Arterial Pulse Pressure Variation with Mechanical Ventilation

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Abstract

Fluid administration leads to a significant increase in cardiac output in only half of ICU patients. This has led to the concept of assessing fluid responsiveness before infusing fluid. Pulse pressure variation (PPV), which quantifies the changes in arterial pulse pressure during mechanical ventilation, is one of the dynamic variables that can predict fluid responsiveness. The underlying hypothesis is that large respiratory changes in left ventricular stroke volume, and thus pulse pressure, occur in cases of biventricular preload responsiveness. Several studies showed that PPV accurately predicts fluid responsiveness when patients are under controlled mechanical ventilation. Nevertheless, in many conditions encountered in the ICU, the interpretation of PPV is unreliable (spontaneous breathing, cardiac arrhythmias) or doubtful (low V_T). To overcome some of these limitations, researchers have proposed using simple tests such as the V_T challenge to evaluate the dynamic response of PPV. The applicability of PPV is higher in the operating room setting, where fluid strategies made on the basis of PPV improve postoperative outcomes. In medical critically ill patients, although no randomized controlled trial has compared PPV-based fluid management with standard care, the Surviving Sepsis Campaign guidelines recommend using fluid responsiveness indices, including PPV, whenever applicable. In conclusion, PPV is useful for managing fluid therapy under specific conditions where it is reliable. The kinetics of PPV during diagnostic or therapeutic tests is also helpful for fluid management.

Keywords: fluid responsiveness; cardiac preload; heart–lung interaction; cardiac output

Until the mid-1990s, hemodynamic resuscitation of patients with circulatory failure has most often been guided by data provided by the pulmonary artery catheter. Since then, the use of the pulmonary artery catheter has declined dramatically (1). The factors that have contributed to this decline are multiple, including the publication of randomized controlled trials that showed either no benefit (2) or even deleterious effects (3) of strategies aimed at maximizing oxygen delivery in ICU patients. Moreover, a multicenter nonrandomized clinical study showed increased mortality associated with pulmonary artery catheterization (4), although subsequent randomized controlled trials did not confirm these findings (5, 6). These findings eventually contributed to *1*) a move away from the concept of maximizing oxygen delivery toward the concept of individualizing the therapeutic strategy, and *2*) the development of novel and less-invasive alternatives to pulmonary artery catheterization (7).

Twenty Years Ago: Emergence of the Concept of Fluid Responsiveness and its Bedside Assessment

The concept of prediction of fluid responsiveness has emerged in the context of individualization of hemodynamic therapy and occupies a major place in guiding resuscitation of ICU patients today (8, 9). Fluid responsiveness is generally defined as the ability of the heart to increase its stroke volume or cardiac output in response to a fluid bolus (10). Physiologically, fluid responsiveness implies that both ventricles are preload responsive (i.e., they operate on the steep part of the curve plotted from the respective stroke volume vs. preload relationship; Frank-Starling mechanism) (Figure 1). A systematic review of the literature reported that fluid responsiveness is present in only 50% of the ICU patients for whom a decision for fluid administration was made by the treating physician (10). However, one cannot rule out the possibility that the percentage would be higher if only patients in shock are considered. Nevertheless, given

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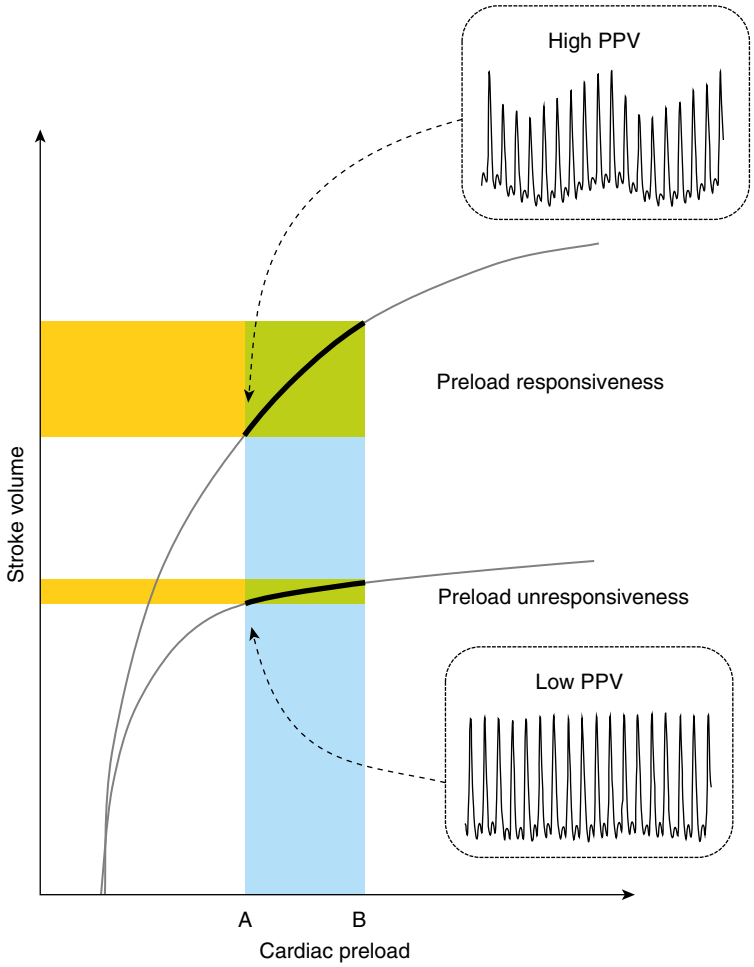

Figure 1. Frank-Starling relationship, preload responsiveness, and pulse pressure variation (PPV). When the heart is operating on the steep part of the Frank-Starling relationship (top), an increase in cardiac preload (from A to B) results in an increase in stroke volume (preload responsiveness). When the heart is operating on the plateau of the Frank-Starling relationship (bottom), the same increase in cardiac preload (from A to B) does not result in a significant increase in stroke volume (preload unresponsiveness). Importantly, a given value of cardiac preload (however it is measured) before preload increase (A) cannot predict preload responsiveness/unresponsiveness, whereas PPV is especially valuable for that purpose.

that fluid overload may cause harm to ICU patients (11), particularly to fluid nonresponders (12), it is important to detect fluid responsiveness before administering any fluid.

Measures of cardiac preload, such as central venous pressure (CVP), are not useful for predicting fluid responsiveness (13). A given value of cardiac preload can be associated with either fluid responsiveness or fluid unresponsiveness, depending on the shape of the Frank-Starling curve (Figure 1). There is large variability in the shape of the Frank-Starling curve among individuals, mainly related to differences in cardiac contractility.

A more valuable method for predicting fluid responsiveness is to estimate the slope of the Frank-Starling curve by measuring the short-term changes in stroke volume (or in its surrogates) in response to a brief change in cardiac preload. The classical method (infusing a small volume of fluid within a short time) involves fluid administration, which may harm fluid nonresponders, especially when the test is repeated frequently. Thus, dynamic tests, which challenge the Frank-Starling relationship without the need for any fluid infusion, have become very popular. In the late 1990s, quantification of the respiratory variation of stroke volume emerged as a practical application of the theoretical heart–lung interaction principles described in the 1980s by experts in cardiovascular and respiratory physiology (14–16).

The physiological background is that mechanical ventilation induces cyclic changes in loading conditions of both ventricles (14–17) (Figure 2). Insufflation decreases right ventricular (RV) preload as a consequence of the decrease in venous return due to the inspiratory increase in intrathoracic pressure. Insufflation generally increases RV afterload as a consequence of the inspiratory increase in transpulmonary pressure (18), in particular when West's zone 2 conditions—when alveolar pressure becomes higher than the pulmonary venous pressure—are extended (19). As a result, RV stroke volume is minimal at the end of insufflation (15, 18). It is generally assumed that the inspiratory reduction in venous return is the predominant mechanism, especially if the right ventricle operates on the steep part of the Frank-Starling curve (RV preload responsiveness). The inspiratory decrease in RV stroke volume leads to a decrease in left ventricular (LV) filling after a phase lag of two to four heartbeats due to the blood pulmonary transit time. This generally occurs during expiration (17). In cases of LV preload responsiveness, the decrease in LV preload eventually results in a decrease in the LV stroke volume, which is thus minimal during expiration. From these physiological principles, it has been postulated that during mechanical ventilation, large changes in LV stroke volume should occur in cases of biventricular preload responsiveness, whereas no change in LV stroke volume should occur if at least one ventricle is preload unresponsive (17). Numerous studies have consistently demonstrated that the magnitude of respiratory variation of stroke volume predicts fluid responsiveness with great accuracy in mechanically ventilated patients (12, 20, 21).

Pulse Pressure Variation: A Valuable Index to Predict Fluid Responsiveness in Patients Receiving Mechanical Ventilation

Among all the indices of fluid responsiveness, pulse pressure variation (PPV) has been one of the most studied and the most used in clinical practice.

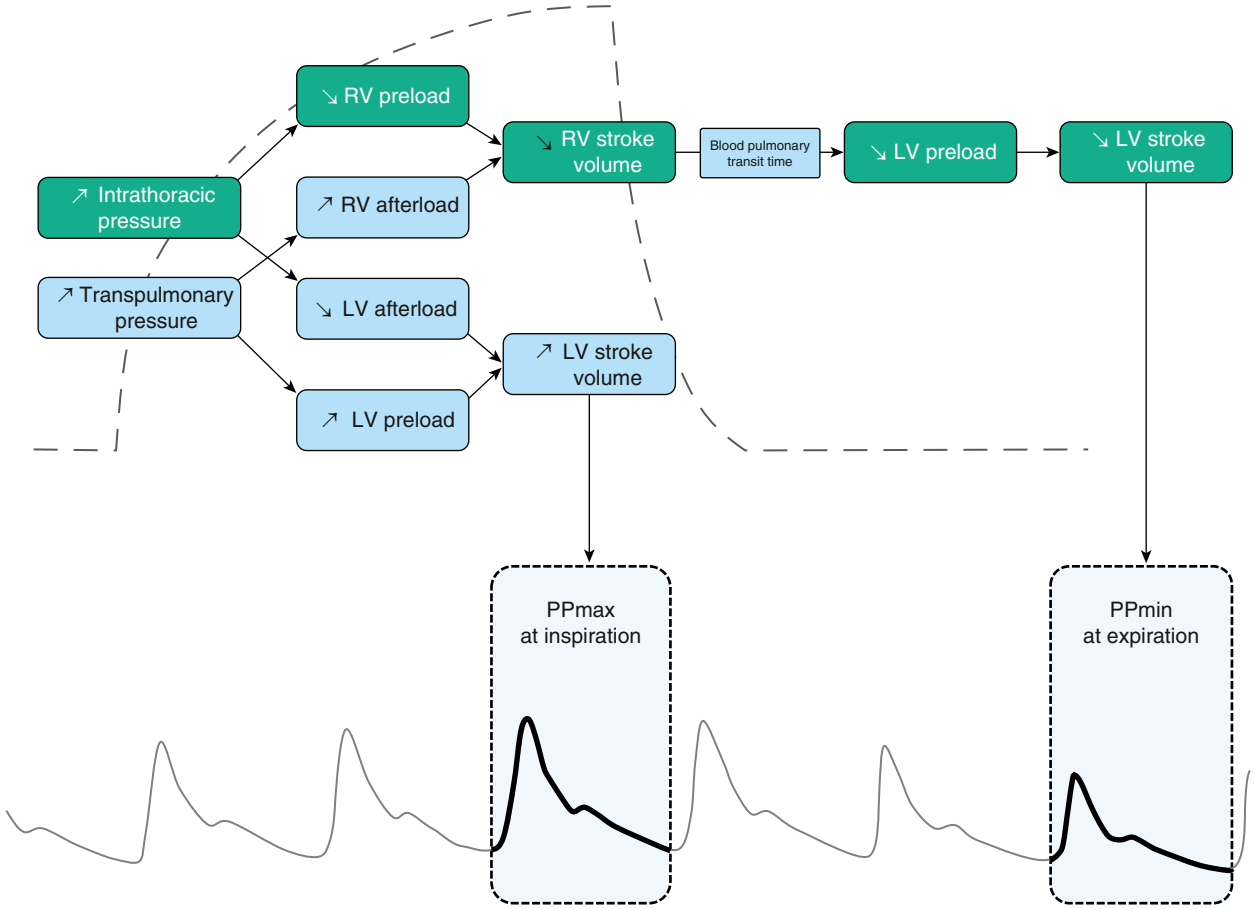

Figure 2. Mechanisms of heart–lung interactions explaining pulse pressure variation Top: airway pressure tracing; bottom: arterial pressure tracing. LV = left ventricle; PP = pulse pressure; RV = right ventricle. Adapted by permission from Reference 17.

Aortic pulse pressure (systolic pressure minus diastolic pressure) is directly proportional to LV stroke volume and inversely related to aortic compliance (22). We postulated that PPV reflects the respiratory changes in stroke volume and, thus, should help assess fluid responsiveness (23, 24). This is assuming that arterial compliance does not change over the respiratory cycle, a hypothesis that has been confirmed experimentally (25). One of the advantages of using PPV rather than systolic pressure variation, which had been previously evaluated (26, 27), is that pulse pressure, which is differential, is less directly influenced by the cyclic changes in intrathoracic pressure than systolic pressure. Accordingly, we showed that PPV was a better predictor of fluid responsiveness than systolic pressure variation (24). Importantly, in this study conducted in patients with septic shock, PPV was a far better predictor of fluid responsiveness than cardiac filling pressures. We defined fluid responsiveness by an increase in thermodilution-derived cardiac output by more than 15% in response to a fluid bolus (500 ml colloid infused over 30 min) (24). Further studies in different clinical settings confirmed the utility of PPV as a reliable predictor of fluid responsiveness in patients ventilated with a V_T of at least 8 ml/kg (19, 20), provided that they have no spontaneous breathing activity and/or cardiac arrhythmias (28). In a meta-analysis including 22 studies and 807 patients, PPV predicted fluid responsiveness with an area under the receiving operating characteristic (AUROC) curve of 0.94 and a threshold of 12% (20). It is noteworthy that in the studies included, fluid responsiveness was defined using one of the following techniques: thermodilution, transpulmonary thermodilution, pulse contour analysis, or esophageal Doppler (20).

Initially, PPV was manually determined as the ratio of the difference between the maximal and minimal values of pulse pressure over the mean of these two values and expressed as a percentage (Figure 3) (23). Today, most hemodynamic monitors allow automatic calculation of PPV with continuous display of its value in real time (29). Using proprietary algorithms, some monitors estimate the stroke volume on the basis of the arterial pressure waveform analysis. (30). They also calculate and display stroke volume variation (SVV), which has been assumed to reflect the respiratory variation of stroke volume in patients receiving mechanical ventilation (31). In a meta-analysis, SVV predicted fluid responsiveness with greater accuracy than markers of cardiac preload in mechanically ventilated patients (19). However, SVV was significantly less accurate than PPV (AUROC curve: 0.84 vs. 0.94, respectively) (19). The results are not

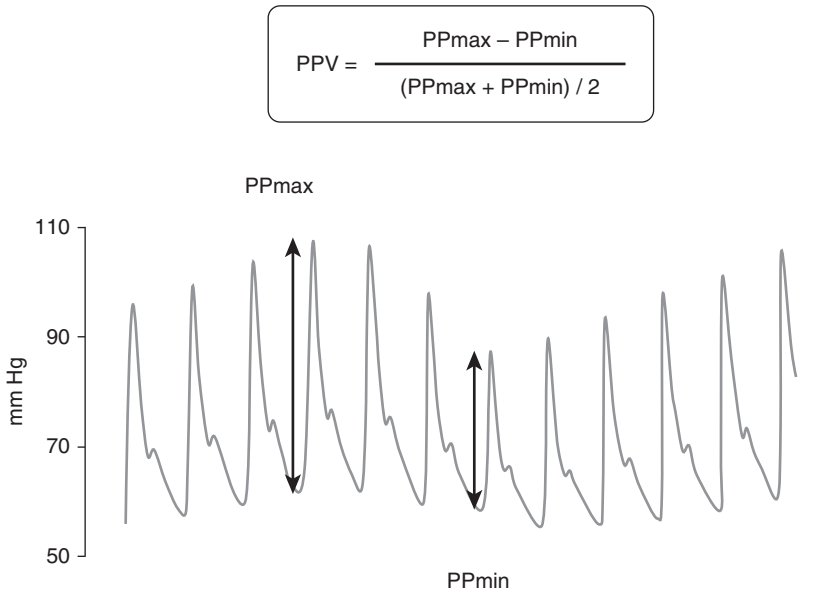

Figure 3. Calculation of pulse pressure variation (PPV) from an arterial pressure curve. PP = pulse pressure.

surprising, because the calculation of PPV is prone to fewer errors than the more complex computation of SVV. Another advantage of PPV over SVV is that it only requires a simple arterial catheter to collect data.

Noninvasive devices that measure respiratory changes in various hemodynamic signals can also be used to predict fluid responsiveness during mechanical ventilation. The PPV measured using a finger blood pressure device (volume clamp method) was as reliable as the invasively obtained PPV to predict fluid responsiveness in the operating room (OR) (32) as well as in the ICU (33) setting. The respiratory changes in the amplitude of the plethysmographic signal predict fluid responsiveness with acceptable accuracy (34, 35), except in patients receiving norepinephrine (36, 37). The respiratory changes in ultrasound variables, such as the velocity time integral of the LV outflow track (echocardiography) (38), the aortic blood flow (esophageal Doppler) (39), the diameter of the inferior (40) or superior vena cava (41), and the diameter of the internal jugular vein (42), have been shown to be acceptable predictors of fluid responsiveness, although they have been less studied and less validated than PPV. Overall, this confirms the theoretical basis that heart–lung interactions during mechanical ventilation can be judiciously used for predicting fluid responsiveness. Noninvasive fluid responsiveness indices, as described above, can be useful when an arterial cannulation is not performed. In addition, it is recommended that echocardiography be performed as soon as possible, as it is the preferred modality to initially evaluate the type of shock (8).

PPV in Specific Clinical Situations: What Are the Limitations? How Can They Be Overcome?

Several conditions, listed in Table 1, limit the interpretation of PPV (27). It is thus important to discuss the utility of PPV in some specific settings.

PPV and Acute Respiratory Distress Syndrome

At least two factors limit the use of PPV in acute respiratory distress syndrome (ARDS): low V_T ventilation and low lung compliance (43).

Low V_T ventilation is recommended in patients with ARDS (9). Under these conditions, respiratory changes in intrathoracic pressure might be insufficient to produce significant changes in preload. Accordingly, it was reported that in the case of V_T greater than or equal to 8 ml/kg, PPV accurately predicted fluid responsiveness

Table 1. Conditions Where Pulse Pressure Variation Is Less Reliable

Spontaneous breathing	False +
Cardiac arrhythmias	False +
Low V_T	False −
Low lung compliance	False −
Increased intraabdominal pressure	False +
Very high respiratory rate (HR/RR < 3.6)	False −
Right ventricular dysfunction	False +

Definition of abbreviations: HR = heart rate; RR = respiratory rate.

(AUROC curve, 0.89, with a threshold value of 12%), whereas the prediction was weaker (AUROC curve, 0.70, with a threshold value of 8%) when V_T was less than 8 ml/kg (44). Nevertheless, during low V_T ventilation, three important issues need to be highlighted. First, a high PPV (e.g., >12%) still suggests fluid responsiveness. Second, a low PPV cannot rule out fluid responsiveness. Third, to overcome the difficult interpretation of low PPV, it has been suggested to measure the response of PPV to a transient (<1 min) increase in V_T (45). Myatra and colleagues confirmed that PPV poorly predicts fluid responsiveness at 6 ml/kg V_T (AUROC curve, 0.69) (46). After V_T was increased to 8 ml/kg, PPV more reliably predicted the hemodynamic response to fluid infusion, documented at 6 ml/kg (AUROC curve, 0.91) (46). Interestingly, an increase in the absolute value of PPV greater than or equal to 3.5% during the V_T challenge predicted fluid responsiveness with excellent accuracy (AUROC curve, 0.99) (46). Another way to overcome the limitation of using PPV in case of low V_T is to divide PPV by the respiratory changes in esophageal pressure (AUROC curve, 0.94 with vs. 0.78 without adjustment) (47). The disadvantage of using this index is the need for an esophageal probe.

Low lung and respiratory system compliance (Crs = V_T/driving pressure), which are characteristics of ARDS, can also result in a misleading interpretation of PPV by reducing the transmission of airway pressure to the intrathoracic structures (43). Although chest wall compliance is generally reduced, the resultant effect is a decrease in that airway pressure transmission, as illustrated by the correlation between Crs and the airway pressure transmission found in a previous

study (48). It has been shown that when Crs was greater than 30 ml/cm H_2O, PPV accurately predicted fluid responsiveness (AUROC curve, 0.98), whereas when Crs was less than or equal to 30 ml/cm H_2O, the prediction was poor (AUROC curve, 0.69) essentially because of a high rate of false negatives (49). Interestingly, among fluid responders, there was a subset of patients ventilated with a VT less than 8 mL/kg, a Crs greater than 30 ml/cm H_2O and a high PPV, and another subset with a VT greater than 8 ml/kg, a Crs less than or equal to 30 ml/cm H_2O and a low PPV (5% on average), suggesting that the decreased Crs might play a more important role than the low VT in the poor predictive value of PPV (49).

Other factors limit the interpretation of PPV in ARDS. Persistent breathing activity during mechanical ventilation is common, because the current recommendation is to minimize the use of sedative agents and allow the patient to partially use his/her respiratory muscles (9), although this issue is still a matter of debate (50). In such cases, PPV cannot predict fluid responsiveness (51), because the respiratory changes in intrathoracic pressure are irregular, either in rate or in amplitude. In case of low VT controlled ventilation, a high respiratory rate may be needed. In such a case, the decrease in LV filling secondary to the insufflation-induced decrease in RV stroke volume might occur at insufflation and not at expiration, resulting in low PPV even in cases of fluid responsiveness. A clinical study showed that PPV cannot be interpreted reliably when the heart rate/respiratory rate ratio is lower than 3.6 (52). In addition, Prone positioning is often used in patients with severe ARDS. One clinical study reported a poor predictive value of PPV during prone positioning in patients with ARDS (53), probably due to the low VT ventilation and the low lung compliance. Finally, the level of positive end-expiratory pressure (PEEP) should not affect the predictive value of PPV. Indeed, PEEP increases mean airway pressure but does not change cyclic variation in airway pressure during a mechanical breath, which is the main determinant of PPV. Some publications that showed a good predictive value for PPV during mechanical ventilation included patients receiving PEEP (23, 24, 33, 41, 54). Interestingly, PPV can also be used to predict the hemodynamic effects of PEEP (23). We speculated that a high PPV in patients with ARDS was linked to the presence of biventricular preload dependence and that a significant decrease in cardiac output with PEEP should occur only in patients with biventricular preload dependence. Accordingly, we showed that the higher the PPV before applying PEEP, the more pronounced the decrease in cardiac output with PEEP (23).

PPV and RV Dysfunction

It has been suggested that RV dysfunction could result in false-positive values of PPV (i.e., high PPV despite fluid unresponsiveness). This would occur because of the predominant effect of mechanical insufflation on RV afterload through the compression of intraalveolar microvessels by transpulmonary pressure. As a failing and dilated right ventricle is more sensitive to its afterload than to its preload, the decrease in RV stroke volume during insufflation would be more related to RV afterload dependence than to RV preload dependence. Two clinical studies reported high PPV values (>12%) despite fluid unresponsiveness in the context of RV dysfunction (55, 56). However, in these studies, the VT was greater than 8 ml/kg, and one cannot exclude an attenuation of the phenomenon of RV afterload dependence during low VT ventilation. In addition, in these studies, which were performed before the publication of international guidelines (57), the way RV function was assessed can be debated.

PPV and Congestive Heart Failure

Few studies have addressed the issue of fluid responsiveness in patients with congestive heart failure, probably because fluid administration is rarely indicated in such patients. In some studies, including patients with left ventricular dysfunction and in sinus rhythm, PPV (or SVV) was reported to predict fluid responsiveness with an acceptable accuracy (41, 58). However, the presence of cardiac arrhythmias is an obvious contraindication to the use of PPV, because the variation of pulse pressure is mainly related to the irregularity of the cardiac diastole, irrespective of the respiratory cycle.

PPV and Intraabdominal Hypertension

Experimental data suggest that PPV can still predict fluid responsiveness in cases of increased intraabdominal pressure but that the threshold value might be higher than in the case of normal abdominal pressure (59). However, the experimental conditions (acute increase of intraabdominal pressure, very high values of intraabdominal pressure achieved, high VT, and low chest compliance) were far from those encountered in ICU patients. In a series of ventilated patients with acute liver failure, the authors concluded that PPV predicted fluid responsiveness, whereas the respiratory changes in velocity time integral were not predictive (60).

PPV in the General Population of ICU Patients

An international observational study published in 2015 investigated 2,213 patients to determine whether fluid responsiveness indices were used before performing fluid challenges (61). The CVP was the most used variable (576 cases) compared with PPV or SVV (176 cases) and passive leg raising (248 cases) (61). These practices are likely to evolve, as the most recent version of the Surviving Sepsis Campaign guidelines suggests using dynamic indices of fluid responsiveness (including PPV when applicable) instead of CVP when hemodynamic reassessment is required after the initial fluid bolus (9).

Low VT ventilation is commonly used in critically ill patients without ARDS, as illustrated by a study that enrolled 540 mechanically ventilated patients, the vast majority of whom received low VT ventilation (62). PPV as well as echocardiographic fluid responsiveness indices did not reliably predict fluid responsiveness (62). However, in this study, fluid responsiveness was identified by passive leg raising and not by fluid administration (62). Because passive leg raising was shown to be hemodynamically equivalent to 312 ml (minimum, 250 ml; maximum, 350 ml) of fluid administration (63), there were likely a significant number of true 500-ml fluid responders rated nonresponders using passive leg raising and some fluid nonresponders classified as responders using passive leg raising (62). This may have resulted in higher rates of false-positive, and to a lesser extent in higher rates of false-negative, cases of PPV (64) compared with studies that assessed fluid responsiveness using a real fluid administration.

The conditions under which the reliability of PPV is limited are quite common in the ICU. As an example, in a prospective study that included ICU patients who actually received fluid boluses, the incidence of cases where PPV could be used without limitation was 17% (65). This incidence may vary in function of the case mix and the ventilator settings. Another study showed that in cases of shock, the conditions of applicability of PPV were present in 39% of patients with sepsis and 53% of patients with trauma (66). Performance of V_T challenge in cases of low V_T ventilation should extend the conditions of PPV use.

PPV and Impact on Outcome

In surgical patients, there is strong evidence that both insufficient and excessive fluid administrations are associated with an increased rate of postoperative complications (67). Tailoring fluid administration to individual needs is recommended (68). Maintaining patients close to the inflection part of the Frank-Starling curve (i.e., maintaining PPV within the range 10–15%) should protect from the consequences of both hypovolemia and fluid overload (69). A meta-analysis of 14 randomized controlled trials concluded that using PPV or SVV to guide fluid management during and/or right after surgery was associated with a significant decrease in postoperative morbidity (70). However, as a result of heterogeneity and inconsistency among the assessed studies, further confirmation is needed.

In medical ICU patients, no outcome-focused randomized controlled trial has yet compared PPV-based fluid management with standard care.

Practical Use of PPV

PPV should be considered for predicting fluid responsiveness only when the patient has no spontaneous breathing activity and has a sinus rhythm (Figure 4).

In cases of high values (e.g., >13%), PPV should have a good predictive value even if V_T or Crs are low. In case of doubt about a false-positive PPV (e.g., in the setting of RV dysfunction diagnosed by echocardiography), a passive leg raising test can be performed; a decrease in PPV during passive leg raising would suggest that the patient is actually fluid responsive, whereas no decrease in PPV would suggest that the patient is not fluid responsive and that the high PPV value was a false positive.

In cases of low values (e.g., <9%), PPV has an excellent predictive value if V_T is at least 8 ml/kg and Crs greater than 30 ml/kg. In patients with ARDS, interpretation is more difficult (false-negative cases) because of low V_T ventilation or low Crs. As mentioned above, this is a good scenario in which to perform a V_T challenge consisting of transiently increasing V_T (from 6 to 8 ml/kg) and measuring the absolute changes in PPV (45, 46).

Some authors described a "gray zone" for PPV (e.g., between 9% and 13%) where no conclusion can be drawn about fluid responsiveness, even when V_T is greater than or equal to 8 ml/kg (71). Challenging PPV after a transient increase in V_T from 8 to 12 ml/kg has been proposed to overcome this limitation (72).

Finally, PPV should not be interpreted in cases of cardiac arrhythmias and spontaneous breathing activity (Table 1 and Figure 4). This latter condition includes: *1*) patients who are intubated and ventilated with persistent respiratory efforts, *2*) patients with noninvasive ventilation, and *3*) patients with no mechanical ventilation, whether or not expiration is active (73). This is the right place for other dynamic tests of fluid responsiveness, such as passive leg raising or end-expiratory occlusion tests (49).

Whatever the method used, the presence of fluid responsiveness, which is a physiological phenomenon, should not lead

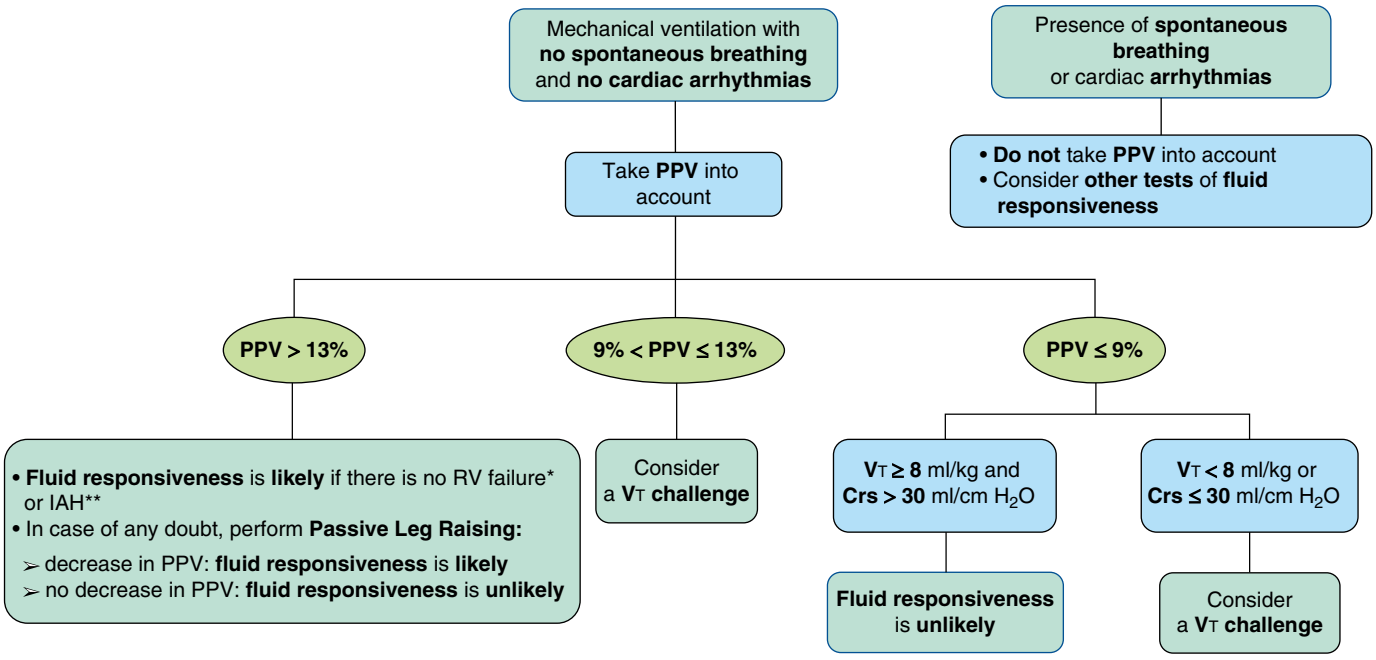

Figure 4. Practical use of pulse pressure variation (PPV). *Detected by echocardiography; **detected by measuring intraabdominal pressure. Crs = compliance of the respiratory system; IAH = intraabdominal hypertension; RV = right ventricular.

automatically to fluid administration. In reality, three different situations should be distinguished.

The decision to initiate fluid administration urgently at the early phase of shock should not be based on the presence of fluid responsiveness indices, especially in cases of septic shock (9), active bleeding, or evident fluid losses.

The decision to continue fluid infusion after initial fluid resuscitation is a different issue, except in cases of active bleeding or persistent fluid losses. Because not all patients are fluid responsive, predictors of fluid responsiveness, although required, represent only one element of the decision-making process (Figure 5). The decision to infuse fluids should be based on the presence of three elements: *1*) signs of shock, *2*) fluid responsiveness, and *3*) limited risks of fluid overload. In cases where lung edema is a possible risk, additional variables, such as extravascular lung water (74) or the presence of B-lines on lung ultrasound examination (75), are useful in making the appropriate decision. The decision to discontinue fluid administration should be based on the presence of only one of the following three elements: *1*) disappearance of signs of shock, *2*) appearance of fluid unresponsiveness, or *3*) appearance of signs of pulmonary edema.

In the OR setting, fluid therapy is not limited to patients with shock. During high-risk surgery, the rationale for fluid administration is to optimize hemodynamics to prevent postoperative complications. As mentioned above, fluid strategies guided by PPV (or SVV) may result in improved outcomes (70).

Recently, researchers have proposed using the PPV/SVV ratio—called dynamic arterial elastance—to predict the blood pressure response to fluid administration. Two clinical studies (76, 77) suggested that a low PPV/SVV ratio predicts the absence of blood pressure response to fluid and hence would recommend the use of vasopressors. However, other studies have failed to replicate these results (78, 79). An experimental study (80) showed that vasopressors decrease the PPV/SVV ratio, calling into question the recommendation to give vasopressors when the ratio is low. Some authors have reported that age is the main determinant of the PPV/SVV ratio because it is higher in the elderly than in younger patients (81). Thus, caution should be used in interpreting the PPV/SVV ratio as an indicator to initiate vasopressor therapy (82).

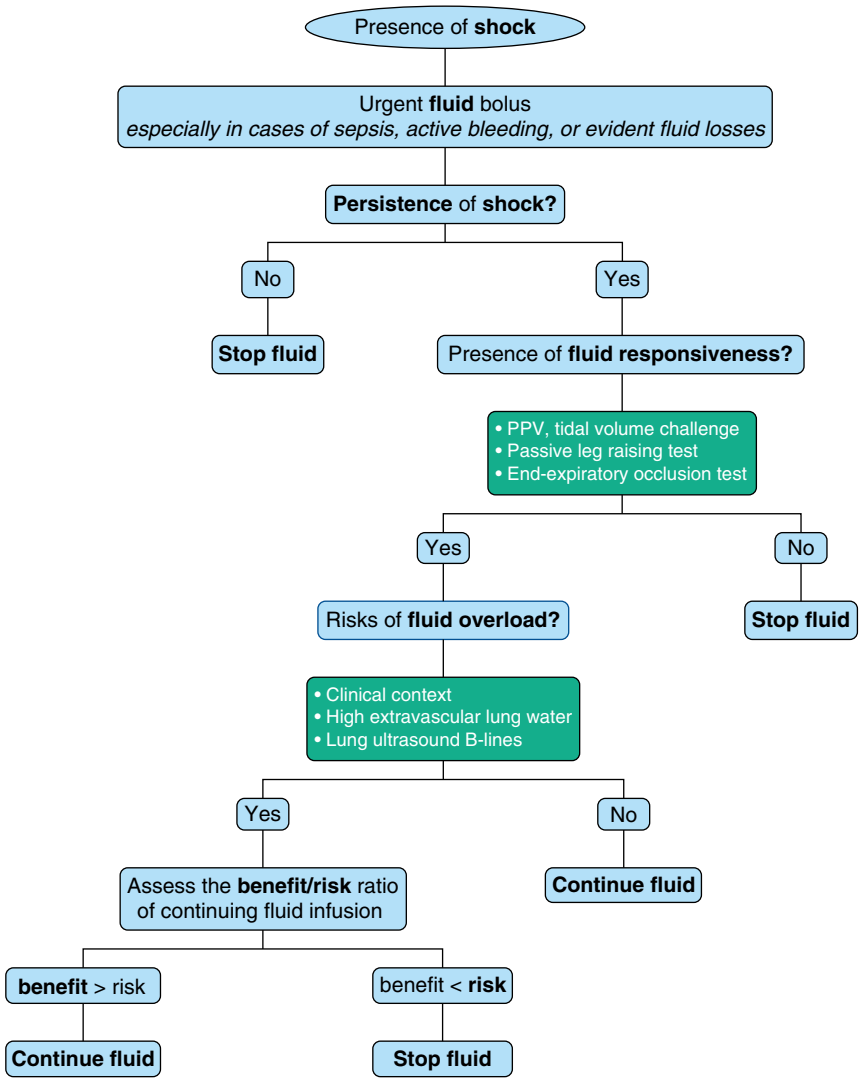

Figure 5. Decision algorithm for fluid administration in the ICU. PPV = pulse pressure variation.

Conclusions

PPV is an easy-to-obtain but not always easy-to-use marker of fluid responsiveness. Ignoring its limitations could lead to serious misinterpretation. A survey showed that a large proportion of intensivists did not have complete knowledge of factors confounding the interpretation of PPV (83). This indicates a need to improve education.

PPV is applicable for mechanically ventilated patients with no spontaneous breathing and no arrhythmias. Its validity is indisputable in cases of ventilation with a V_T greater than or equal to 8 ml/kg, with minimally altered lung compliance, and with no RV failure and no intraabdominal hypertension. Conditions for its optimum use are usually met in the OR, where fluid management strategies made on the basis of PPV (or SVV) monitoring have been shown to reduce postoperative morbidity (70). Moreover, monitoring the dynamics of PPV is of interest, because during fluid administration the decrease in PPV inversely correlates with the increase in cardiac output (23, 24, 84). Noninvasive PPV monitoring tools should be increasingly

used in the OR (85), provided that technological advancements would optimize their validity.

In the ICU, however, the optimal conditions for utilizing PPV are less likely to occur. Nevertheless, some limitations of PPV interpretation can be overcome by performing tests that rely on the dynamics of PPV, such as the V_T challenge. ■

Author disclosures are available with the text of this article at www.atsjournals.org.

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