Predicting fluid responsiveness in the intensive care unit: a clinical guide

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Abstract - Fluid administration in critically ill patients is an important everyday therapeutic measure to improve organ perfusion. However, during the past decade, excessive fluid administration has been related to increased morbidity and mortality. This has led to the hypotheses that fluid administration without increasing cardiac output is inappropriate and is of no benefit to the patient. Over the past 10 years, many parameters for the prediction of fluid responsiveness have been suggested and validated. Implementation of these parameters in clinical practice may reduce the amount of inappropriate fluid. In this paper we discuss these methods for predicting fluid responsiveness and present a clinical strategy for fluid resuscitation. We make separate recommendations for patients on controlled mechanical ventilation, on mechanical ventilation with spontaneous activity and those breathing spontaneously.

Keywords - Fluid responsiveness, cardiac output, volume therapy, intensive care.

Introduction
Fluid resuscitation is one of the cornerstones to improve organ perfusion in patients with a critically compromised circulation. By increasing cardiac preload, fluid administration may increase cardiac output. When cardiac output increases as a result of fluid administration, the patient is considered to be fluid responsive. Excessive fluid resuscitation is associated with increased morbidity and mortality. In the presence of pulmonary oedema inappropriate fluid gain is associated with a worsened outcome [1,2]. The ARDS Network showed that conservative fluid management in patients with acute lung injury significantly shortened the duration of mechanical ventilation and of intensive care treatment [3].

In the past, optimal endpoints of fluid resuscitation have often relied on static indices such as blood pressure, central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) [4]. However, nowadays the validity of static indices as a guide for fluid resuscitation is being questioned. Osman et al. [5] showed that a CVP <8 mmHg and a PCWP <12 mmHg predicted fluid responsiveness with a positive predictive value of only 47% and 54 %, respectively. More recently, Marik et al [6] showed that the pooled correlation coefficient from 24 studies, between baseline CVP and a change in cardiac index was 0.18 (95% CI, 0.08-0.28) with an area under the ROC curve of 0.56 (95% CI, 0.51-0.61).

In the past 10 years many parameters have been proposed to predict and monitor fluid responsiveness. The accuracy of these methods has been established by their ability to predict an increase in cardiac index >15%. The purpose of this paper is to discuss these parameters for predicting fluid responsiveness in patients on controlled mechanical ventilation, mechanical ventilation with spontaneous activity and spontaneously breathing.

Predicting fluid responsiveness in patients on controlled mechanical ventilation
Positive pressure ventilation causes an intermittent change in preload of the heart. During inspiration the venous return to the right heart decreases thereby lowering preload and, in seconds is followed by a decrease in preload of the left heart. According to the Frank-Starling relationship, a decrease in preload results in a reduction of stroke volume. The magnitude of this effect depends on where the heart is operating on the Frank-Starling curve (Figure 1). If the heart is operating on the steep part of the curve this results in a significant change in stroke volume. If the heart moves higher up the curve, the change in stroke volume decreases. On the flat part of the curve stroke volume changes are minimal or absent. This heart-lung interaction during mechanical ventilation is the basis of the dynamic indices to predict fluid responsiveness such as stroke volume variation (SVV), and derivatives and the echocardiographic measurement of the caval vein collapsibility and distensibility.

Measurement of dynamic indices
Mechanical ventilation causes cyclic changes of left ventricular stroke volume and thereby cyclic changes of systolic pressure and pulse pressure. The increase in pleural pressure engendered by a mechanical breath causes a modest rise in arterial pressure (dUp), followed by a steady decrease (dDown). To measure dDown and dUp an end-expiratory hold must be performed to establish a baseline (Figure 2).

The augmentation of the arterial pressure at the onset of a me-
Mechanical breath has been explained by a temporary increase in left ventricular preload. The alveolar pressure squeezes the blood in the pulmonary capillaries towards the left atrium [7]. At the same time the transmural pressure of the left ventricle decreases due to an increase in pleural pressure effectively lowering afterload. A prominent dUp has been linked to an increase in afterload of the left ventricle and left ventricular failure [8,9]. In these situations temporary lowering of the afterload of the left ventricle may have a pronounced effect on cardiac output.

In 1987 Perel et al [10] showed in an animal model that dDown is closely related to graded haemorrhage and retransfusion. Tavernier et al [11] conducted the first clinical study in 15 patients with sepsis. This study showed that dDown and systolic pressure variation (SPV) were far better predictors of fluid responsiveness as compared to PCWP and echocardiographic left ventricular end diastolic area index. Today a substantial number of studies have confirmed these initial results in a variety of patient groups (Table 1).

SVV due to mechanical ventilation is the principal physiological explanation that predicts fluid responsiveness, while SPV and pulse pressure variation (PPV), are derivatives of SVV. Most studies use the arterial pressure reading, but the variation in the amplitude of the plethysmographic pulse (ΔPplet), analogue to the arterial pressure reading, can also be used with comparable results [23]. Monnet et al [18] used oesophageal Doppler to trace stroke volume variation.

### Table 1. Dynamic indices validated in patients on controlled mechanical ventilation.

<table>
<thead>
<tr>
<th>AUTHOR (YEAR)</th>
<th>PATIENTS</th>
<th>METHOD</th>
<th>RESPONDERS DEFINED AS</th>
<th>THRESHOLD VALUE FROM ROC</th>
<th>AUROC (95% CI)</th>
<th>SENS./SPEC. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tavernier (11)</td>
<td>15 septic</td>
<td>SPV art.</td>
<td>SVI&gt;15%</td>
<td>10mmHg</td>
<td>0.91(0.76-0.98)</td>
<td>na/na</td>
</tr>
<tr>
<td></td>
<td></td>
<td>DDown art.</td>
<td>SVI&gt;15%</td>
<td>5mmHg</td>
<td>0.94(0.81-0.99)</td>
<td>na/na</td>
</tr>
<tr>
<td>Michard (12)</td>
<td>40 septic</td>
<td>PPV art.</td>
<td>CI&gt;15%</td>
<td>9.5%</td>
<td>0.91(0.04)*</td>
<td>94/96</td>
</tr>
<tr>
<td>Kramer (13)</td>
<td>32 after CABG</td>
<td>PPV art.</td>
<td>CI&gt;12%</td>
<td>11%</td>
<td>0.99(0.96-1.0)</td>
<td>91/100</td>
</tr>
<tr>
<td>Reuter (14)</td>
<td>15 LVEF &gt;50%</td>
<td>SV PICCO</td>
<td>SV&gt;5%</td>
<td>9.5%</td>
<td>0.88(0.77-0.99)</td>
<td>79/85</td>
</tr>
<tr>
<td></td>
<td>15 LVEF&lt;35%</td>
<td>SV PICCO</td>
<td>SV&gt;5%</td>
<td>9.5%</td>
<td>0.76(0.59-0.96)</td>
<td>71/80</td>
</tr>
<tr>
<td>De Backer (15)</td>
<td>27 critically ill</td>
<td>PPV art.</td>
<td>CI&gt;15%</td>
<td>12%</td>
<td>0.89(0.07)#</td>
<td>88/89</td>
</tr>
<tr>
<td>Höfer (16)</td>
<td>40 off-pump</td>
<td>PPV art.</td>
<td>SV&gt;25%</td>
<td>13.5%</td>
<td>0.81(0.67-0.95)</td>
<td>72/72</td>
</tr>
<tr>
<td></td>
<td>CABG</td>
<td>SV PICCO</td>
<td>SV&gt;25%</td>
<td>12.5%</td>
<td>0.82(0.68-0.97)</td>
<td>74/71</td>
</tr>
<tr>
<td>Preisman (17)</td>
<td>18 CABG</td>
<td>dDown art.</td>
<td>SV&gt;15%</td>
<td>5mmHg</td>
<td>0.92(0.85-1.0)</td>
<td>86/86</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Art. PPV</td>
<td>SV&gt;15%</td>
<td>11.5%</td>
<td>0.95(0.89-1.0)</td>
<td>86/89</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SV PICCO</td>
<td>SV&gt;15%</td>
<td>9.4%</td>
<td>0.96(0.92-1.0)</td>
<td>93/89</td>
</tr>
<tr>
<td>Monnet (18)</td>
<td>38 critically ill</td>
<td>ABFV</td>
<td>ABF&gt;15%</td>
<td>16%</td>
<td>0.93(0.04) #</td>
<td>90/94</td>
</tr>
<tr>
<td>Solus-</td>
<td>8 major hepatic</td>
<td>PPV Finapress</td>
<td>SVI&gt;10%</td>
<td>14.0%</td>
<td>0.81(0.70-0.93)</td>
<td>na/na</td>
</tr>
<tr>
<td>Biguenet (19)</td>
<td>surgery</td>
<td>PPV art.</td>
<td>SV&gt;10%</td>
<td>12.5%</td>
<td>0.79(0.67-0.92)</td>
<td>na/na</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PPV pleth.</td>
<td>SV&gt;10%</td>
<td>9.5%</td>
<td>0.68(0.54-0.82)</td>
<td>na/na</td>
</tr>
<tr>
<td>Charron (20)</td>
<td>21 critically ill</td>
<td>PPV art.</td>
<td>CI&gt;15%</td>
<td>10.0%</td>
<td>0.96(0.86-1.0)</td>
<td>89/83</td>
</tr>
<tr>
<td></td>
<td></td>
<td>VTIao ED</td>
<td>CI&gt;15%</td>
<td>20.4%</td>
<td>0.87(0.69-1.0)</td>
<td>78/92</td>
</tr>
<tr>
<td>Natalini (21)</td>
<td>22 critically ill</td>
<td>PPV art.</td>
<td>CI&gt;15%</td>
<td>15%</td>
<td>0.74(na)</td>
<td>na/na</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PPV pleth.</td>
<td>CI&gt;15%</td>
<td>15%</td>
<td>0.72(na)</td>
<td>na/na</td>
</tr>
<tr>
<td>Lafanechère</td>
<td>21 critically ill</td>
<td>PPV art.</td>
<td>ABF&gt;15%</td>
<td>12%</td>
<td>0.78(0.12)#</td>
<td>70/92</td>
</tr>
<tr>
<td>Feissel (23)</td>
<td>23 septic</td>
<td>PPV art.</td>
<td>CO&gt;15%</td>
<td>13%</td>
<td>0.99(0.98-1.0)</td>
<td>100/70</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PPV pleth.</td>
<td>CO&gt;15%</td>
<td>12%</td>
<td>0.96(0.85-1.0)</td>
<td>94/80</td>
</tr>
<tr>
<td>Cannesson (24)</td>
<td>25 pre-CABG</td>
<td>PPV art.</td>
<td>CI&gt;15%</td>
<td>11%</td>
<td>0.85(0.08)*</td>
<td>80/90</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PPV pleth.</td>
<td>CI&gt;15%</td>
<td>13%</td>
<td>0.85(0.08)*</td>
<td>93/90</td>
</tr>
<tr>
<td>Huang (25)</td>
<td>22 severe ARDS</td>
<td>PPV</td>
<td>CI&gt;15%</td>
<td>11.8%</td>
<td>0.77(na)</td>
<td>68/100</td>
</tr>
</tbody>
</table>

ROC = Receiver Operating Characteristic Curve, AUROC = Area Under the Receiver Operating Characteristic Curve, CI= Confidence interval, Sen. = Sensitivity, Spec. = Specificity, art = measured from arterial pressure registration, pleth. = measured from pulse oxymetry plethysmographic curve, SPV = Systolic Pressure Variation, PPV = Pulse Pressure Variation, SVI = Stroke Volume Index, CI = Cardiac Index, CO = Cardiac Output, CABG = Coronary Artery Bypass Graft, SE = Standard Error, SV = Stroke volume, ED= esophageal Doppler, * standard error, # standard deviation, ABF = Abdominal Aortic blood Flow (Esophageal Doppler), ABFV = Abdominal Aortic blood Flow variation (Esophageal Doppler), na = not available.
For adequate interpretation of SVV, PPV and SPV it is important to note that they require a regular heart rhythm and that they are influenced by tidal volume. De Backer et al [15] showed that PPV is only a reliable predictor of fluid responsiveness when a tidal volume equal or greater than 8ml/kg is used. When a tidal volume < 8ml/kg was used sensitivity and specificity for prediction of fluid responsiveness dropped from 86% to 39% and from 89% to 65%, respectively. Lower tidal volumes may insignificantly affect pleural pressure and loading conditions of the left ventricle. Recently, however, Huang et al [25] used a low tidal volume strategy (6.4±0.7ml/kg) with high PEEP (13.9±1.4 cm H2O), in 22 patients with severe ARDS and showed that a PPV > 11.8% predicted a positive response to volume expansion with a sensitivity of 68% and a specificity of 100%. In the accompanying editorial,Michard et al [27] argued that PEEP induces an increase in mean airway pressure and pleural pressure causing a leftward shift on the Frank-Starling curve. Therefore, a patient operating on the flat part of the curve may move to the steep part and become fluid responsive. The relatively low sensitivity means that about one-third of patients who may benefit from a fluid challenge, are predicted not to. In an experimental animal model Kim et al [28] measured PPV at tidal volumes of 5, 10, 15 and 20 ml/kg. PPV tended to increase with higher tidal volumes. Only a tidal volume of 20 ml/kg differed significantly (p<0.05) from the baseline tidal volume (10 ml/kg). From this study it was concluded that separate validation is required to define threshold pulse pressure. However, in clinical practice tidal volumes of 6-10 ml/kg are used. The threshold values for SVV and PPV for tidal volumes of 8-10ml/kg have been validated (Table 1). For lower tidal volumes sensitivity will rapidly decrease [15,25], but specificity may remain high [25].

Another dynamic method that is used to predict fluid responsiveness in ventilated patients is the measurement of the endoluminal diameter change of the caval vein with echography. Mechanical ventilation causes fluctuations in blood flow to the right heart. This results in a cyclic change in the endoluminal diameter of the compliant inferior caval vein (ICV) and superior caval vein (SCV). The diameter of the ICV can be measured with trans-thoracic echography using the sub-xiphoidal long axis view, and from the minimum (Dmin) and maximum (Dmax) diameters a collapsibility or distensibility index can be calculated. Feissel et al. [29] studied 39 patients with septic shock on controlled mechanical ventilation and showed that a distensibility index of >12% allowed identification of responders to a fluid challenge with a positive and negative predictive value of 93% and 92%, respectively. The index was calculated as the difference between Dmax and Dmin, normalized by the mean of the two values, and expressed as a percentage. Barbier et al [30] used a slightly different calculation for the distensibility index (ratio of Dmax –Dmin / Dmin expressed as a percentage). In 23 septic, mechanically-ventilated patients a threshold of 18% discriminated responders (increase in CI ≥15%) from non-responders with a sensitivity of 90% and a specificity of 90%.

In only one study was the diameter of the SCV measured [31]. Measurement of the diameter of the SCV requires transoesophageal echocardiography (long axis view). In 66 septic, mechanically-ventilated patients a collapsibility (Dmax-Dmin/Dmax expressed as a percentage) of 36% allowed discrimination between responders and non-responders with a sensitivity of 90% and a specificity of 100%.

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**Figure 1.** Preload change (ΔPreload) is identical for situation A and B, but stroke volume change (ΔSV) decreases moving up the Frank-Starling curve (A→B).

**Figure 2.** Systolic pressure variation (SPV) after an end-expiratory hold in a patient on pressure controlled ventilation.

SPV can be divided in dUup and dDown after an end-expiratory hold. For pulse pressure variation and stroke volume variation the maximal and minimal values in one breathing cycle are used from the arterial blood pressure tracing. ABP = arterial blood pressure, AWP = airway pressure.
Since this method also depends on the interaction between mechanical ventilation and venous return it is likely to be influenced by the size of the tidal volume. In our experience, the cyclic fluctuation of the caval vein diameter indeed decreases when tidal volume is lowered. Appropriate training is needed for accurate measurement, although the diameter of the inferior caval vein is quite easy to determine. It is also unclear whether this method can be used in patients with an irregular heart rate as the flow in the caval vein is non-pulsatile. None of the current studies have specified this issue.

Predicting fluid responsiveness in patients with spontaneous breathing with or without mechanical support

Until recently it was assumed that the dynamic indices were less useful for predicting fluid responsiveness in patients with spontaneous breathing activity because breathing frequency, tidal volume and the intrathoracic pressure are not controlled. However, spontaneous breathing also results in stroke volume variation. During expiration, preload of the right ventricle is lowered and during inspiration it increases, contrary to mechanical ventilation. Apart from these dynamic indices, an endogenous fluid challenge, the passive leg raising test, has been proposed.

Figure 3. Clinical flow diagram for prediction of fluid responsiveness.

FC = fluid challenge, CI = Cardiac Index, PLR = Passive Leg Raising, CV = Controlled Ventilation, TV = Tidal Volume, PPV = Pulse Pressure Variation, SVV = Stroke Volume Variation.
as a predictive test for fluid responsiveness [32] for intubated patients as well as patients breathing spontaneously.

Measurement of dynamic indices
Soubrier et al [33] evaluated PPV in unstable patients breathing spontaneously. Thirty-two patients received a fluid challenge of 500 ml (6% hydroxyethyl starch). A PPV of ≥12% resulted in a sensitivity of 63% and a specificity of 92%. The low sensitivity can be explained by insufficient changes in pleural pressure when breathing spontaneously, as has also been shown in mechanically-ventilated patients with low tidal volumes [15]. The high specificity, however, was a remarkable finding. This implies that when a PPV ≥12% is present in a patient breathing spontaneously, a response to fluid is likely. These results, however, are in contrast to the findings of Heenen et al [34]. In 12 patients, breathing spontaneously through a face mask with oxygen, PPV had an area under the ROC curve of 0.29±0.17 for prediction of fluid responsiveness. The use of PPV in spontaneously breathing patients therefore is still questionable [35].

Perner et al. [36] studied SVV, measured with the PiCCO system, in 30 patients with septic shock ventilated in the pressure-support mode. A fluid challenge of 500 ml of colloid was given. Responders were defined as having an increase of >10% in the cardiac index. SVV did not change significantly before and after the fluid challenge (13±5% vs. 16±6%, p=0.26). Mean area under the ROC curve was 0.52 (95% CI, 0.39-0.73). It was concluded that SVV does not predict the response to a fluid challenge in patients on pressure support. Similar results were found by Heenen et al [34] in mixed group of 9 critically ill patients on pressure support with an area under the ROC curve of 0.64±0.26.

Magder et al [37] raised the hypothesis that right atrial pressure does not decrease during voluntary inspiration if the heart is not volume responsive. Inspiration and expiration cause a variable preload to the right ventricle depending on where the heart is operating on the Starling curve. This concept was tested in 33 patients after cardiopulmonary surgery. Twelve patients were breathing spontaneously and 21 were breathing in an assist mode. All patients received fluid loading in order to increase CVP more than 2 mmHg. In only 1 out of 14 patients with an absent respiratory response on right atrial pressure did cardiac output increase more than 250ml/min. In the group with a positive respiratory response on right atrial pressure (decrease in CVP≥1 mmHg during inspiration), fluid loading resulted in an increase in cardiac output of more than 250ml/h in 16 out of 19 patients. Comparable results were found in an additional study by Magder et al [38]. Heenen et al [34] studied this concept in 9 critically ill patients on pressure support and 12 patients breathing spontaneously. The predictive value to identify responders to fluid was poor, with an area under the ROC curve of 0.53±0.13 (mean±SD). No separate analysis was made for patients on pressure support or those breathing spontaneously.

Passive leg raising test
Raising the legs to 45° for 4 minutes results in a transient increase in venous return [39]. Using radiolabelled erythrocytes, it was shown that the infused volume of blood from the legs is approximately 150 ml [40]. Besides raising the legs, the trunk of the patient can be positioned horizontally to maximize the effect of the endogenous volume challenge [41]. The amount of the endogenous fluid challenge will be vary between patients and strongly depends on vasomotor tone. In a hypovolaemic, vasoconstricted patient less volume will be recruited than in a vasodilated patient in septic shock. Theoretically the PLR test might be false negative in severely vasoconstricted patients. However, most of these clinical situations are straightforward, e.g. severe hypovolaemia.

According to the Starling principle, a PLR test increases CI immediately when the heart is on the steep portion of the curve. Various studies have shown that a PLR test is able to increase CI and that CI returns to baseline when lowering the legs [41]. Therefore the PLR test can be regarded as a completely reversible, endogenous volume challenge. The haemodynamic changes occur within seconds and are maximal approximately 1 minute after starting the manoeuvre [42].

Boulain et al [32] showed in fully sedated, mechanically-ventilated patients that changes in stroke volume induced by passive leg raising (PLR), and infusion of 300 ml gelatin were strongly correlated (r = 0.89, p<0.001). Monnet et al [42] found that the PLR predicted fluid responsiveness with a sensitivity of 97% and a specificity of 94% in 71 mechanically ventilated patients, of whom 31 had spontaneous breathing activity and/or arrhythmias. Lafanèchere et al [22] conducted a similar study in 22 fully sedated and mechanically-ventilated, critically-ill patients. The PLR test had a sensitivity of 90% and a specificity of 83% to predict an increase in aortic blood flow of 15%. Galas et al. [43] found a sensitivity of 95% and a specificity of 94% for the PLR to predict fluid responsiveness in 44 patients on controlled mechanical ventilation after cardiac surgery. Fourteen patients were included with an irregular heart rate.

Lamia et al [44] conducted a study in 14 patients on assisted mechanical ventilation and 10 patients breathing spontaneously. The PLR test had a sensitivity of 77% and a specificity of 100% for predicting fluid responsiveness. There was no difference between intubated and non-intubated patients. In this study transthoracic echocardiography was used to measure stroke volume. Other echocardiographic measures, such as E/Ea and left ventricular end-diastolic area, were not useful for predicting fluid responsiveness.

As shown by these studies, the PLR test can be used in ventilated patients and in patients breathing spontaneously, and is independent of cardiac arrhythmias. However, the PLR test has several limitations. This method requires the continuous measurement of changes in cardiac output. Transesophageal Doppler was used in the study of Monnet et al [42] Lamia et al [44] used transthoracic echocardiography. Today there are numerous methods for rapid and valid measurement of cardiac output [45]. Changes in blood pressure are not sufficient to evaluate the effect of a PLR test [42,44]. In some patients the PLR test leads to considerable discomfort or is not possible, e.g. in trauma patients.
Clinical algorithm

Nowadays it is possible to predict fluid responsiveness in the majority of intensive care patients. However, different methods have to be used in different clinical situations. The method of choice is mainly directed by the limitations of a method and skills of the doctor. A clinical flow chart for choosing a method is shown in Figure 3.

After diagnosing inadequate organ perfusion, the first step is to determine the patient’s heart rhythm. An irregular heart rhythm excludes the use of dynamic indices such as SPV, PPV and SVV. The use of echocardiography to assess the collapsibility of the IVC or SCV has not been validated in cases of irregular heart rhythm. A PLR test is the most valid option.

If the patient is on controlled mechanical ventilation, has a regular heart rhythm and the tidal volume is ≥ 8ml/kg, we advise the use of SVV or PPV. These indices are easy to monitor and can be measured continuously. If SVV or PPV is > 12%, we advise fluid administration if clinical or biochemical signs of tissue hypoperfusion are present. Alternative measurements in this patient category are the ICV distensibility or SCV collapsibility indices. If tidal volume is <8ml/kg, sensitivity for prediction of fluid responsiveness using SVV and PPV rapidly declines. However, as Huang et al [25] have shown in severe ARDS patients, specificity may still be high. De Backer et al [15] found a specificity of only 65% for PPV in a mixed group of intensive care patients using a tidal volume <8 ml/kg. In our opinion, the use of SVV or PPV in case of a tidal volume <8ml/kg needs more validation to be clinically useful. Therefore we advise a PLR test in case of a tidal volume < 8 ml/kg. If a PLR test cannot be performed, a traditional fluid challenge must be done with a small, rapid bolus, e.g. 250 ml, with monitoring of CO. If cardiac index does not increase >15%, fluid loading should be stopped.

For patients on mechanical ventilation with spontaneous activity the only method validated in the literature is the PLR test. Further research is needed on dynamic indices in these patients. A fluid challenge with CO measurement should be performed if PLR is not possible.

In patients breathing spontaneously more evidence is needed to support the use of SVV or PPV. Although Soublier et al [33] showed that specificity still may be high, SVV or PPV can not yet be advised to predict fluid responsiveness in these patients. The same is true for the measurement of the inspiratory drop in CVP proposed by Magder et al [37,38]. Therefore, in this situation we advise a PLR test. Otherwise, a fluid challenge with CO measurement is indicated if a PLR test is not possible.

In conclusion, prediction of fluid responsiveness is possible in most critically ill patients and should be implemented in routine clinical practice. The suggested algorithm may prevent inappropriate fluid boluses in most critically ill patients. Future studies should address the question if a fluid management strategy based on prediction of fluid responsiveness results in an improvement in clinical outcome.

References

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