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ORIGINAL ARTICLE

Static and dynamic preload indicators in multiple organ dysfunction syndrome

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Abstract

In patients with multiple organ dysfunction syndrome, early optimization of cardiac preload is required to improve outcome. This review describes available static and dynamic indices used as bedside indicators of cardiac preload and fluid responsiveness in critically ill patients. Static indices (mostly derived from a pulmonary artery catheter or echocardiography) are based on measurements of cardiac pressures and volumes. Dynamic indices are based on a functional assessment of the pressure–volume relationship and the heart–lung interaction. Available data demonstrated that dynamic indices such as pulse pressure variation and stroke volume variation were more reliable predictors of fluid responsiveness than static indices. Dynamic indices using non-invasive devices are also proposed and it is expected that they will be used more often in future. However, spontaneous breathing movements and arrhythmia are the major limitations to the use of dynamic indices in clinical practice. In this respect, new volumetric bedside assessment methods could represent good alternatives.

Key words: *Arterial systolic pressure variation, cardiac preload, central venous pressure, global end-diastolic volume, pulmonary artery occlusion pressure, pulse pressure variation, severe sepsis, stroke volume variation*

Introduction

Multiple organ dysfunction syndrome (MODS) is defined as the presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention (1). Identification of this abnormality should occur during a period in which organ support can achieve better function and prevent organ failure. MODS is a normal complication of septic shock in which hypovolemia plays an important factor contributing to the impairment of tissue perfusion. Therefore, prompt correction of a vascular volume deficit is a prerequisite for improving survival in patients with MODS. Apart from situations in which hypovolemia is evident and a favourable response to fluid administration will be seen, clinical and biological parameters often fail to predict hypovolemia (2). Inappropriate use of volume expansion carries the risk of generating volume overload and pulmonary oedema and/or right ventricular dysfunction. Consequently, reliable predictors of fluid responsiveness are needed, especially in the early phase of cardiocirculatory deterioration. In the clinical setting, different static and dynamic indices have been shown to be useful indicators of cardiac preload. Static indices are based on methods that are able to assess ventricular volumes and pressures while dynamic indices consist of the assessment of fluid responsiveness using the relation between changes in stroke volume (SV) with positive pressure ventilation. In this review we analyse the clinical value and limitations of the methods most frequently used to assess cardiac preload and predict fluid responsiveness.

Static indices

Cardiac filling pressures

Central venous pressure (CVP) and pulmonary artery occlusion pressure (Ppao) are widely used to measure right and left ventricular preload in patients

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requiring invasive hemodynamic monitoring (3). These hemodynamic parameters are recommended by the American Society of Critical Care Medicine (4) to monitor fluid resuscitation in patients with septic shock in order to optimize cardiac output. Nevertheless, cardiac filling pressures have been found to be inaccurate for predicting fluid responsiveness as the relation between cardiac filling pressure and ventricular end-diastolic volume depends on various factors, including the effect of mechanical ventilation on extramural pressure and changes in ventricular compliance induced by sepsis, ischemia and catecholamine treatment (5–8). Ventricular and vascular compliance is frequently altered in septic patients. For a given pressure, different Frank–Starling curves are possible, depending on cardiac systolic and diastolic function (Fig. 1). In a critical review of available studies evaluating the value of static and dynamic parameters for predicting fluid responsiveness, Michard and Teboul (9) demonstrated that neither CVP nor Ppao was a reliable predictor of volume responsiveness. These results are in agreement with a recent analysis of data on 150 fluid challenges in septic patients published by the same group (5). It was shown that pre-infusion CVP was similar in responders and non-responders (8 ± 4 vs 9 ± 4 mmHg), while pre-infusion Ppao was slightly lower in responders than non-responders (10 ± 4 vs 11 ± 4 mmHg; $p < 0.05$). The authors also demonstrated that available threshold values currently recommended to predict fluid responsiveness are not appropriate to guide fluid therapy in septic patients.

In the setting of acute respiratory distress syndrome, in which mechanical ventilation with a high positive end-expiratory pressure is generally used, Ppao may reflect not the left ventricular end-diastolic pressure but rather the alveolar pressure. In these patients, Ppao, even when measured at end expiration, still significantly overestimates the left ventricular (LV) filling pressure. Two simple methods have been proposed to correct for this overestimation and thus to calculate the true LV filling pressure (7,10). Pinsky et al. (10) demonstrated that in cardiac surgery patients, nadir Ppao measured after airway disconnection from the ventilator was a better estimate of LV filling pressure than Ppao when high levels of positive end-expiratory pressure were used. Teboul et al. (7) have demonstrated that an indexing of the transmission of proportional alveolar pressure to Ppao in the estimation of LV filling pressure is equivalent to the nadir method.

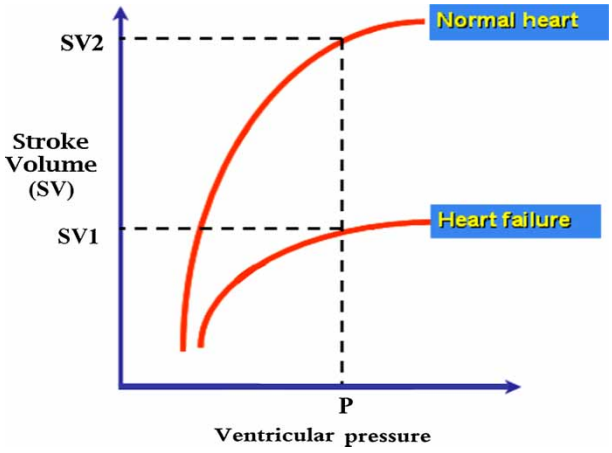

Fig. 1. Frank–Starling curves in normal and failing heart. A given value of cardiac ventricular pressure (P) can be associated with higher SV in the case of a normal heart (SV2) compared to a non compliant failing heart (SV1).

Ventricular end-diastolic volumes

Right ventricular end-diastolic volume. Because right ventricular performance may be the limiting factor in determining cardiac output in many clinical situations associated with pulmonary hypertension, right ventricular end-diastolic volume (RVEDV) was considered an indicator of ventricular preload. Using a pulmonary artery catheter with a fast-response thermistor, it was demonstrated that an RVEDV index (RVEDVI) < 90 ml/m^2 was consistently associated with an increase in cardiac output in response to volume loading (11–14). Conversely, an RVEDVI > 140 ml/m^2 seems to be the upper limit to volume responsiveness (12,13). The presence of tachycardia (> 130 beats/min), arrhythmia and important tricuspid insufficiency constitutes the major limitation to the use of RVEDV as a preload indicator.

Left ventricular end-diastolic area. Conflicting results have been obtained (15–19) concerning the predictive value of the left ventricular end-diastolic area (LVEDA) for predicting cardiac output changes in response to fluid loading. In one study (15) a value of the LVEDA index (LVEDAI) < 9 cm^2/m^2 was predictive of a positive response to fluid loading, whereas other studies failed to identify such a cut-off value. Of note, a great variation in LVEDA was observed in healthy subjects (20) and in anaesthetized patients without cardiac failure (21). Moreover, LVEDA as assessed by echocardiography is operator-dependent and does not always accurately reflect the ventricular end-diastolic volume (22).

Global end-diastolic volume. Volumetric measures of preload, such as intrathoracic blood volume and global end-diastolic volume (GEDV), have repeatedly been shown to reflect preload better than commonly used filling pressures in septic patients. GEDV is the

volume of blood in the four heart chambers measured by means of the transpulmonary thermodilution curve (23). GEDV is considered to be an indicator of cardiac preload and its measurement is less influenced by changes in intrathoracic pressures and myocardial compliance than cardiac filling pressures. This technique needs only a femoral artery sheath and is considered to be less invasive than the use of a pulmonary artery catheter. In patients with septic shock, GEDV was shown to increase with volume loading but not with dobutamine, despite a similar increase in cardiac output (24). In addition, it was observed (24) that the lower the pre-infusion GEDV, the more marked the hemodynamic effects of volume loading. Most studies in which the value of GEDV was assessed were performed in a cardiac surgery setting. In patients studied in the postoperative period after coronary artery bypass grafting (25) and heart transplantation (26), Göedje and co-workers found a significant correlation between changes in SV and changes in GEDV. By contrast, they did not find any correlation between changes in SV and changes in CVP and Ppao. It must be kept in mind that GEDV reflects the preload of the heart as a whole, and does not distinguish between its left and right components. Recently, in a study comparing two thermodilution-based volumetric preload assessment tools with echocardiography, Hofer et al. (27) demonstrated that the GEDV index assessed by means of the PiCCO system better reflects fluid responsiveness than the RVEDVI measured by means of a modified pulmonary artery catheter and the LVEDA measured by means of transesophageal echocardiography. A value of the GEDV index <600 ml/m^2 is predictive of a positive response to fluid resuscitation (24). However, the presence of large aortic aneurysms or catheters placed too peripherally (i.e. in the radial artery) will lead to volumes being overestimated.

Dynamic indices

The main advantage of dynamic over static indices is that they can distinguish between hypovolemia and fluid responsiveness. Indeed, an increase in SV as the result of an increase in preload depends more on the slope of the Frank–Starling curve (Fig. 2) than on a given level of cardiac preload (28). For the clinician, the relevant question is not whether the patient has an absolute blood deficit but whether volume expansion will induce a significant increase in cardiac output. Indeed, the intensive care unit (ICU) patient with hemodynamic compromise needs volume expansion when his/her heart is operating on the steep portion of the Frank–Starling curve (fluid responsiveness); conversely, he/she does not respond to fluids when the heart is operating on the flat portion (fluid unresponsiveness). There is now a great deal of evidence to suggest that dynamic indices based on the heart–lung interaction are better indicators of fluid responsiveness, in particular in patients with severe sepsis receiving mechanical ventilation (15, 17, 29–35).

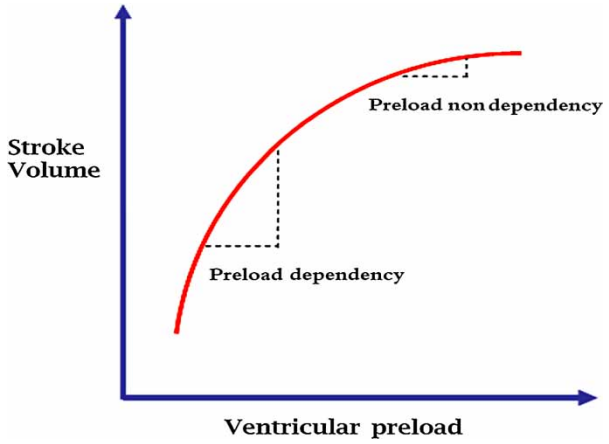

Fig. 2. The Frank–Starling curve showing its two components: the steep (preload dependency) and flat (preload non-dependency) components. The SV increases with cardiac preload in a non-linear fashion. On the initial steep part of the Frank–Starling curve, the increase in preload induces a significant increase in SV (preload dependency). On the distal flat part of the curve, the increase in preload does not induce a significant change in SV (preload non-dependency).

Invasive dynamic indices

Respiratory changes in systolic arterial pressure. When both ventricles are operating on the steep portion of the Frank–Starling curve (preload reserve), mechanical ventilation, by inducing cyclic changes in intrathoracic pressure, may induce changes in LVSV. The higher the change in LVSV before fluid administration, the more important the increase in SV as a result of fluid administration (36). As systolic arterial pressure (SAP) depends on SV, it is assumed that cyclic changes in SV induced by mechanical ventilation could be objectively reflected by changes in SAP. The mechanisms of these changes are explained in Fig. 3. The difference between the maximal and minimal SAP during one mechanical breath is called the systolic pressure variation (SPV). When a significant SPV is observed, it is proposed that an end-expiratory pause should be performed to separate the inspiratory increase in SAP (dUp) from the expiratory decrease in SAP (dDown) (Fig. 4A). The dDown component reflects the inspiratory decrease in venous return resulting in a fall in LVSV during expiration (36). The dUp component is the inspiratory increase in SAP related to emptying of pulmonary capacitance vessels and to increased left ventricular pressure relative to extrathoracic vessels (37). It is accepted that changes in SAP

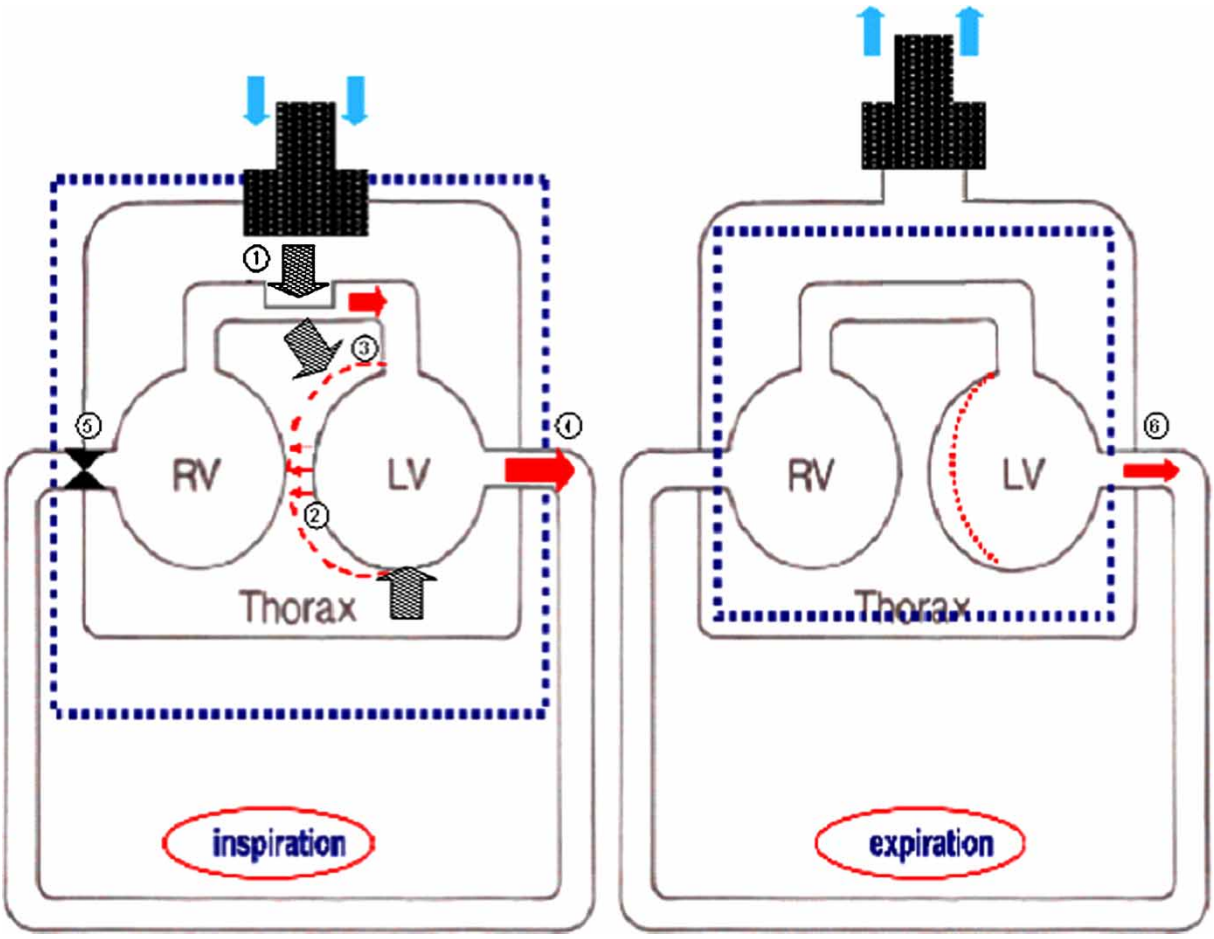

Fig. 3. Mechanisms of cyclic changes in arterial pressure during respiratory support. (1) A sudden increase in alveolar pressure during inspiratory tidal delivery boosts blood from the pulmonary capillary bed, which transiently increases pulmonary venous return. (2) LV enlargement during inflation as a result of an increase in pulmonary venous return. (3) Lung inflation produces an increase in pleural pressure exerting an external pressure on the left ventricle and leading to an improvement in LV systole. (4) The combined result of the effects described above is an increase in SV and SAP described as dUp. (5) The sudden rise in pleural pressure during inspiration increases right ventricular (RV) outflow impedance and decreases venous return. This effect decreases the filling reserve of the left ventricle during the next expiration. (6) The decrease in LV preload is associated with an expiratory drop in SV and arterial pressure.

with changes in blood volume are mainly due to changes in dDown. Accordingly, dDown is a better indicator of fluid responsiveness than SPV and a baseline dDown threshold value of 5 mmHg is an indicator of fluid responsiveness (15).

Respiratory changes in arterial pulse pressure. Because SPV depends not only on changes in SV but also on the cyclic direct effects of intrathoracic pressure on the thoracic aorta wall (38), respiratory changes in arterial pulse pressure (ΔPP) were proposed as an alternative to SPV measurement because the effect of intrathoracic pressure is similar on both systolic and diastolic pressure. The arterial PP is the difference between the arterial systolic and diastolic pressures (Fig. 4B). The arterial PP is directly proportional to the LVSV and inversely related to the compliance of the arterial system. Assuming that arterial compliance does not change during a mechanical breath, respiratory changes in LVSV should be reflected by respiratory changes in peripheral PP. ΔPP is calculated as the difference between the maximal and minimal values of PP over a single respiratory cycle divided by the mean of the two values and expressed as a percentage:

$$\Delta PP\ (\%) = (PPmax - PPmin)/[(PPmax + PPmin)/2] \times 100$$

A threshold value of 13% has been considered (29) as an indicator of hypovolemia and predictive of a positive response to fluid administration.

Pulse contour analysis. The area under the systolic part of the arterial pressure curve is directly related to the SV, at least at the aortic level. Using specific peripheral arterial catheters connected to a compu-

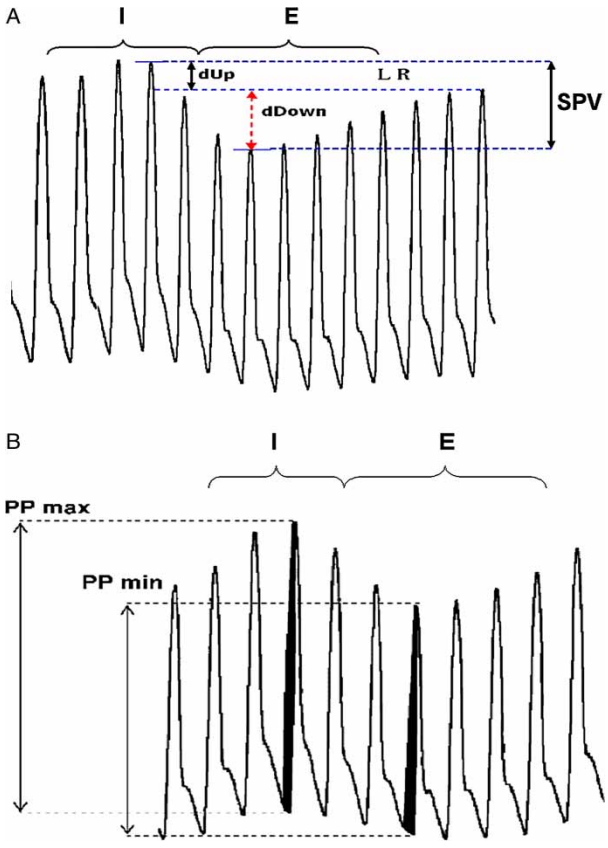

Fig. 4. Simultaneous recording in a mechanically ventilated patient of (A) SAP and (B) PP with airway pressure. In the presence of fluid responsiveness there is an inspiratory increase and an expiratory decrease in SAP. The SPV is the difference between the maximal and minimal SAP. An end-expiratory pause of a few seconds could identify a cyclic increase (dUp) and decrease (dDown) from a reference line (LR) in SAP during tidal ventilation. In the presence of fluid responsiveness, the arterial PP increases during inspiration and decreases during expiration. I = inspiration; E = expiration; PP min = minimal PP; PP max = maximal PP.

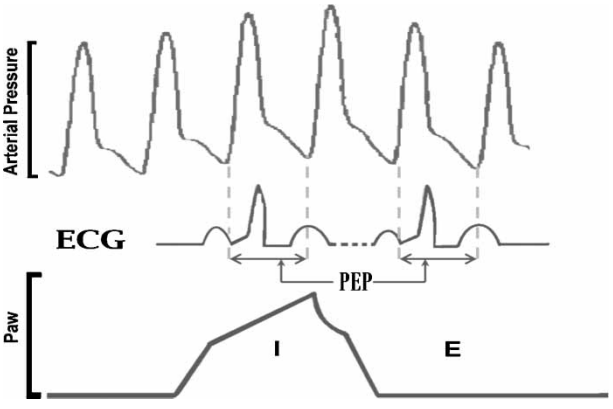

Fig. 5. Respiratory changes in the PEP defined as the time interval between the Q wave in the electrocardiogram and the upstroke of the arterial pressure. In the presence of fluid responsiveness there is an inspiratory (I) decrease and expiratory (E) increase in PEP. Paw = airway pressure.

ter, it is possible to record the area of the systolic part of the arterial pressure curve and therefore to monitor SV, provided that the system uses an accurate factor of the proportionality between SV and the specific curve area. It has been demonstrated (30) that respiratory SV variation (SVV) assessed by means of commercially available systems can predict fluid responsiveness in patients receiving mechanical ventilation.

Respiratory changes in pre-ejection period. This is a new method to predict fluid responsiveness. The pre-ejection period (PEP) is the time from the onset of ventricular depolarization to the beginning of left ventricular ejection (the time interval between the Q wave on the electrocardiogram and the upstroke of the radial arterial pressure) which allows assessment of ventricular function. Even if PEP depends slightly on afterload and cardiac contractility, it still depends predominantly on the change in ventricular preload. In post-coronary artery bypass surgery patients under mechanical ventilation, Bendjelid et al. (39) demonstrated that the respiratory variation in PEP (ΔPEP) is directly related to the respiratory change in arterial pressure (Fig. 5) and to the increase in SV induced by fluid challenge.

Non-invasive indices

Pulse oximetry plethysmographic signal variation. As the pulse oximetry plethysmographic signal is apparently similar to the peripheral arterial pressure waveform, Cannesson et al. (40) used pulse oximetry to assess preload responsiveness in mechanically ventilated patients. They found a good correlation between respiratory variation in the amplitude of the pulse wave calculated from the pulse oximetry plethysmographic waveform and the respiratory variation in arterial pulse pressure recorded with an arterial catheter. These findings were confirmed in two recent studies (41,42). However, additional studies are mandatory before oximetry waveform variation can be recommended as a guide to fluid therapy in mechanically ventilated patients in ICUs and operating rooms.

Plethysmographic PEP variation. It was demonstrated (43) that changes in PEP as measured using both tracing of invasive pressure waves and non-invasive pulse plethysmographic waveforms are as accurate as invasive pulse pressure changes in the prediction of fluid responsiveness in mechanically ventilated septic patients.

Respiratory changes in aortic blood flow. Transesophageal echocardiography allows beat-to-beat measurement of the peak aortic blood velocity and flow. The respiratory change in aortic blood velocity (ΔVpeak) was calculated as the difference between the maximal and minimal peak aortic blood velocities over a single respiratory cycle divided by the mean of the two values and expressed as a percentage. A threshold value of 12% allowed discrimination between responders and non-responders in septic shock patients (17). Recently, it has been demonstrated (31,32) that respiratory changes in the descending aortic blood flow (ABF) assessed by means of esophageal Doppler provide a simple and accurate prediction of fluid responsiveness.

Respiratory changes in vena cava diameter. The inferior vena cava (IVC) can be easily visualized in mechanically ventilated patients by transthoracic echocardiography using a subxiphoid approach (Fig. 6i). The distensibility index of the IVC (dIVC) calculated as the ratio of

Dmax (IVC diameter at end inspiration)–

Dmin (IVC diameter at end expiration)/Dmin

and expressed as a percentage was proposed to detect fluid responsiveness. It has been shown in two studies (33,34) that dIVC of 12% and 18%, respectively were accurate cut-off values for predicting fluid responsiveness in septic patients.

While the IVC is distended by means of mechanical insufflation, the superior vena cava (SVC) collapses. Using a transesophageal approach, a superior vena caval collapsibility index (SVCI) reflecting the variation in SVC diameter under mechanical ventilation was used to assess fluid responsiveness. It is calculated as follows (Fig. 6ii):

(maximum diameter on expiration–

minimum diameter on inspiration)/

maximum diameter on expiration

A threshold value > 36% allowed discrimination between responders and non-responders with a sensitivity of 90% and a specificity of 100% (35).

Passive leg raising. Passive leg raising is a manoeuvre by means of which it is possible to abruptly increase preload by transferring blood (≈300 ml) from the legs to the intrathoracic compartment. In passive leg raising, the lower limbs are lifted in a straight manner to an angle of 45°. It has been demonstrated (44) that an increase in arterial pulse pressure of 10% predicts a positive response to fluid administration.

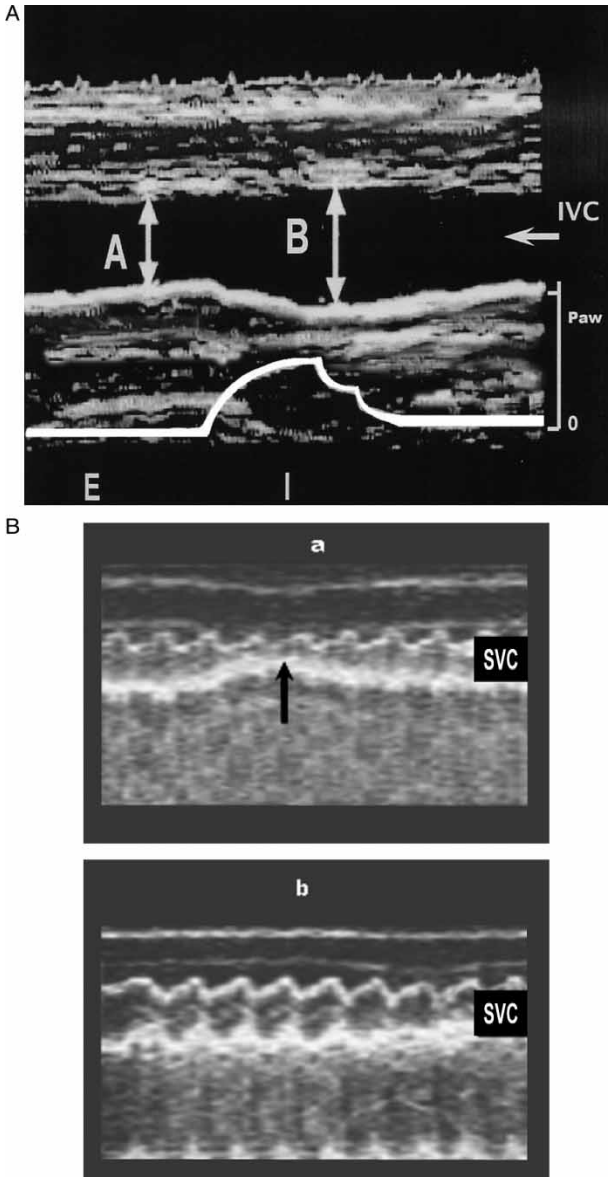

Fig. 6. (i) Echocardiographic view showing the respiratory changes in IVC diameter with (B) inspiratory enlargement and (A) expiratory return to baseline level. E = expiration; I = inspiration; Paw = airway pressure. (ii) Echographic view showing the respiratory changes in the SVC diameter in one illustrative fluid responder patient under mechanical ventilation. Before volume expansion (a), the SVC diameter decreased (*arrow*) at inspiration while it did not change significantly between inspiration and expiration after volume expansion (b).

Limitations

Use of the respiratory variations in SV or surrogates to detect preload reserve may result in a misleading interpretation in some clinical situations. In this

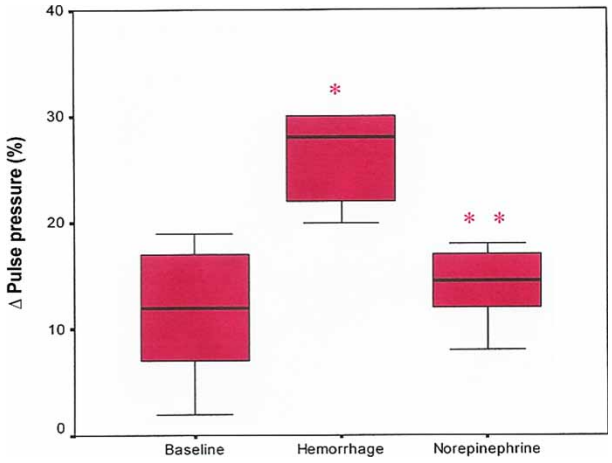

Fig. 7. Box plots showing changes from baseline in PP following haemorrhage and norepinephrine treatment. The line in each box indicates the median. The upper and lower limits of each box indicate the 75th and 25th percentiles, respectively. The error bars above and below each box represent the 90th and 10th percentiles, respectively. ΔPulse pressure indicates the respiratory variation in PP. $^{*}p < 0.05$ vs baseline; $^{**}p < 0.05$ vs haemorrhage.

regard, it should be highlighted that these parameters were validated in mechanically ventilated patients who were deeply sedated and did not make any spontaneous respiratory effort. When assessed in patients with spontaneous respiratory movements (under pressure support or breathing through a face mask) ΔPP failed to predict the response to volume expansion (45). Magder and coworkers (46,47) demonstrated the usefulness of respiratory variation in right atrial pressure (RAP) in patients without respiratory assistance. They showed that an inspiratory decrease in RAP of $\geq$ 1 mmHg predicted a positive response to volume expansion in spontaneously breathing patients. However, these findings were not confirmed. In addition, the use of respiratory variation in SV is influenced by catecholamines (Fig. 7) and by the tidal volume delivered by the ventilator. Indeed, noradrenaline was shown to underestimate ΔPP and SPV through the shift of blood from unstressed to stressed vascular bed (48). Increasing tidal volume should increase SVV but should also decrease venous return and cardiac preload (49). However, by inducing a small change in intrathoracic pressure, a low tidal volume may be theoretically associated with a low ΔPP and SVV. De Backer et al. (50) recently reported that ΔPP could not predict fluid responsiveness in patients with a tidal volume of <8 ml/kg. Finally, dynamic indices cannot be used in patients with arrhythmias.

Table I. Threshold values of static indices of cardiac preload.

Index	Value
CVP (mmHg)	≤ 8
Ppao (mmHg)	≤ 12
RVEDVI (ml/m^2)	≤ 90
LVEDAI (cm^2/m^2)	≤ 5
GEDV (ml/m^2)	≤ 600

Table II. Threshold values of dynamic indices of fluid responsiveness.

Index	Value
ΔRAP (mmHg)	≥ 1
SPV (mmHg)	≥ 10
dDown (mmHg)	≥ 5
ΔPP (%)	≥ 13
SVV (%)	≥ 10
ΔVpeak (%)	≥ 12
ΔABF (%)	≥ 18
IVC distensibility (%)	≥ 12–18
SVC collapsibility (%)	≥ 36
ΔPEP (%)	≥ 4
PLS test (%)	≥ 10

PLS test = passive leg raising test based on variation in PP.

Conclusions

In critically ill patients requiring mechanical ventilation, static indices of cardiac preload such as CVP and Ppao are of limited value for predicting fluid responsiveness because they do not correlate with fluid expansion-induced changes in SV. Conversely, dynamic indices were found to be good predictors of the hemodynamic response to fluid challenge. Although the best dynamic index probably remains to be determined, ΔPP seems to predict volume responsiveness better than other indices (51). In the future, non-invasive measures of ΔPP, PEP and changes in vena cava diameter could be valuable methods for assessing fluid responsiveness which will be particularly attractive for use in routine clinical practice. Threshold values for the most widely available indices are summarized in Tables I and II. It should be highlighted that these indices should only be used in patients who are under mechanical ventilation and deeply sedated. In patients who have either partial or complete spontaneous ventilatory activity, only static indices can be used and special emphasis is placed on new volumetric indices, in particular GEDV.

Acknowledgements

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