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Prediction of fluid responsiveness in critical care: Current evidence and future perspective

Luigi La Via [a,*], Francesco Vasile [a], Francesco Perna [a], Mateusz Zawadka [b]

[a] Department of Anesthesia and Intensive Care, Azienda Ospedaliera Universitaria Policlinico "G. Rodolico- San Marco", Via Santa Sofia, 78, 95123, Catania, Italy
[b] Second Department of Anesthesia and Intensive Care, Medical University of Warsaw, 1A Banacha Str., 02-097, Warsaw, Poland

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ABSTRACT

Background: Fluid responsiveness is a crucial concept in the management of critically ill patients, guiding fluid resuscitation to optimize hemodynamics while avoiding fluid overload. Various methods have been proposed to predict fluid responsiveness, but their applicability and accuracy vary.
Objectives: To provide a comprehensive review of the different methods used to assess fluid responsiveness in critically ill patients, including their principles, clinical applications, and limitations.
Methods: A narrative review of the literature was conducted, focusing on both static and dynamic indices of fluid responsiveness, as well as their advantages and disadvantages.
Results: Static indices, such as central venous pressure and pulmonary artery occlusion pressure, have limited accuracy in predicting fluid responsiveness. Dynamic indices, including stroke volume variation, pulse pressure variation, and respiratory changes in inferior vena cava diameter, have demonstrated better predictive value in mechanically ventilated patients. Passive leg raising and end-expiratory occlusion tests are useful in both spontaneously breathing and mechanically ventilated patients. Carotid flow time has been shown to predict fluid responsiveness in both mechanically ventilated and spontaneously breathing patients but may be influenced by factors such as arterial compliance and operator skill.
Conclusions: Dynamic indices are more accurate predictors of fluid responsiveness than static indices in critically ill patients. However, each method has its limitations, and a comprehensive understanding of the principles, clinical applications, and potential confounding factors is essential for optimal patient management. Individualized assessment and a multimodal approach should be considered in the evaluation of fluid responsiveness.

1. Introduction

Fluid resuscitation is generally considered a fundamental aspect in managing critically ill patients because it is intended to help optimize cardiac output and enhance tissue perfusion [1]. However, determining the appropriate volume of fluids to administer can be challenging, as both under- and over-resuscitation can result in significant morbidity and mortality [2–4]. Fluid responsiveness, defined as the ability of a patient's cardiac output to increase in response to a fluid bolus, has become an important concept in guiding fluid therapy [5], both in spontaneously breathing [6] and mechanically ventilated patients [7]. This narrative review will discuss the various methods to predict fluid responsiveness in critically ill patients and their clinical applications. Although fluid responsiveness is an important concept, it is debatable whether it should be solely used to manage fluid therapy in critically ill patients. Over the last year, new concepts have emerged assessing venous congestion [8–10].

2. Static indices of preload

Traditionally, static indices of preload, such as central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP), have been used to guide fluid therapy. However, several studies have demonstrated that the predictive value of these static indices is poor [11–13].

2.1. Central venous pressure

CVP is the pressure within the thoracic vena cava near the right

* Corresponding author. Department of Anesthesia and Intensive Care, Azienda Ospedaliera Universitaria Policlinico "G. Rodolico- San Marco", Via Santa Sofia, 78, 95123, Catania, Italy.
E-mail address: luigilavia7@gmail.com (L. La Via).

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atrium and is commonly used as an estimate of right atrial pressure [14] and an indicator of right ventricular preload. CVP has long been used as a hemodynamic parameter to assess intravascular volume status and guide fluid management in critically ill patients, as it is easy to measure and requires minimal apparatus [15,16]. Also, surveys regularly report that clinicians still continue to use CVP for predicting fluid responsiveness [17]. However, its predictive value has been questioned [18,19]. In fact, recent studies have demonstrated that CVP, as a static measure, may not reliably guide fluid administration in various clinical settings [13], and there is no association between CVP and circulating blood volume. Also, the variation of CVP (Delta CVP) has shown its inability to predict the hemodynamic response to a fluid challenge [19]. One potential reason for this is that CVP provides only a snapshot of the complex interplay between cardiac function, intravascular volume, and venous tone. However, some authors recently hypothesized that "extreme" CVP values might be more helpful to guide fluid administration than intermediate values. In fact, in a recent systematic review by Eskesen et al. [20] that evaluated the response to a fluid bolus and reported CVP in 1148 patients from 51 studies, the overall predictive value of CVP was poor. However, almost two thirds of the patients with a CVP <8 mmHg but only one third of patients with CVP values > 12 mmHg responded to fluids. In another study by Biais et al. [21] including 556 patients, the authors used the grey zone approach to determine CVP values between which no decisions on fluid responsiveness could be taken. A positive response to fluids was observed when CVP values were less than 6 mmHg but was unlikely when values were greater than 15 mmHg. These data might suggest that extreme CVP values can help to guide the response to fluids whereas intermediate values cannot [18].

2.2. Pulmonary capillary wedge pressure

Pulmonary capillary wedge pressure (PCWP), also known as pulmonary artery occlusion pressure, is measured by inflating a balloon-tipped catheter in a branch of the pulmonary artery. PCWP is a potentially useful index of left ventricular filling pressure and pulmonary vascular congestion [22]. Similarly to CVP, several studies found PCWP to be a poor predictor of fluid responsiveness in critically ill patients [11,12,23]. In fact, in these studies, CVP <8 mmHg and PCWP <12 mmHg failed to correlate significantly with changes in cardiac performance in response to fluid infusion. Furthermore, measuring PCWP is invasive and carries risks such as pulmonary artery rupture and catheter-related infections [24]. However, in a small study by Roy et al., the authors hypotisized that if the v wave of PCWP (representing the passive filling of the right atrium) is lower and smaller than the a wave (atrial contraction), so that the a/v ratio is > 1, then the heart could accommodate further volume and increase cardiac output in response to fluids. This study showed an association between the a/v wave ratio>1 of the PCWP tracing and fluid responsiveness, thus suggesting the possible value of PCWP waveform analysis [25]. Moreover, the a/v wave ratio of PCWP may be a marker of diastolic function. In fact, the v wave peaks during isovolumetric ventricular relaxation, the earliest part of diastole. Reduced diastolic function, and its consequent intolerance to fluids, may increase left atrial pressure during late systole and early diastole, increasing the v wave [25]. Large v waves from the PCWP tracing have been observed in the absence of mitral regurgitation in association with moderate-to-severe diastolic dysfunction [26].

3. Dynamic indices of fluid responsiveness derived from heart-lung interaction

Dynamic indices of fluid responsiveness, which incorporate the effects of respiratory changes on cardiac output or blood pressure, have emerged as more accurate predictors of fluid responsiveness in critically ill patients. These indices include pulse pressure variation (PPV) and stroke volume variation (SVV).

3.1. Pulse pressure variation and stroke volume variation

PPV is defined as the percentage change in pulse pressure (the difference between systolic and diastolic pressure) during a single respiratory cycle. It is calculated as:

$$PPV = [(\text{Pulse pressure\_max} - \text{Pulse pressure\_min}) / \text{Pulse pressure\_mean}] \times 100$$

PPV is currently the most studied marker of preload responsiveness [27,28]. It has been shown to be a reliable predictor in patients ventilated at low tidal volumes with regular cardiac rhythms [29]. The median threshold of the PPV is believed to be 12% (interquartile range 10–13%) with a pooled sensitivity of 88% with a specificity of 89% [28]. However, it was estimated that 24% of PPV values measured in practice remain between 9 and 13%, called the "grey zone", where the sensitivity or the specificity is lower than 90% [30]. Nevertheless, the concept of the grey zone analysis performed by Cannesson et al. implies that the farther PPV from the diagnostic threshold, the stronger the accuracy of the prediction of fluid responsiveness or unresponsiveness. During positive pressure ventilation, insufflation decreases preload of the right ventricle, which induces a decrease in preload of the left ventricle. If left ventricular stroke volume changes in response to cyclic positive pressure ventilation, this indicates that both ventricles are preload dependent. SVV is, in fact, the percentage change in stroke volume during a single respiratory cycle. It is calculated as:

$$SVV = [(\text{Stroke volume\_max} - \text{Stroke volume\_min}) / \text{Stroke volume\_mean}] \times 100\%$$

Like PPV, SVV has been shown to be a good predictor of fluid responsiveness in specific settings [31,32]. A SVV of greater than 10–12% is generally considered to predict preload with high sensitivity and specificity [33].

During the years, other dynamic indices have been studied as surrogates of PPV and SVV.

3.2. Other dynamic indices of preload

Systolic pressure variation (SPV) is defined as the difference between the maximum and minimum systolic pressure during a single respiratory cycle. It has been studied since 1987 in several animal models as an indicator of hypovolemia [34–36]. A following trial including patients with sepsis-induced hypotension showed a good performance of SPV in predicting fluid responsiveness, with an area under the ROC curve of 0.91 (95% confidence interval: 0.76 to 0.98) [37]. However, some authors suggested that the SPV does not reflect true changes in left ventricular stroke volume since it is caused by direct transmission of intrathoracic pressure to the aorta, similar to a Valsalva maneuver [38,39]. This may account for the SPV being somewhat less accurate and less used than the PPV or SVV [40].

Also, plethysmographic indices, obtained by continuous analysis of the raw pulse oximeter signal, have been studied as predictors of fluid responsiveness under general anaesthesia. In particular, the variation in pulse oximetry plethysmographic waveform amplitude and the Pleth Variability Index have shown to be equally effective for predicting fluid responsiveness in ventilated adult patients in sinus rhythm [41,42]. According to a recent meta-analysis, prediction seems more accurate when a large fluid bolus is administered [43].

In the last few years, research has been focusing on non-invasive techniques to obtain dynamic indices of preload. Those techniques are particularly useful when there is no need for arterial line placement, such as in operating theatre. Among them, the ClearSight system is an example of non-invasive hemodynamic monitoring being marketed by Edwards Lifesciences and using a finger-cuff-based pulse contour analysis [44]. This device is currently being implemented with the hypotension prediction index algorithm, which can predict episodes of

intraoperative hypotension with good accuracy [45,46].

3.3. Limitations of dynamic indices of preload

PPV, SVV and SPV should be considered unreliable in some specific conditions. Those include spontaneous breathing (even if the patient is intubated), open-chest surgery, cardiac arrhythmias [5], low intrathoracic pressures (<20 cm H_2O), high intra-abdominal pressure [47], low pulmonary compliance (<30 mL/cmH_2O) [48], right ventricular dysfunction [49] and Heart Rate/Respiratory Rate Ratio <3.6 [5]. Also, in the case of ARDS, the low tidal volume strategy reduces the amplitude of the change in intrathoracic pressure that causes the PPV and SVV [5]. In fact, a prospective study by Myatra et al. proved that an increase in tidal volume from 6 to 8 mL/kg resulted in an increase in the absolute value of PPV ≥3.5% or of SVV ≥2.5% [31]. Another condition associated with a low accuracy of PPV and SVV is intra-abdominal hypertension [50]. This eventuality may cause an increase in the threshold value used to identify fluid-responding patients, as respiratory variations of stroke volume are influenced both by volaemia and intra-abdominal pressure [51]. Despite these limitations, some recent studies have shown that the relative changes in PPV and SVV may help assess fluid responsiveness, even in cases where its absolute value is not interpretable. During tests that are usually performed by assessing changes in cardiac output, changes in PPV and SVV might be used as surrogates of cardiac output, allowing one to perform these tests with a simple arterial line and no hemodynamic monitor [52].

4. Echocardiographic methods to predict fluid responsiveness

Echocardiography has emerged as a valuable tool for predicting fluid responsiveness, as it allows for the non-invasive assessment of cardiac function and can be performed at the bedside. Echocardiographic methods to predict fluid responsiveness include the assessment of inferior vena cava (IVC) collapsibility, superior vena cava (SCV) collapsibility and respiratory variations in aortic blood flow velocity.

4.1. Inferior vena cava assessment

Modifications in intrathoracic pressure induced by spontaneous breathing or mechanical ventilation are reflected in the IVC diameters in close proximity to the heart when the central blood volume is low. The variation of the inferior vena cava diameter measured by transthoracic echocardiography in M-mode from the subcostal view has been reported to detect preload responsiveness (Fig. 1) [5]. In mechanically ventilated patients, fluid responsiveness can be assessed by the calculation of the IVC distensibility index, with 18% as the best cut-off [53]:

IVC distensibility index = [(IVC_diameter_max - IVC_diameter_min) / IVC_diameter_min] × 100

A recent meta-analysis of 20 studies on the accuracy of ultrasonographic measurements of IVC found a sensitivity and specificity of 0.71 (95% CI: 0.62–0.80) and 0.75 (95% CI: 0.64–0.85), respectively [54]. Fluid responsiveness can also be assessed in spontaneously breathing patients by transthoracic echocardiography, thus calculating the IVC collapsibility index:

IVC collapsibility index = [(IVC_diameter_max - IVC_diameter_min) / IVC_diameter_max] × 100

In this case, the optimal cut-off ranges between 40% and 48%, according to the different studies [55–57].

However, the predictive value of the IVC collapsibility index was found to be poor in spontaneously breathing patients [27,58], with a sensitivity of 0.63 (95% CI: 0.46–0.78) and specificity of 0.83 (95% CI: 0.76–0.87) [59].

The reliance on IVC respiratory variation has several limitations. In fact, IVC assessment is not reliable in cases of open chest surgery, low tidal volume, low lung compliance, left ventricular dysfunction and increased intra-abdominal pressure [60–62]. Nevertheless, the interest in this parameter is still high due to its relatively high feasibility in most critically-ill patients [63]. Also, IVC assessment with a standard subcostal approach is not always feasible in case of enlarged bowel, obesity, presence of chest drains or laparotomy wounds. Some recent studies investigated the role of an alternative approach represented by the assessment of the IVC with a *trans*-hepatic approach, with a latero-lateral visualization of the vessel in M-mode (Fig. 2) [64]. However, these studies showed that the subcostal and the *trans*-hepatic approaches to IVC are not interchangeable, both in mechanically ventilated [65] and spontaneously breathing patients [66].

4.2. Superior vena cava assessment

It has been described by Vieillard-Baron and colleagues that it is feasible to assess the superior vena cava collapsibility index (SVC-CI) to predict fluid responsiveness [67]. During the respiratory cycle under mechanical ventilation SVC collapses regularly with the increase in inthrathoracic pressure which is reversed to IVC changes. SVC can be visualized on transesophageal and on transthoracic echocardiography by experienced sonographers [68]. A variation in SVC >37–39% is predective of fluid responsiveness [69,70].The collapsibility index is

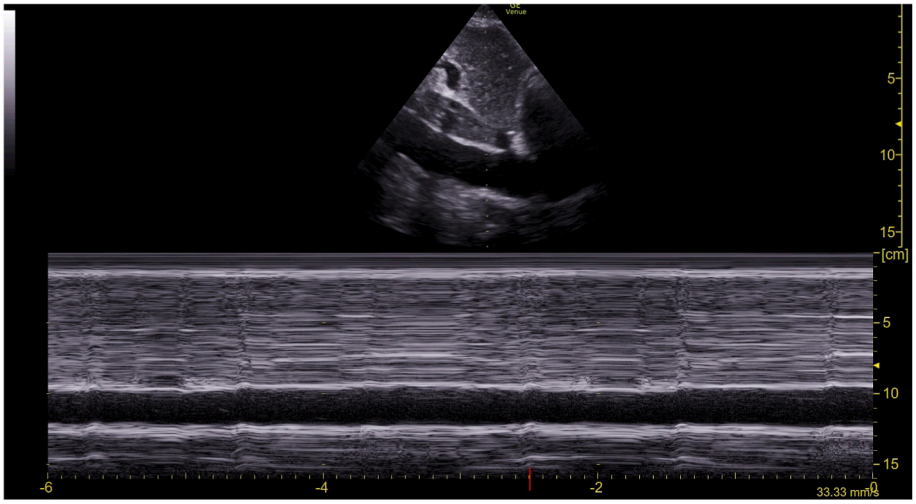

Fig. 1. Subcostal view of inferior vena cava for assessment of fluid responsiveness.

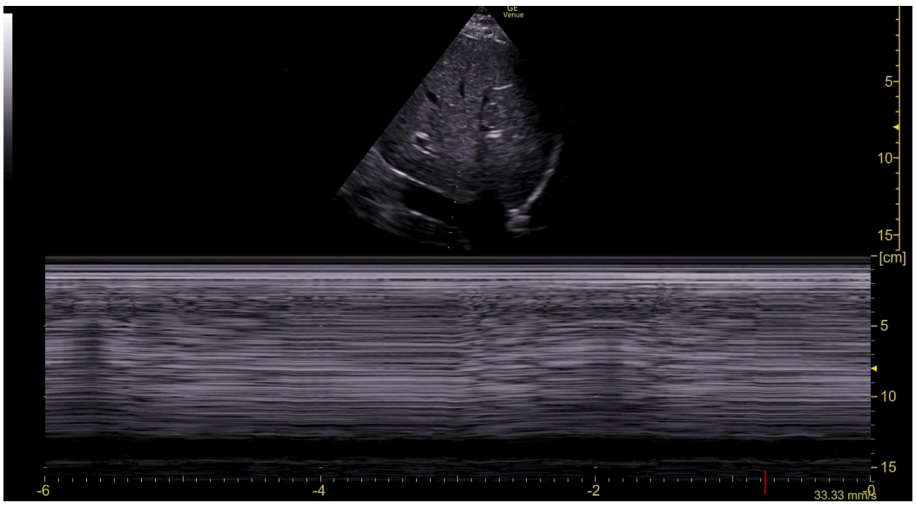

Fig. 2. Transhepatic view of inferior vena cava for assessment of fluid responsiveness.

calculated as:

SVC collapsibility index = [(SVC_diameter_max - SVC_diameter_min) / SVC_diameter_min] × 100

Recently transesophageal, monoplane echocardiographic detachable probe (hTEE) has been introduced in clinical practice. It is a 5.5-mm monoplane echocardiographic detachable probe, interfaces with a dedicated ultrasound system for uninterrupted 72-h monitoring of cardiac function and hemodynamic status. Offering direct visualization of three heart views (the *trans*-gastric short-axis, the mid-esophageal four-chamber view, and the mid-esophageal ascending aortic short-axis), this innovation marks a significant leap in echocardiography and hemodynamic monitoring [70]. However, hTEE has also some limitations, such as the required experience of the operator, and the fact that giving the superior resolution offered by hTEE, phenomena such as spontaneous contrast and ghosting are observed much more commonly than they are with transthoracic imaging [71].

4.3. Respiratory variations in aortic blood flow velocity

Transesophageal Doppler monitoring is a minimally invasive method for measuring blood flow in the descending thoracic aorta. Since a relatively fixed proportion of total flow travels down the thoracic aorta, descending aortic blood flow is considered a reliable estimate of cardiac output and its change [72]. It was recently proven that transesophageal Doppler enables the prediction of fluid responsiveness, either by assessing the hemodynamic effects of passive leg raising or by analyzing the respiratory variation of aortic blood flow [73]. The latter is obtained by Doppler measurements of descending aorta blood velocity and diameter. The percentage change in peak aortic blood flow velocity during a single respiratory cycle, also known as Delta Vpeak is calculated as:

Delta Vpeak = [(Vpeak_max - Vpeak_min) / Vpeak_mean] × 100

Feissel et al. [74] first described Delta VPeak measured by transesophageal Doppler as an accurate predictor of fluid responsiveness in adult patients with septic shock receiving mechanical ventilation. Before volume expansion, a Delta Vpeak threshold value of 12% allowed discrimination between responders and non-responders with a sensitivity of 1 and a specificity of 0.89. Another study by Monnet et al. [75] showed that a respiratory variation in Delta Vpeak before volume expansion predicted fluid responsiveness with a sensitivity of 90% and a specificity of 94%, with a cut-off of at least 18%. Similar to other dynamic indices, Delta Vpeak is less accurate in predicting fluid responsiveness in patients with spontaneous breathing efforts or arrhythmias. Furthermore, Delta Vpeak may be influenced by other factors such as the aortic compliance [72].

5. Dynamic tests to assess the response to fluids

Several tests have been studied as dynamic methods to assess fluid responsiveness. Those include the Passive leg raising (PLR), the mini-fluid challenge and the tidal volume challenge.

5.1. Passive leg raising (PLR)

PLR is a bedside maneuver that involves elevating the patient's legs to 30–45° while supine, which results in the transfer of roughly 300 mL of venous blood from the lower extremities to the central circulation. In this way, PLR increases cardiac preload, consequently inducing a significant rise of the mean systemic pressure [76]. In fluid responders, this rise is accompanied by an increase in venous return and thus in cardiac output. On the contrary, in fluid non-responders, the increase in the right atrial pressure balances the increase in mean systemic pressure, such that the pressure gradient of venous return (and cardiac output) does not change [76]. This maneuver has the advantage of being reversible [77], and it can be repeated as frequently as required without infusing a drop of fluid. Also, PLR has been proven to be accurate in spontaneously breathing patients and with cardiac arrhythmias, low tidal volume ventilation and low lung compliance [48,78,79]. A PLR-induced increase in cardiac output greater than 10% is generally considered to predict fluid responsiveness with high sensitivity and specificity (85 and 91%, respectively) [80]. In fact, PLR is recommended by the Surviving Sepsis Campaign for the hemodynamic management of septic shock patients [81]. However, the hemodynamic effects of PLR cannot be measured on simple invasive [80] or non-invasive arterial pressure monitoring [82], due to its scarce sensitivity. So, the effects of the PLR must be assessed by the continuous and real-time measurement of cardiac output. In fact, those effects reach their peak within 1 min, dropping rapidly thereafter, especially in patients with severe sepsis and capillary leak [83]. Also, PLR testing is contraindicated in patients with intracranial hypertension, and may result in some false negatives in case of intra-abdominal hypertension [84]. Several invasive and non-invasive techniques have been proposed to measure PLR-induced variations of cardiac output. Both the calibrated and uncalibrated pulse contour analysis techniques are very useful for this purpose [5]. Also, many studies showed the high reliability of measuring the changes in the velocity time integral of the left ventricular outflow tract by transthoracic echocardiography [85–89]. PLR-induced increase in

end-tidal carbon dioxide of more than 5% or 2 mmHg has been shown to be a non-invasive method to reflect changes in cardiac output in mechanically ventilated patients, thus providing an attractive alternative to previous methods [90,91]. In fact, since partial pressure of CO2 at the end of an exhaled breath is influenced by arterial PaCO2 and pulmonary blood flow, is could also be used as a marker of cardiac output [92]. Some authors also suggested the variation in the peak velocity of the carotid and femoral arteries as indicators of the response of cardiac output to PLR, with contradictory results [93,94]. More recent research is focusing on the role of arterial PPV [95], SVV [96], capillary refill time [97,98] and perfusion index [91,99] to monitor the effects of PLR, but the evidence is still insufficient to draw any conclusion.

5.2. Mini-fluid challenge

Administering a fluid challenge, with the infusion of 250–500 mL of fluids, and measure its effect on cardiac output, has long been used as a method to assess fluid responsiveness. However, this test cannot be considered as a "challenge", but as the treatment itself. Moreover, this test is not reversible. In fact, in case of no preload responsiveness, which occurs in half of the cases, it is not possible to withdraw the fluid administered in excess, leading to fluid overload. For this reason, some authors have suggested the idea of administering only a "mini-fluid challenge", consisting in infusing 100–150 mL of fluids over one or 2 min and measuring the response of cardiac output or one of its surrogates [100]. Two systematic reviews and meta-analyses, recently published, confirmed that the mini-fluid challenge test is reliable [29,101]. However, the threshold for the variation of cardiac output after a mini-fluid challenge is small (5% on average), thus requiring a very precise technique to measure cardiac output during the test. The pulse wave contour analysis is very useful in this context, as its sensibility is high enough to detect 1–2% changes in cardiac output [102]. On the contrary, echocardiography is not suitable, as its least significant change in cardiac output is 12% [103].

5.3. Tidal volume challenge

As discussed before, PPV is not reliable in case of low Vt ventilation. In order to overcome this issue, some authors suggested to perform a Tidal Volume Challenge. In a mechanically ventilated patients, the test consists of increasing tidal volume transiently from 6 to 8 mL/kg and measuring the related changes in PPV [31]. If the value of PPV increases more than 15%, this means that both ventricles are preload-dependent. Not many studies have been investigating reliablility of tidal volume challenge. However, a recent meta-analysis confirmed its value [29], especially in the context of operating room [104] and in prone patients [105], when PLR test is not feasible. The main limitation of this test is related to the necessity of mechanically controlled ventilation, as Tidal Volume Challenge is not feasible when a patients is not connected to a ventilator, and its diagnostic value is poor in case of some spontaneous breathing under mechanical ventilation [106].

6. Future perspective in predicting fluid responsiveness

Several other methods to predict fluid responsiveness have been proposed, including end-expiratory occlusion test (EEOT), carotid flow time (CFT), bioreactance-based non-invasive cardiac output monitoring (NICOM) and machine learning approaches applied to transthoracic echocardiography. A comparison between the various methods in use to predict fluid responsiveness according to the equipment needed, their advantages and disadvantages is provided in Table 1.

6.1. End-expiratory occlusion test (EEOT)

EEOT is a bedside maneuver that involves transiently interrupting mechanical ventilation at end-expiration for 15 seconds, which results in a transient increase in preload due to the absence of intrathoracic pressure variations [107]. The change in cardiac output following EEOT can be assessed using echocardiography or other hemodynamic monitoring devices. EEOT has been shown to predict fluid responsiveness in mechanically ventilated patients. An EEOT-induced increase in cardiac output or stroke volume of greater than 5% is generally considered to predict fluid responsiveness with high sensitivity and specificity [108]. However, EEOT is less accurate in predicting fluid responsiveness in patients with spontaneous breathing efforts or arrhythmias [107]. Additionally, the performance of EEOT may be influenced by factors such as lung compliance [109].

Table 1
Comparison between the methods in use to predict fluid responsiveness. CVP: Central Venous Pressure; PCWP: Pulmonary Capillary Wedge Pressure; CVC: Central Venous Catheter; SVV: Stroke Volume Variation; PPV: Pulse Pressure Variation; SPV: Systolic Pressure Variation; PVI: Pleth Variability Index; ARDS: Acute Respiratory Distress Syndrome; IVC: Inferior Vena Cava; SVC: Superior Vena Cava; PLR: Passive Leg Raising; LV: Left Ventricular; ICP: Intra Cranial Pressure; EEOT: End Expiratory Occlusion Test; CFT: Carotid Flow Time; NICOM: Bioreactance-based non-invasive cardiac output monitoring.

	Equipment	Advantages	Disadvantages
Static indices of preload			
- ***CVP***	o CVC, transducer	o Hemodynamic parametrs integration	o Poor fluid resposivness prediction
- ***PCWP***	o Pulmunory catheter set		o Pulmunary artery damage, infections
Dynamic indices od preload			
- ***SVV***	o Pulse contour analysis device (es. ClearSight, Hemosphere, PiCCO)	o High fluid resposivness prediction sensitivity	o ARDS, cardiac arrhythmia, spontaneous breathing, open chest surgery
- ***PPV***			
- ***SPV***	o Plethysmography	o No specific devices needed	
- ***PVI***		o Non-invasive or mini-invasive	
Echocardiographic methods			
- ***IVC index***	o Ultrasound device	o High fluid resposivness prediction sensitivity	o Operator-dependent
- ***SVC index***	o Critical care bed unit	o Non-invasive	o Low Tidal Volume, Arrhythmia, LV dysfunction, increased abdominal pressure, high ICP
Dynamic tests			
- ***PLR test***	o Hemodynamic monitoring (or Ultrasound device)	o Easy to perform	o Low diagnostic thresholds
- ***Mini-fluid Challenge***		o Repeatable	o Sensible monitoring required
- ***Tidal Volume Challenge***	o Critical care bed unit	o Reliable	o Irreversible (Mini-fluid Challenge)
Future perspective methods			
- ***EEOT***	o EtCO2 monitoring	o High fluid resposivness prediction sensitivity	o Operator-dependent
- ***CFT***	o Ultrasound doppler		o Low lung compliance, aortic valve disease, LV dysfunction
- ***Machine learning applied to echocardiography***	o Specific equipment (NICOM)	o Moderate sensitivity of fluid resposivness	o High thoracic impedance, lung disease, electrical interference, chest deformities
- ***Bioreactance***			

6.2. Carotid flow time (CFT)

CFT, also known as carotid artery corrected flow time, is the duration of carotid blood flow during systole, corrected for heart rate. CFT can be measured noninvasively using Doppler ultrasound of the carotid artery [110,111]. CFT has been shown to predict fluid responsiveness in both mechanically ventilated and spontaneously breathing patients [112]. A CFT of less than 330–340 ms is generally considered to predict fluid responsiveness with moderate sensitivity and specificity [113]. However, CFT may be influenced by factors such as arterial compliance, aortic valve disease, and left ventricular function [114]. Additionally, the accuracy of CFT in predicting fluid responsiveness may be operator-dependent [113].

Moreover, the innovative integration of bio-adhesive ultrasound (BAUS) technology into wearable devices holds potential for revolutionizing real-time access to patients' hemodynamics [115]. Previous studies have reported a direct relationship between the CVP and the ratio of the internal jugular-to-common carotid artery diameters. Since Doppler power is directly related to the number of red cell scatterers within a vessel, BAUS technology uses the ratio of internal jugular-to-carotid artery Doppler power as a surrogate for the ratio of the vascular areas of these two vessels. Given that, the ratio of internal jugular-to-carotid artery Doppler power would track the CVP change during hemorrhage and resuscitation [116]. This marks a paradigm shift in real-time ultrasound monitoring [117], unlocking doors to novel research fields.

6.3. Bioreactance-based non-invasive cardiac output monitoring (NICOM)

NICOM is a non-invasive method to measure cardiac output based on the analysis of variations in transthoracic electrical impedance caused by changes in blood flow and the aortic pulsatile volume [110]. NICOM has been proposed as a tool to predict fluid responsiveness by assessing changes in cardiac output following a PLR test or a fluid challenge [118].

Several studies have shown that NICOM can predict fluid responsiveness in both mechanically ventilated and spontaneously breathing patients. A NICOM-derived PLR-induced increase in cardiac output or stroke volume of greater than 10% is generally considered to predict fluid responsiveness with moderate sensitivity and specificity [119].

However, the accuracy of NICOM in predicting fluid responsiveness may be influenced by factors such as body habitus, electrode placement, and electrical interference. Additionally, NICOM's performance may be less reliable in patients with significant lung disease, chest wall deformities, or intrathoracic fluid accumulations [110].

6.4. Machine learning approaches applied to transthoracic echocardiography

Machine learning can be considered as the application of artificial intelligence as it enables the extraction of patterns from training examples, which is a key component of human intelligence. Algorithms derived from machine learning may be useful components of computer-aided diagnosis and decision support systems [120]. In critical ill patients, static cardiac-ultrasound-derived parameters have been shown to have a low ability to predict fluid responsiveness [121]. In this context, machine learning may be useful to combine echocardiographic parameters to increase their predictive power [122]. This hypothesis has been tested in small prospective studies both in spontaneously breathing [123] and mechanically ventilated critically ill patients [124], showing promising results. However, this approach is new and it still needs to be validated in further larger studies in order to define its role in the future of fluid responsiveness prediction.

7. Discussion

Predicting fluid responsiveness in critically ill patients is essential for optimizing fluid therapy and avoiding the adverse effects of under- and over-resuscitation. Although static indices of preload, such as CVP and PCWP, have been widely used in clinical practice, they are poor predictors of fluid responsiveness. Dynamic indices, such as PPV, SVV, and SPV, have emerged as more accurate predictors of fluid responsiveness in mechanically ventilated patients with regular cardiac rhythms. However, their accuracy is limited in patients with spontaneous breathing efforts, arrhythmias, or low tidal volumes. Echocardiographic methods, such as IVC collapsibility, delta Vpeak, and PLR, have shown promise in predicting fluid responsiveness, with PLR being applicable to both mechanically ventilated and spontaneously breathing patients. Other methods, such as EEOT, CFT, and NICOM, have also been proposed as potential tools for predicting fluid responsiveness. The choice of method should be tailored to the individual patient's clinical context and the availability of resources.

8. Conclusions

A comprehensive understanding of the various methods to predict fluid responsiveness and their respective limitations is crucial for the management of critically ill patients. Further research is needed to refine these methods and develop novel, non-invasive, and reliable techniques to optimize fluid therapy in the critically ill population.

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CRediT authorship contribution statement

Luigi La Via: Writing – original draft, Conceptualization. **Francesco Vasile:** Writing – original draft, Investigation. **Francesco Perna:** Writing – original draft. **Mateusz Zawadka:** Writing – review & editing, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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