. From a transgastric view, the LV end-diastolic area (LVEDA) can be measured at the papillary muscle level. Values of 5.2–18.8 cm^2 have been found in a normal population [20]. A good correlation was found between LV area obtained from echocardiography and LV volume obtained from angiography [21]. Cheung et al. [18] demonstrated that TEE was sensitive enough to assess changes in cardiac preload, since in this study, 5% of the blood volume change could be detected using TEE measurement of LVEDA. In another study performed in a pediatric department, TEE was able to detect 2.5% of blood volume changes. In contrast, others found a low sensitivity of TEE in tracking changes in volume status [18]. In a non-published study, we measured LV size using transthoracic echocardiography before and after hemodialysis. After 2 liters of ultrafiltration – which represents a blood volume loss of 250–300 ml – the LV size did not change; this was confirmed by others [22]. Technical problems including low reproducibility of LV measurements in ICU patients could explain these findings. Therefore, in our opinion, LVEDA seems to have a low sensitivity to detect blood volume changes in critically ill patients. Moreover, the LV size has never been described as a predictive index of a positive hemodynamic effect after fluid expansion in patients with shock. Because the LV size is a highly variable parameter, the individual 'optimal' size to obtain the best preload to eject the highest stroke volume is unknown. Patients with LV systolic dysfunction, dilated left ventricle, and a normal or high LV diastolic pressure experience a high preload but may be in hypovolemic shock because their preload may be insufficient to eject the best stroke volume. After a small fluid challenge, such patients may increase LV size and stroke volume without a marked increase in end-diastolic pressure. Thus, the 'optimal' LV size to obtain the optimal stroke volume in such patients cannot be comparable with the optimal LV size in patients without LV systolic dysfunction and dilated cardiomyopathy. It has to be noted

that knowledge of LVEDA has been demonstrated to be of little value in predicting an increase in cardiac output in response to fluid infusion in patients with cardiovascular instability [1]. In patients with sepsis-induced hypotension, responders and non-responders to fluid could not be clearly discriminated before fluid infusion by using baseline values of LVEDA measured using echocardiography. Moreover, considerable overlap of baseline individual values of LVEDA was observed between responders and non-responders supporting the interpretation that a given LVEDA value cannot reliably predict fluid responsiveness in an individual patient [1, 23].

Left Diastolic Pressure Assessment Using Doppler Techniques

Wedge, left atrial, or LV mean or end-diastolic pressures have been proposed to reflect LV preload. Many studies have tried to assess these pressures, using cardiac Doppler.

Mitral Flow

From a 4-apical view, mitral flow may be recorded using pulsed Doppler. This flow is composed by an early (E wave) and late wave (A wave). Several indices have been found to correlate with diastolic pressures: ratio of E to A maximal velocity (E/A), deceleration time of E (DTE) wave, and deceleration time of A wave (DTA). A small E wave, E/A <1, DTE>150 ms [24], DTA >60 ms [25] are usually associated with low LV diastolic pressures [26]. Unfortunately, the mitral flow depends on numerous factors, such as LV relaxation and compliance, heart rate, etc. To this extent, 'normal' mitral flow may be recorded in the presence of high LV pressure in patients with diastolic dysfunction. Recently, it has been proposed that the velocity of the E wave (which is very dependent on diastolic function) should be 'normalized' by a preload-independent Doppler parameter. Maximal early diastolic velocity of the mitral annulus (Em) recorded using DTI and early diastolic mitral flow propagation velocity (Vp) using M-mode color Doppler have been proposed to assess the LV end-diastolic pressure (LVEDP). Values of E/Em <8 [27, 28] and E/Vp <2.5 [29] were found to be usually associated with low LVEDP. Finally, it must be stressed that in the presence of tachycardia (> 120 beats/min) or arrhythmias, little information can be drawn from transmitral flow recordings in terms of assessment of filling pressures.

Venous Pulmonary Flow

Venous pulmonary flow can be used to assess LVEDP. Kucherer et al. [30] were the first authors to report a relationship between the systolic fraction (ratio between velocity time integral [VTI] of the systolic wave and the sum of the VTI of diastolic and systolic waves) and the left atrial diastolic pressure. The systolic fraction (SF) < 55% was described as a sensitive parameter to detect a high left

atrial pressure (>15 mmHg). This flow is also influenced by LV diastolic function and hence should be used with caution in patients with LV diastolic dysfunction.

Combination of Mitral and Venous Pulmonary Flows

During atrial contraction, the blood is ejected into the LV (A wave on mitral flow) and into the pulmonary veins (reverse a wave on venous pulmonary flow). In the presence of high LV diastolic pressure, duration of the A wave shortens and the ratio between the duration of the A and a waves becomes less than 1. Therefore, normal or low LV diastolic pressures are usually associated with an A/a ratio > 1 (31, 32).

This approach of assessing left diastolic pressures has many limitations. First these pressures are different from each other, in particular with mitral valve disease or reduced LV compliance. Second, the relationship between LV diastolic volume and pressure is not linear but curvilinear and depends on the LV compliance such that, for a given LV volume, filling pressures are higher in patients with a reduced LV compliance than in those with normal LV compliance and a change in volume results in more marked changes in pressures in the former group of patients. Third, these indices have never been evaluated in terms of prediction of fluid responsiveness.

Cardiac Output

The cardiac output can be measured easily using echocardiography and Doppler [33]. Many methods using either transthoracic and/or transesophageal approaches have been described and validated in ICU patients [34–36]. Measuring cardiac output at the level of the aortic annulus represents the best technique. Using the transthoracic method, the diameter of the aortic annulus should be measured from a long axis view of the LV at the level of insertion of the aortic valve while aortic blood flow must be recorded using continuous wave Doppler from an apical 5-chamber view. Using the transesophageal approach, the aortic area can be measured directly and aortic flow can be obtained either from a transgastric 5-chamber view or from a transgastric proximal view with an angle of 110–130°. In terms of diagnosis of volume depletion, the information provided by the sole measurement of cardiac output is non specific, since hypovolemic conditions are associated with low cardiac output values as are cardiac failure conditions. However, since echocardiography also gives information on cardiac function, cardiac chamber dimensions, and mitral and pulmonary vein flow patterns, combined measurements of several variables may help to diagnose low volume status. For example, in a patient with no history of cardiac disease, the association of a low cardiac output with a normal ejection fraction should most often lead to the diagnosis of hypovolemia, even if more sophisticated indices are not recorded. Obviously, in the case of prior cardiac dysfunction, the diagnosis of volume depletion could be more difficult to make from such static cardiac echo-Doppler measurements.

Evaluation of Preload Dependence using Doppler Parameters

In patients receiving mechanical ventilation, the magnitude of stroke volume variation over a respiratory cycle has been proposed to provide relevant information on volume status [37]. Indeed, by reducing the pressure gradient for venous return, mechanical insufflation decreases right ventricular (RV) filling and consequently the RV stroke volume, if the RV is sensitive to changes in preload. In this condition, the following decrease in LV filling will also induce a significant decrease in LV stroke volume if the LV is sensitive to changes in preload. Therefore, the magnitude of the respiratory changes in LV stroke volume, that reflects the sensitivity of the heart to changes in preload induced by mechanical insufflation, has been proposed as a predictor of fluid responsiveness [38]. Because the arterial pulse pressure is directly proportional to LV stroke volume, the respiratory changes in LV stroke volume have been shown to be reflected by changes in pulse pressure [39]. Accordingly, the respiratory changes in pulse pressure have been demonstrated to accurately predict fluid responsiveness in mechanically ventilated patients with septic shock [40]. The magnitude of the respiratory changes in systolic pressure has also been proposed to assess fluid responsiveness in patients with acute circulatory failure related to sepsis [41]. Using cardiac echo-Doppler, LV stroke volume can be obtained by calculating the product of aortic VTI and aortic area, measured at the level of the aortic annulus. Because aortic area is assumed to be unchanged over the respiratory cycle, respiratory variation in stroke volume can be estimated by respiratory variation in VTI. Using this hypothesis, we have shown, in a recent experimental study, that the magnitude of the respiratory changes in VTI (recorded by transthoracic echocardiography at the level of aortic annulus) was a highly sensitive indicator of blood withdrawal and blood restitution in rabbits receiving mechanical ventilation [42]. Moreover, this dynamic parameter was able to predict fluid responsiveness more reliably than conventional static markers of cardiac preload measured by echocardiography [42]. The superiority of such dynamic parameters over static ventricular preload parameters to predict fluid responsiveness in critically ill patients has been emphasized recently [1]. In this way, Feissel et al. [23] using TEE, demonstrated that the magnitude of respiratory variation of the peak value of blood velocity recorded at the level of the aortic annulus (Vpeak), was better than static measurement of LVEDA for predicting the hemodynamic effects of volume expansion in septic shock patients receiving mechanical ventilation. In this study, Feissel et al demonstrated that when patients with septic shock experienced a value of Vpeak > 12%, 500 ml fluid infusion increased stroke volume and cardiac output by more than 15% while decreasing Vpeak proportionally [23].

It must be stressed that the use of dynamic parameters such as respiratory variation of surrogates of stroke volume to assess volemic status, must be applied only in patients who receive mechanical ventilation with a perfect adaptation to their ventilator and who do not experience cardiac arrhythmias.

Conclusion

In summary, using echocardiographic and Doppler parameters, low volume status is often characterized by a small inferior vena cava size and large diameter respiratory changes, large respiratory movements of the interatrial septum, small LV size, E/A ratio < 1, DTE > 150 ms, TDA > 60 ms, A/a > 1, SF > 55 %, E/Em < 8 and E/Vp < 2.5, low cardiac output and large respiratory variations of aortic flow or stroke volume.

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