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Assessment of loading conditions with cardiac ultrasound. A comprehensive review

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Abstract

Optimization of the preloading conditions and concomitant determination of endpoints of fluid administration are the most frequent therapeutic actions in critically ill patients. Besides a clinical appraisal, reproducible data should be acquired at the bedside to estimate the adequacy of fluid resuscitation. The dynamic assessment and determination of fluid responsiveness plays a major role in this respect. Right-sided cardiac variables, such as inferior and superior caval vein diameter variation during mechanical ventilation, are easily obtained with cardiac ultrasound. Moreover, left sided variables, including aortic flow variation, with intermittent swings of intrathoracic pressure during mechanical ventilation, may be achieved non-invasively with Doppler-echocardiography. Both in terms of resuscitation, as well as correct interpretation of various two-dimensional and Doppler variables, it is essential to acquire a clear understanding of the filling status of a patient. Doppler-echocardiography plays herein a pivotal role.

Key words: echocardiography, preload, fluid responsiveness, right ventricle, left ventricle

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Adequacy of volume resuscitation and assessment of fluid administration are daily questions in critically ill patients. Fluid loading is the most frequent therapeutic handling performed in anaesthetized and critically ill patients. The appropriateness of loading conditions includes some clinical signs, such as low perfusion pressure, low diuresis and malperfusion of tissues. However, clinical estimation of filling and subsequent optimization needs more than some subjective and rough clinical parameters.

Fluid status determination may be performed by either static or dynamic variables, which should be integrated within the clinical findings. Static variables include preload descriptors without any dynamic component. Dynamic variables include more a physiological approach for testing fluid responsiveness.

Cardiac ultrasound allows bedside assessment of haemodynamics and has been shown to provide invaluable information on ventricular systolic and diastolic function, loading conditions (preload and afterload), valve morphology and function, as well as the status of the great vessels [1]. Whereas traditional haemodynamic monitoring relies on an assessment of pressures and cardiac output, echo-Doppler techniques provide insight into volumes and flows. Therefore, incorporation of cardiac ultrasound into clinical practice offers a much more complete and detailed picture of the haemodynamic status, in a non-invasive manner. Furthermore, correct interpretation of many echo-Doppler parameters obliges one to determine optimal filling status each time an echo-Doppler assessment is performed due to the load dependency of many of these ultrasound variables (Table 1).

As with other monitoring tools, correct handling of cardiac ultrasound needs extensive knowledge of anatomic and physiologic features, besides its handling in order to obtain the imaging and signals in the most optimal and

Table 1. Static load dependent variables, obtained with cardiac ultrasound

Left ventricular end-diastolic area indexed for body surface area (LVEDAI)
Right ventricular end-diastolic area indexed for body surface area (RVEDAI)
Systolic Doppler flow wave in a pulmonary vein (S)
Early filling wave across the mitral valve (E)
Systolic Doppler tissue wave, obtained in the mitral annulus (S')

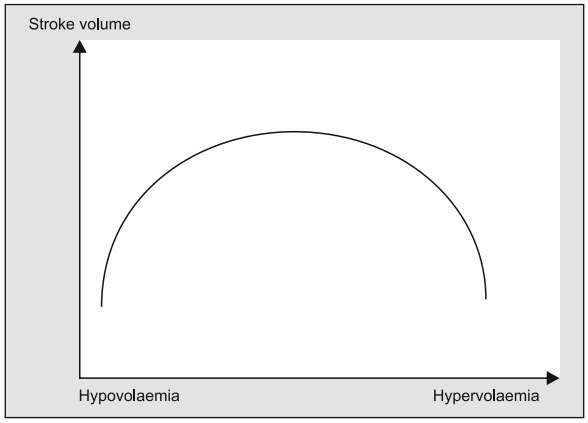


Figure 1. Relationship between stroke volume and the critical optimal zone between hypovolaemia and hypervolaemia. Neither a low filling state nor hypervolaemia will result in an optimal cardiac output

trustworthy manner. Assessment of ventricular function has been well described. However, correct interpretation of ventricular systolic function needs estimation of loading conditions; indeed, optimization of preload often improves ventricular function. The aim of assessing fluid responsiveness should be an objective determination of loading conditions in order to have fluid loading only being reserved for those patients whom it will benefit and to prevent excessive fluid loading (Fig. 1). This analysis aims to review the correct interpretation of the different variables describing loading conditions in the critically ill, being obtained with echo-Doppler techniques, besides indications of clinical confounders, hampering correct analysis of each technique.

PHYSIOLOGICAL UNDERSTANDING OF LOADING CONDITIONS

In essence, preload is a static variable, describing loading conditions of the heart before muscular contraction occurs. It is related to left ventricular end-diastolic pressure (LVEDP) and, through some simplifications, also to left atrial and pulmonary artery occluding pressure (PAOP). Nonetheless, the relationship between pressure and loading conditions is hampered mainly by ventricular compliance. The latter is governed by the function of the opposite ventricle, mostly, though not only, by the interventricular septum, coronary perfusion pressure, pericardial constraint and intra-thoracic pressure. End-diastolic pressure relates to volume whenever ventricular compliance is normal. Hence, in only a few critically ill patients does it follow that LVEDP could be a useful descriptor of preloading conditions.

The balance between optimal preload, contractility and afterload is the mainstay of haemodynamic management and becomes more important whenever the pump (contraction) is seen to be failing. Sedation relieves the sympathic tone, reduces afterload and unloads the heart from the preloading side, inducing a total imbalance with haemodynamic deterioration when pump failure is present. Therefore, estimation and optimization of preload is essential and the first measure in enhancing haemodynamics and even prevailing haemodynamic deterioration (Fig. 1). However, when preload irresponsiveness is present, volume resuscitation may also aggravate pulmonary oedema, with subsequent respiratory failure and weaning difficulties.

While static variables of loading conditions provide a momentary tableau, which could suggest hypovolaemia only in conjunction with some of the general measures listed above, it becomes clear that, nowadays, a dynamic aspect should be included to optimally assess and predict fluid responsiveness. Several possibilities exist, such as an internal transfusion with passive leg raising [2], a mini-bolus of 100 mL colloids [3], or usage of intra-thoracic pressure swings owing to cyclic mechanical insufflation in order to safely determine fluid status of the critically ill [4].

STATIC VARIABLES OF PRELOAD

As with haemodynamic monitoring including assessment of various intra-cardiac pressures, such as central venous pressure (CVP), pulmonary artery occlusion pressure (PAOP) and left ventricular end diastolic pressure (LVEDP), several static variables have been described in cardiac ultrasound. Table 1 provides a list of examples of static load dependent variables in this respect. All of them give a momentary insight of preload, often in conjunction with a measure of systolic function. Is a temporary picture worthless in view of estimating optimal preloading conditions in a haemodynamically unstable patient? As with various filling pressures, static variables may offer adequate understanding of global volume status, if interpreted in a correct context [5]. The most classical example is left ventricular end-diastolic area, indexed for body surface area (LVEDAI). There is no relationship between PAOP and LVEDAI [5–7]. In cardiac surgical patients, LVEDAI has been demonstrated to be sensitive to detect alterations of blood volume, even in patients with regional wall motion abnormalities. Although eyeballing is generally accepted in clinical practice with respect to estimate largeness of the LVEDA, it has been described that a LVEDAI < 5.5 cm m^{-2} clearly depicts a low preloaded status [8], though this finding could not be confirmed in an intensive care unit (ICU) setting [6]. The presence of an end-systolic obliteration in a patient with a hypertrophic left ventricle — with normal contractility — suggests clearly a low filling state, though compliance of the left ventricle should be taken into account with respect to the amount and the velocity of loading [9].

Fluid infusion could induce an increase of LVEDAI up to a certain level, after which it will remain constant, con-

cordant with stabilization of cardiac output [10, 11]. PAOP, however, will rise further, concomitant with further filling. Therefore, LVEDAI is superior to pressure-related static preload descriptors, such as CVP or PAOP, in order to predict fluid responsiveness in a cardiac surgical setting [12]. Left ventricular end-diastolic diameter [13], taken in a short axis view, or — with 3-D echocardiography — left ventricular end-diastolic volume, in a mid-oesophageal (ME) long axis view, may also be utilized as a static variable.

An important shortcoming is the fact that LVEDA always should be assessed at the same position. Though the papillary muscles have been used as an easy marker of position, inclination of the probe within the oesophagus could interfere with a correct estimation of the LVEDA, in particular, in those patients with severely depressed left ventricular systolic function. Hence, a dynamic evaluation of loading conditions is urged.

FLUID RESPONSIVENESS

Traditional measures of preload, such as CVP and changes of CVP with volume loading, have failed to predict responsiveness to fluids [14, 15]. Assessment of loading conditions in patients with increased intra-thoracic or intra-abdominal pressures, intraoperative Trendelenburg positioning (major pelvine surgery), pericardial constraint or right ventricular failure, particularly appears to be an indication for dynamic load evaluation, rather than using static preload characteristics. Furthermore, only dynamic variables followed the changes induced by transfusion in a rabbit model [13].

Thus, either mechanical ventilation induced alteration of intra-thoracic pressure, passive leg-raising or a mini-bolus may be used to determine fluid responsiveness in sedated or anaesthetized patients on a mechanical ventilator.

PASSIVE LEG RAISING AND STROKE VOLUME

Passive leg-raising has been utilized already for many decades and offers the possibility to safely transfuse 150–200 mL of whole blood into the central circulation [16]. A rapid increase of ventricular preload and, hence, cardiac output could be achieved whenever preload dependency is present. In addition, this technique offers complete reversibility by returning the legs horizontally. Important with this technique is the definition of a positive response, which is often set at an increment of 10–15% [16, 17]. LVEDAI may be monitored by a transthoracic or transoesophageal approach, assessing the increase of this measure during passive leg-raising. Therefore, invasive arterial pressures are not directly necessary to determine fluid responsiveness.

Reversibility of the testing with a short-term increase of preload underlines the safety of this technique. Nevertheless, it should be taken into account to evaluate global ventricular function previous to a passive leg-raising test. A dilated right or left ventricle certainly will hamper the effects of rapid filling.

Several mechanisms interplay with the increased preload. Firstly, increased systemic venous return is achieved in preload dependent patients. Secondly, stimulation of atrial baroreceptors with inhibition of vagal outflow and stimulation of sympathetic efferent fibers to the heart may also lead to haemodynamic changes during passive leg raising [18]. Thirdly, awakening could induce reflexes during sedation. Finally, the choice of sedation could interfere with presence of preload responsiveness: indeed, propofol was shown to increase preload responsiveness, whereas dexmedetomidine had no impact [19].

MECHANICAL VENTILATOR INDUCED INTRA-THORACIC PRESSURE CHANGES AND THE RIGHT HEART

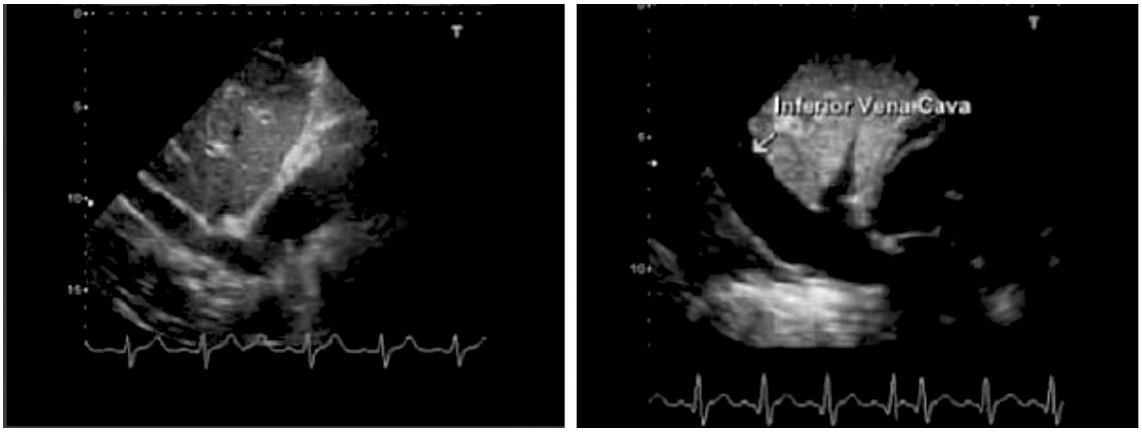
During mechanical ventilation in a well-sedated adult patient, cyclic alterations of intra-thoracic pressure induce changes of the diameter of the venous inlet into the thorax, i.e. the inferior and superior caval veins (Figs. 2, 3). With transthoracic echocardiography, it is easy to demonstrate the dilation and decrease of diameter of the inferior caval vein (IVC) with inspiration and expiration, respectively. Barbier *et al.* and M Feissel *et al.* demonstrated clearly that respiratory variation of the IVC reliably predicts fluid responsiveness [20, 21]. Conversely, in acutely decompensated heart failure patients, the rate of fluid withdrawal during haemodialysis can be guided by intermittent evaluation of the respiratory induced alterations of the IVC diameters [22]. In this particular study, hypotension was observed in those patients with an IVC variation of > 30%.

Superior caval vein (SVC) variation during mechanical ventilation can be monitored by means of transoesophageal echocardiography in a minimally invasive manner [23]. Collapse of the SVC during inspiration has been related to low intra-thoracic blood volume [24]. A collapsibility index (CI) has been defined [25] as:

$$CI = SVC\ max - SVC\ min\ /\ SVC\ max$$

It has to be noted that SVC max is observed during expiration (lowest intra-thoracic pressure), owing the position of the SVC in the thorax. This CI index exceeds 36%, providing a good discrimination of responders to blood volume [25].

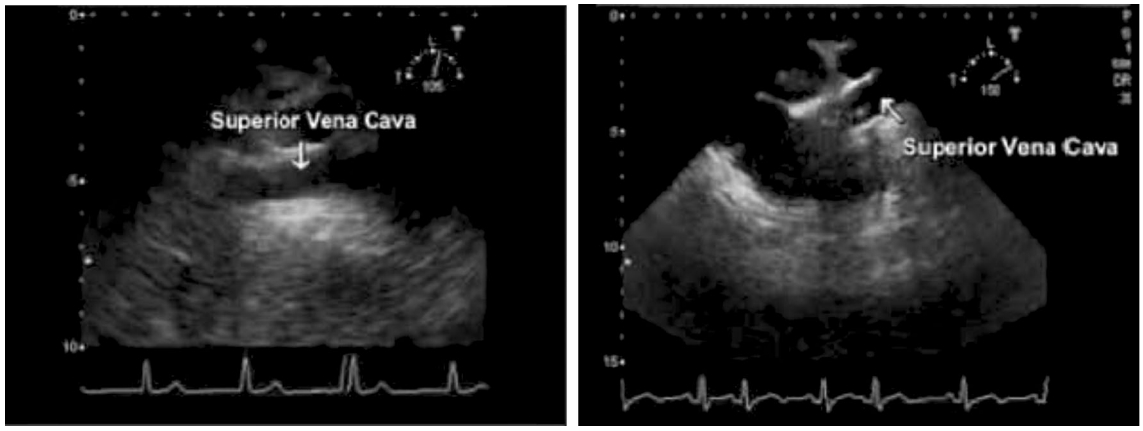
Recently, a simultaneous comparison between IVC (by the transthoracic approach) and SVC (by transoesophageal echocardiography) variation in mechanically ventilated patients showed a better performance of SVC variation in predicting fluid responsiveness [26]. The threshold for the SVC was found to be 29% (sensitivity 54% and specificity 89%). Apparently, the impact of intra-thoracic pressure



Responsive Non-responsive

Figure 2. Variation of inferior vena cava, IVC (left panel: responsive, right panel: non-responsive) with cyclic swings of intrathoracic pressure, e.g. during mechanical ventilation. Responsiveness is defined as ΔIVC > 18% according to the formula below:

$$\Delta IVC = 100 \times \frac{IV\,C\,insp - IV\,C\,exp}{IV\,C\,insp} > 18\%$$



Responsive Non-responsive

Figure 3. Variation of superior vena cava, SVC (left panel: responsive, right panel: non-responsive) with cyclic swings of intrathoracic pressure, e.g. during mechanical ventilation. Responsiveness is defined as ΔSVC > 36% according to the formula below:

$$\Delta SVC = 100 \times \frac{SV\,C\,exp - SVC\,insp}{SVC\,exp} > 36\%$$

changes during mechanical ventilation, including increased right atrial pressure, squeezing the inter-alveolar capillaries and hence, increased right ventricular impedance was larger upon the SVC than the influence on backflow or, at least, delayed filling of the right atrium, as assessed in the IVC. The anatomical position of the SVC inside the thoracic cavity may explain the better performance of this vessel in demonstrating fluid responsiveness.

Nevertheless, in many critical situations with mechanical ventilation, it is clear the transthoracic approach assessing cyclic IVC variations is easy and clinically useful. Therefore, it appears logical that the IVC-view has been integrated in FAST imaging protocols [27] and is the first choice of assessment. Only in those situations where transoesophageal echocardiography and Doppler is used, will SVC imaging guide decision making with respect to fluid management.

Of note, both IVC and SVC diameter variations, with altering intra-thoracic pressure during mechanical ventilation, do provide an insight in right ventricular fluid responsiveness. Correct interpretation will be hampered whenever there is an occurrence of right ventricular failure [28], increased abdominal pressures [29], open chest (during or after cardiac surgery) [30, 31], or too small shifts of intrathoracic pressure (low tidal volume [32, 33], increased intrathoracic pressures, increased work of breathing). In contrast, an increased respiratory rate (neonates and small children) allow for a still correct estimation of fluid responsiveness by means of IVC variation [34].

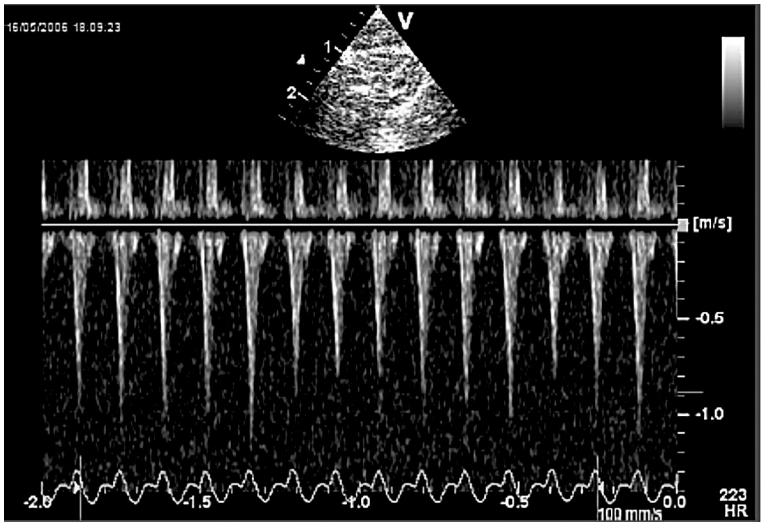


Figure 4. Variation of trans-aortic flows, assessed with continuous wave Doppler, with cyclic swings of intrathoracic pressure

MECHANICAL VENTILATOR INDUCED INTRA-THORACIC PRESSURE CHANGES AND THE LEFT HEART

Stroke volume variation is the physiological effect of cyclic altering intra-thoracic pressure during mechanical ventilation of the left heart. Stroke volume can be derived from the area under the curve of a transaortic valvular Doppler signal (velocity time integral, VTI), obtained in a deep transgastric view [35, 36]. VTI is actually the distance at which one red blood cell is pushed with a single contraction of the left ventricle. The following formula permits the calculation of SV:

$$VTI * AVA = SV$$

AVA may be be determined by calculation of this area at the level of the aortic valve (**π*** diameter/2) or using the mean aortic valve area over the whole ejection cycle [37], which is a more practical approach in daily clinical practice.

An increase of SV with > 15% induced by passive leg raising was shown to have a specificity of 93% and a sensitivity of 81%, a positive predictive value of 91% and a negative predictive value of 85% [2]. The whole estimation could be simplified by replacing SV by VTI: indeed, this has the advantage that SV is much more rapidly estimated. Furthermore, this approach is far less prone to mistakes and over- or underestimations by omitting the issue of aortic valve area. The magnitude of the mechanical ventilation induced alterations of VTI accurately predicts the changes of cardiac output during acute bleeding or transfusion [13]. Hence, the formula to be determined in estimating fluid responsiveness could be rewritten as follows:

$$\Delta VTI\ (\%) = 100*(VTImax - VTI\ min)/[(VTI\ max + VTI\ min)/2]$$

with a responder variation of 20% [38]. Figure 4 shows clearly the mechanical ventilation induced variations in the aortic Doppler signal. Delta down could be noted; the latter is supported by a decline of systemic venous return or an increased right ventricular afterload. Only echocardiography may differentiate between the two phenomena: collapse of the IVC or SVC suggests a preload effect, whereas intermittent dilation of the right ventricle supports the idea of increased right ventricular impedance.

Delta up is the consequence of a squeezing of the alveolar capillaries during inspiration of blood into the left atrium or/and a decrease of left ventricular afterload in patients with afterload dependent hearts [39].

SVV has been shown to be an adequate predictor of fluid responsiveness in various studies [40]. ΔVTI has been compared with Vigileo (Edwards Lifesciences, Irvine, USA) derived SVV with similar performance in a setting of liver transplantation and vasopressor support [4], though with normal systemic vascular resistance. The area under the ROC curves to discriminate volume responders versus non-responders by both methods, were not different. Nevertheless, caution is advisable as different monitors use different algorithms while stroke volume monitors have never been validated for rapid changes of stroke volume during one breath [41]. A major contraindication of the use of ΔVTI to estimate fluid responsiveness is aortic valve disease (stenosis, insufficiency), even with low trans-aortic pressure gradients. In this situation, right-sided measures should be utilized in this respect.

Similarly, in spontaneously breathing patients, increases of stroke volume by means of passive leg raising, assessed by

cardiac ultrasound, has been shown to correlate with those changes estimated by a Vigileo system [42]. In intermittent spontaneous breathing, interpretation is more difficult as the swings of intra-thoracic pressure will not always be equal. Longer periods of evaluation should be included to gather the required information.

CONCLUSIONS

Both right-sided and left-sided dynamic descriptors of loading conditions may be obtained with Doppler-echocardiography. Whereas the SVC variations with changing intra-thoracic pressures appear to be more accurate, both SVC and IVC diameter variations are useful in this setting. Velocity-time variation is more difficult to obtain across the aortic valve, even though this physiological signal offers similar and often non-invasive information of stroke volume variation. Cardiac ultrasound offers one the huge advantage of estimating fluid responsiveness in a mostly non-invasive and speedy manner at the bedside. Three-dimensional cardiac ultrasound of left and right-sided ventricular volumes may result in a quick and easy assessment of preloading data. Moreover, association of a mini-bolus of fluid loading or passive leg raising will help one to identify fluid responsive patients.

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