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Lopes MR, Oliveira MA, ..., Auler JO, Michard F

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Commentary

Pulse pressure variation: beyond the fluid management of patients with shock

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See related research by Keyl *et al.*, http://ccforum.com/content/11/2/R46

Abstract

In anesthetized patients without cardiac arrhythmia the arterial pulse pressure variation (PPV) induced by mechanical ventilation has been shown the most accurate predictor of fluid responsiveness. In this respect, PPV has so far been used mainly in the decision-making process regarding volume expansion in patients with shock. As an indicator of the position on the Frank–Starling curve, PPV may actually be useful in many other clinical situations. In patients with acute lung injury or with acute respiratory distress syndrome, PPV can predict hemodynamic instability induced by positive end-expiratory pressure and recruitment maneuvers. PPV may also be useful to prevent excessive fluid restriction/depletion in patients with pulmonary edema, and to prevent excessive ultrafiltration in critically ill patients undergoing hemodialysis or hemofiltration. In the operating room, a goal-directed fluid therapy based on PPV monitoring has the potential to improve the outcome of patients undergoing high-risk surgery.

In the previous issue of *Critical Care*, Keyl and colleagues [1] have investigated the effects of cardiac resynchronization therapy on arterial pulse pressure variation (PPV). Many studies [2] have shown that PPV is much more accurate than cardiac filling pressures and volumetric markers of preload to predict fluid responsiveness (that is, the hemodynamic effects of volume loading). PPV is also more reliable than other dynamic parameters such as systolic pressure variation [3,4] or pulse contour stroke volume variation [4]. In this respect, PPV is used increasingly in the decision-making process regarding volume expansion in patients with hemodynamic instability [2]. Limitations to the use of PPV do exist (mainly active breathing, cardiac arrhythmia, and low tidal volume) and have been described in detail elsewhere [2,5].

It is very important to point out that PPV is not an indicator of volume status, nor a marker of cardiac preload, but is an indicator of the position on the Frank–Starling curve [2]. Briefly, patients operating on the flat portion of the Frank–Starling curve are insensitive to cyclic changes in preload induced by mechanical inspiration, such that PPV is low (Figure 1). Conversely, PPV is high in patients operating on the steep portion of the preload/stroke volume relationship (and hence sensitive to cyclic changes in preload induced by mechanical inspiration) (Figure 1). This information has so far been used mainly to predict fluid responsiveness in patients with shock, but actually could be useful in many other clinical situations.

PPV and fluid depletion/restriction

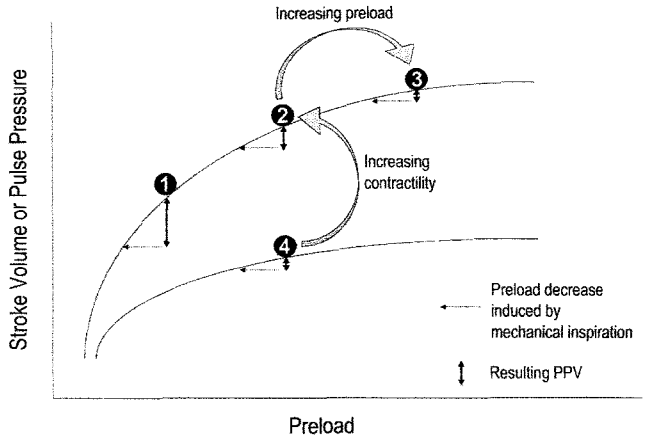
As an indicator of the position on the Frank–Starling curve, PPV is as useful to predict the deleterious hemodynamic effects of fluid depletion as it is to predict the beneficial effects of fluid loading [6]. In critically ill patients undergoing hemodialysis or hemofiltration the volume of ultrafiltration is often determined roughly on the basis of body weight gain or fluid balance, and is further adjusted in case of hemodynamic instability. In patients with acute respiratory distress syndrome, a therapeutic strategy based on fluid restriction/depletion has been shown to shorten the duration of mechanical ventilation and intensive care [7]. In such clinical situations, fluid management could be refined by PPV monitoring: a large PPV or an increase in PPV indicates that the patient is operating on the steep portion of the Frank–Starling curve, and hence indicates that further ultrafiltration or further fluid restriction/depletion will induce hemodynamic instability.

PPV and respiratory settings

The first description of PPV [8] was a study showing that the parameter can be used to predict the deleterious hemodynamic effects of positive end-expiratory pressure. We must

PPV = pulse pressure variation.

Figure 1



Determinants of pulse pressure variation. Pulse pressure variation (PPV) is a marker of the position on the Frank–Starling curve, not an indicator of blood volume or a marker of cardiac preload. Increasing preload induces a decrease in PPV (from ❷ to ❸). PPV is mimimal when the heart is operating on the plateau of the Frank–Starling curve (❸ and ❹). Decreasing preload induces an increase in PPV (from ❷ to ❶), also increasing contractility (from ❹ to ❷).

keep in mind that most patients with acute respiratory distress syndrome still die of multiple organ failure and not of hypoxemia. In this regard, PPV is now used (and normalized by the use of fluid) routinely by renowned groups [9] before performing recruitment maneuvers or before applying positive end-expiratory pressure in patients with acute respiratory distress syndrome, in order to prevent any hemodynamic deterioration. Conversely, PPV can also be used to predict the beneficial hemodynamic effects of positive end-expiratory pressure removal. In patients with chronic obstructive pulmonary disease and high auto-positive end-expiratory pressure, Lee and colleagues [10] have shown that PPV is closely related to the hemodynamic improvement observed in response to Heliox administration.

PPV and perioperative fluid optimization

Another potential field of application for PPV is the intraoperative fluid optimization of patients undergoing high-risk surgery. Several studies [11-13] have shown that monitoring and maximizing stroke volume by fluid loading (until the stroke volume reaches a plateau, actually the plateau of the Frank–Starling curve) during high-risk surgery is associated with improved postoperative outcome. The benefit in using such a peroperative fluid strategy was first established in patients undergoing cardiac surgery or hip surgery, and has been extended more recently to patients undergoing major bowel surgery or general surgery [11-13. This strategy has so far required the measurement of the stroke volume by a cardiac output monitor. By increasing cardiac preload, volume loading induces a rightward shift on the preload/stroke volume relationship and hence a decrease in PPV (Figure 1). Patients who have reached the plateau of the Frank–Starling relationship can be identified as patients in whom PPV is low. The clinical and intraoperative goal of 'maximizing stroke volume by volume loading' can therefore be achieved simply by minimizing PPV. A large multicenter trial is currently ongoing to investigate whether minimizing PPV by volume loading may improve the postoperative outcome of patients undergoing high-risk surgery.

PPV as a tool to track changes in contractility?

In the previous issue of *Critical Care*, Keyl and colleagues [1] reported a slight but significant increase in PPV (from 5.3% to 6.9%) during resynchronization therapy. Although the noninvasive method used by the authors to monitor blood pressure lacks validation, their finding makes sense since increasing left ventricular contractility means increasing the slope of the Frank–Starling curve, and hence increasing PPV (Figure 1). This result also suggests that PPV may be used to track changes in contractility in situations where changes in preload are unlikely. Keyl and colleagues did not, however, assess left ventricular contractility (for example, by measuring the maximum left ventricular pressure derivative, dP/dt_{max}). Moreover, biventricular pacing may induce a decrease in left ventricular volumes [14], which may also explain the increase in PPV. The relationship between changes in PPV and changes in contractility during cardiac resynchronization therefore remains to be proven.

Competing interests

The authors declare that they have no competing interest.

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intensity with PTSD but there was a linear positive relationship with having delirious memories and sedation. PTSD symptom scores were highest in the middle level of wakefulness and lowest when least aroused or the most awake suggesting if confused they are unable to process the meaning of the events they were experiencing. A European study (13) reported that the delusional memories were common (44%–77%), whereas the recall of pain and anxiety was not consistent during the follow-up at 2 weeks, 2 months, and 3 months with only half the patients showing any consistency. The rate of defined PTSD was 9.2% with a range of 3.2%–14.8%. Independent of case mix and illness severity, the factors related to PTSD were recalling of delusional memories, prolonged sedation, a history of preexisting psychological problems such as anxiety or depression, and physical restraint with no sedation. The frequency of delirium varied between units (14%–65%) and was more common in patients receiving high daily doses of benzodiazepines or opiates. The inconsistent reporting of pain during the ICU stay draws into question the results of studies where the reporting is done several months after the events such that the "experience" and its interpretation may have been altered with the passage of time. Initial reports suggested it was the number of "adverse" or "traumatic" memories that caused PTSD (14). However, this may be spurious and related to time in ICU, and not addressing the impact of any particular experience. The most important psychological stressor is the fear of harm or death to oneself and is dependent on the context and the ability to control the outcome. It is the combination of a stressful event plus a loss of personal security that is critical to developing PTSD. We must remember that the psychological interpretation of real and delusional experiences occurs on the background of disturbed or altered memory and amnesia, and that delusions matter (15).

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Using pulse pressure variation in patients with acute respiratory distress syndrome*

"To give or not to give fluid?" is a daily dilemma for intensivists treating patients with acute respiratory distress syndrome (ARDS). On one hand, excessive fluid administration may worsen pulmonary edema and prolong mechanical ventilation (1). On the other hand, underresuscitation may induce hemodynamic instability and multiple organ failure (the main cause of death in ARDS).

Ideally, like all medications, fluids (crystalloids or colloids—the choice between the two is beyond the scope of this editorial) should be administered only when a benefit is expected. In clinical practice, the decision for fluid administration is often based on the measurement of cardiac filling pressures or end-diastolic dimensions, and many studies have shown that fluid loading is responsible for a significant increase in cardiac output in only 50% of the cases (2).

In this issue of *Critical Care Medicine*, Huang et al. (3) have investigated the potential value of global end-diastolic volume index (a volumetric marker of preload evaluated by transpulmonary thermodilution) and of arterial pulse pressure variation (PPV, a dynamic marker of the position on the Frank-Starling curve) to

*See also p. 2810.

Key Words: pulse pressure variation; fluid responsiveness; acute respiratory distress syndrome; passive leg raising; cardiac output monitoring

Dr. Michard coholds patent US 2007/0179386 A1.

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predict fluid responsiveness in ARDS patients ventilated with low tidal volume and high positive end-expiratory pressure (PEEP) The study population was small (22 patients divided in two groups) and some findings were hard to explain. For instance, SvO_2 did not change after fluid loading despite a marked increase in cardiac output and PaO_2 (how could it be? unless hemoglobin dropped and/or oxygen consumption dramatically increased—which is very unlikely). Moreover, PPV was not assessed using the reference method (4). However, the results regarding global end-diastolic volume index and PPV make sense and are consistent with previous reports (5, 6). Before fluid loading (baseline), global end-diastolic volume index was comparable in responders and nonresponders to fluid loading, emphasizing the fact that assessing preload is not useful to predict fluid responsiveness. In contrast, PPV was significantly higher in responders than in nonresponders and significantly correlated with fluid loading induced increase in cardiac index.

In mechanically ventilated patients without cardiac arrhythmia, many studies have shown that PPV is a very specific and sensitive parameter to identify responders to fluid loading (6). In patients undergoing high-risk surgery, there is now evidence that monitoring and optimizing (i.e., minimizing) PPV improves outcome (7). Limitations to the use of PPV have been described in details elsewhere (6, 8). It is important to point out that concerns have been raised regarding the clinical value of PPV in patients with ARDS because both PEEP and tidal volume may affect PPV. The influence of PEEP and tidal volume on PPV can be summarized as follows.

PPV and PEEP. The first clinical study about PPV (4) investigated the behavior of this parameter when PEEP is applied in patients with acute lung injury or ARDS. This study (4) showed that PPV can be used to predict PEEP-induced hemodynamic instability—the higher the PPV on zero end-expiratory pressure, the greater will be the decrease in cardiac output when PEEP is applied. As a consequence, PPV is now used by renowned groups before applying PEEP or performing recruitment maneuvers in patients with ARDS (9). The above-mentioned study (4) also showed that PEEP does increase PPV. At first sight, this finding may be surprising since applying PEEP does not increase the cyclic variation in airway and pleural pressures (from end-expiratory to end-inspiratory values) during a single mechanical breath. Actually, by increasing mean airway and pleural pressures and hence by decreasing mean cardiac preload, PEEP induces a leftward shift on the Frank-Starling curve. Therefore, a patient operating on the flat portion of the Frank-Starling curve on zero end-expiratory pressure (i.e., a fluid nonresponsive patient) may move to the steep part of the curve when PEEP is applied, and become fluid responsive. In other words, if PEEP does affect PPV, it does not affect its physiologic or clinical value: PPV is still a marker of the position on the Frank-Starling curve and logically an accurate predictor of fluid responsiveness (4).

PPV and Tidal Volume. In patients with ARDS, a low tidal volume is recommended to prevent the so-called ventilator-induced lung injury and *in fine* to improve outcome (10, 11). Tidal volume is the main determinant of respiratory variations in pleural pressure and cardiac preload which are responsible for significant PPV in patients operating on the steep portion of the Frank-Starling curve (6). Without any significant change in pleural pressure and cardiac preload, a PPV cannot be observed even in fluid responsive patients (e.g., during apnea, PPV equal zero even in patients operating on the steepest part of the Frank-Starling curve!). De Backer et al. (12) have confirmed that PPV is less accurate to predict fluid responsiveness when tidal volume is <8 mL/kg than when it is >8 mL/kg. In patients with ARDS ventilated with a mean tidal volume of 6.4 mL/kg, Huang et al. (3) observed that a PPV cut-off value of 12% discriminates between fluid-responders and nonresponders with a specificity of 100% and a sensitivity of 68%. It has been suggested that acute cor pulmonale—the incidence of which has been significantly lowered by the use of low tidal volumes (13)—may be responsible for large PPV in patients nonresponder to fluid loading (false positive) (14). The specificity of 100% reported by Huang et al. supports the notion that such a phenomenon (high PPV values in nonresponders) is actually very uncommon. However, the sensitivity of 68% indicates that false negative may be observed (roughly one third of responders were not properly detected by PPV). As explained above, this phenomenon is likely related to small respiratory variations in pleural pressure and cardiac preload in patients ventilated with low tidal volumes (<8 mL/kg).

In summary, we would temper the enthusiastic conclusions of Huang et al. (3) by the following take home message, summarized in Figure 1.

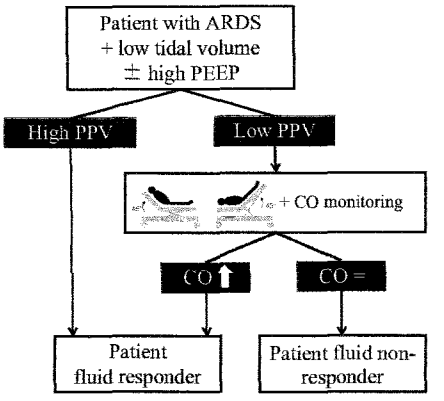

Figure 1. How to use pulse pressure variation in acute respiratory distress syndrome (*ARDS*) patients ventilated with a low tidal volume (at any level of positive end-expiratory pressure [*PEEP*]). *CO*, cardiac output; *PPV*, pulse pressure variation.

In ARDS patients ventilated with low tidal volume (and at any level of PEEP), a high PPV is almost always indicative that the patient will be responsive to fluid administration. However, a low PPV does not exclude the possibility of a positive response. In this clinical situation, it is wise to perform a passive leg raising maneuver while monitoring cardiac output continuously. Such a maneuver is reversible, mimics the effects of fluid loading, and has been shown to be very accurate to predict fluid responsiveness (15). If a significant increase in cardiac output is observed during the passive leg raising maneuver, the patient should respond to fluid loading. If there is not a significant increase in cardiac output, the patient should be a nonresponder and giving fluid would definitely be a mistake.

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Combining audiovisual feedback and debriefing: Learning or just imitating?*

An ancient Chinese proverb states, "I hear and I forget, I see and I remember, I do and I understand." In this issue of *Critical Care Medicine*, Dine et al. (1), at the University of Pennsylvania, have explored the use of immediate verbal feedback, audiovisual feedback, and the combination of both forms of feedback to improve the quality of cardiopulmonary resuscitation (CPR) among nurses. An in-hospital cardiac arrest is a situation ripe for error due to the combination of a dying patient, a frequently incomplete history, time-critical decisions, concurrent tasks, and involvement of several different disciplines. In addition to the complexity of this situation, the usual clinical duties and paperwork after a cardiac arrest, make providing educational feedback to the nursing staff difficult. Good-quality CPR has been shown to significantly improve survival (2–4), unfortunately however, the quality of CPR is highly variable and frequently poor (5, 6). Therefore, Dine's focus on deliberate practice of a single skill and a relatively simple intervention for improving the quality of CPR should be loudly applauded.

Previous studies have found that pronouncement of a learner's competence in performing CPR is entirely dependent on the instructor's judgment and that these judgments are not precise or accurate enough to ensure valid assessments (7). Further, videotape review of CPR practice sessions found that instructors overlooked many errors in CPR performance and that fellow trainees provided little corrective feedback to one another (8). Given the questionable effectiveness of the CPR instructor alone, use of advancing technology to supplement real-time feedback may have a significant role in not only improving the quality of CPR but also survival. In a single, prospective study, of out-of-hospital cardiac arrest, real-time–automated feedback was associated with an increased short-term survival (9).

The results of this single study are encouraging and the work of Dine et al. is an excellent first step, however, application of simulation and advanced technology may not necessarily translate into long-term learning or improved independent performance. At this early stage, the balance of literature on the use of simulation in the critical care setting is descriptive, focused on the perceptions of the learner, or limited to only short-term results.

While the authors of this manuscript have taken simulation to the next level by exploring which type of feedback yields the best short-term results, a more pertinent and important question is "Does this strategy yield durable results in the long term?" Once this training has been completed and the nurses are back on the job; the question remains: did the training work? The type of feedback provided to the study participants in this work was immediate and specific. The belief that greater specificity in feedback leads to improved performance and learning has become an accepted generalization, despite a lack of evidence to support the argument (10). Although it has been shown that immediate, specific feedback is beneficial for short-term performance, its benefits do not endure over time or modification of the task (10, 11). In essence, the trainee is capable of imitation in the short term but has not truly learned the skill. As a result, the trainee runs the risk of becoming dependent on real time feedback to perform at the expected level and may be unable to per-

***See also p. 2817.**

Key Words: education; feedback; cardiopulmonary resuscitation; debriefing

The authors have not disclosed any potential conflicts of interest.

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REVIEW

VOLUME MANAGEMENT IN CRITICALLY ILL PATIENTS: NEW INSIGHTS

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Lopes MR, Auler JOC, Michard F. Volume management in critically ill patients: new insights. Clinics. 2006;61(4):345-50.

In order to turn a fluid challenge into a significant increase in stroke volume and cardiac output, 2 conditions must be met: 1) fluid infusion has to significantly increase cardiac preload and 2) the increase in cardiac preload has to induce a significant increase in stroke volume. In other words, a patient can be nonresponder to a fluid challenge because preload does not increase during fluid infusion or/and because the heart (more precisely, at least 1 of the ventricles) is operating on the flat portion of the Frank-Starling curve. Volumetric markers of cardiac preload are therefore useful for checking whether cardiac preload effectively increases during fluid infusion. If this is not the case, giving more fluid, using a venoconstricting agent (to avoid venous pooling), or reducing the intrathoracic pressure (to facilitate the increase in intrathoracic blood volume) may be useful for achieving increased cardiac preload. Arterial pulse pressure variation is useful for determining whether stroke volume can/will increase when preload does increase. If this is not the case, only an inotropic drug can improve cardiac output. Therefore, the best option for determining the usefulness of, and monitoring fluid therapy in critically ill patients is the combination of information provided by the static indicators of cardiac preload and arterial pulse pressure variation.

KEYWORDS : Delta PP. Systolic pressure variation.

The decision-making process concerning volume expansion is frequently based on the clinical examination and the assessment of cardiac preload indicators, mainly cardiac filling pressures.[1] There is no doubt that in some cases (eg, hemorrhage or severe diarrhea) we can reasonably rely on clinical examination to identify patients who will benefit from fluid loading. However, in more complex—but not uncommon—situations (eg, septic shock) both clinical examination and indicators of cardiac preload have been shown to be of minimal value in answering the question: "can we improve cardiac output and hence hemodynamics by giving fluid?"[2]

Over the past 6 years, many clinical studies have demonstrated the value of arterial pulse pressure variation (ΔPP) to predict fluid responsiveness (ie, an increase in cardiac output as a result of fluid infusion) in sedated patients whose lungs are being mechanically ventilated[3-10] (Figure 1).

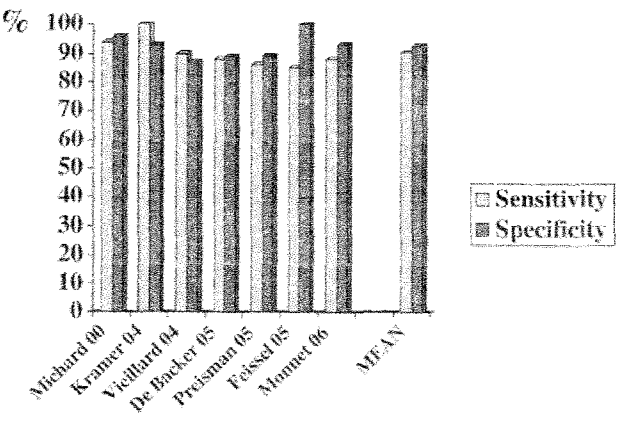

Figure 1 - Sensitivity and specificity of arterial pulse pressure variation (ΔPP) for discriminating between responders and nonresponders to fluid administration.

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The reliability and availability of ΔPP—now automatically calculated and displayed on bedside monitors—and the minimal value of classical markers of preload in predicting fluid responsiveness necessarily raise the following question: "Should we continue to assess cardiac preload to determine fluid therapy?"

Therefore, we will first review the reasons why cardiac preload indicators are poor predictors of fluid responsiveness, and secondly we will describe the circumstances in which they may still be useful for the clinician.

Static indicators of cardiac preload and fluid responsiveness

The little value of cardiac filling pressures in assessing cardiac preload. In many clinical situations, cardiac filling pressures do not accurately reflect cardiac preload for at least 3 reasons. First, measuring cardiac filling pressures is not always easy. Indeed, several studies have demonstrated that observer variability in wedge pressure measurements is of potential clinical importance.[11-13] Second, the pressures that are carefully measured at end-expiration are frequently higher than transmural pressures.[14] This is the case in patients with airflow limitation (autoPEEP), in patients ventilated with an external PEEP, and in patients with intra-abdominal (and hence intrathoracic) hypertension (Figure 2). Third, the relationship between ventricular end-diastolic pressure and end-diastolic volume is not linear, but rather it is curvilinear and unpredictable since it depends on ventricular compliance and thus varies from one patient to another.[15,16] In summary, because in practice we usually measure with poor reproducibility the nontransmural pressures that are not correlated with ventricular dimensions, we cannot rely on these measurements to accurately assess cardiac preload (Figure 2). This is why several volumetric parameters have been proposed to improve the accuracy of cardiac preload assessment at the bedside.

The limited value of volumetric markers of preload for predicting fluid responsiveness. The volumetric indicators of cardiac preload are mainly the right ventricular end-diastolic volume (RVEDV), which is evaluated by specific pulmonary artery catheters;[17-19] the left ventricular end-diastolic area (LVEDA), which is measured by transthoracic or transesophageal echocardiography;[20] and the global end-diastolic volume (GEDV) and intrathoracic blood volume (ITBV), which are evaluated by transpulmonary thermodilution.[21-24] Several studies have demonstrated that these volumetric parameters can be use-

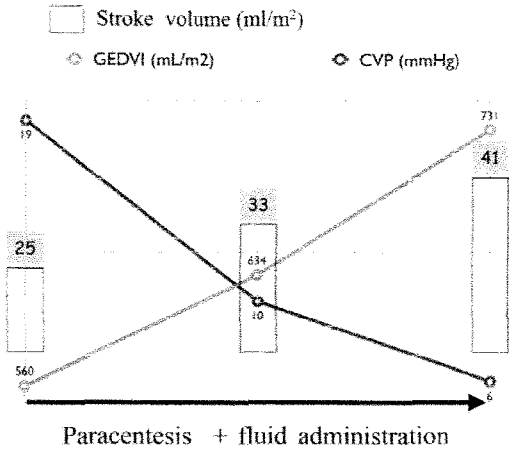

Figure 2 - In this patient with ascites and abdominal hypertension, the central venous pressure (CVP) is not useful for assessing cardiac preload, nor for tracking changes in preload: CVP is high while global end-diastolic volume index (GEDVI) is low, and CVP decreases while GEDI and stroke volume are going up.

ful for predicting fluid responsiveness—but only when they are very low or very high.[17,18,24] For example, it has been shown that the rate of positive response to a fluid challenge is high when the RVEDV index is below 90 mL/m², but low when the RVEDV index is greater than 140 mL/m².[17,18] Similar findings have been recently reported with the GEDV index, which reflects the volume of blood contained in the 4 heart chambers during diastole. When the GEDV index is below 600 mL/m², a positive response to a fluid challenge is very likely; in contrast, when the GEDV index is greater than 800 mL/m², a positive response is very unlikely.[24] However, in all these studies, intermediate values are not more predictive than a random guess.

These findings are quite consistent with cardiac physiology, since the slope of the relationship between preload and stroke volume depends on contractility. Therefore, the pre-infusion cardiac preload is not the only factor influencing the response to a volume load. As illustrated in Figure 3, the increase in stroke volume, which occurs as a result of a rise in preload, depends more on the slope of the Frank-Starling curve than on cardiac preload.[25] In summary, from a physiological point of view, assessing cardiac preload—even with volumetric parameters—cannot be really useful for predicting fluid responsiveness.

Why assess volumetric indicators of cardiac preload? Arterial pulse pressure variation (ΔPP) has been shown to be very useful in predicting fluid responsiveness because it provides the clinician with valuable information concerning the position on the Frank-Starling relation-

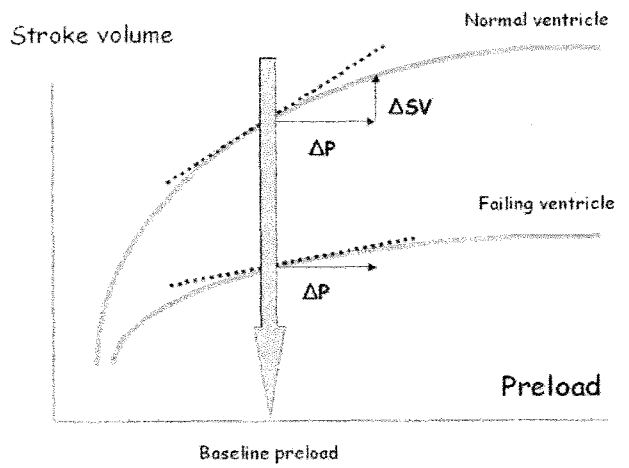

Figure 3 - Schematic representation of the ventricular preload/stroke volume relationship of a normal and a failing ventricle. The increase in stroke volume (ΔSV) as a result of cardiac preload increase (ΔP) depends on ventricular function shown by the slope of the curve (dotted line); assessing baseline preload is not useful in predicting ΔSV.

ship.[26] If the pulse pressure, which is directly proportional to stroke volume, varies during a mechanical breath, it means that the patient's heart is sensitive to changes in preload induced by mechanical insufflation, ie, that both the right and the left ventricles are operating on the steep portion of the Frank-Starling curve.[26] However, ΔPP works only if we assume that 1) respiratory changes in pleural pressure are sufficient to induce significant changes in preload and 2) fluid infusion significantly increases cardiac preload. These two conditions are not always met, and hence the static indicators of cardiac preload remain appropriate in the decision-making process concerning volume expansion.

Limitations of arterial pulse pressure variation (DPP). In a patient sensitive to changes in preload (i.e., one whose heart is operating on the steep portion of the Frank-Starling relationship), the arterial pulse pressure will vary over a single mechanical breath only if preload varies. Since respiratory changes in preload are induced by changes in pleural pressure, in patients ventilated with a low tidal volume (6 mL/kg for example), the respiratory changes in pleural pressure may not be sufficient to induce significant changes in preload.[7,26] Therefore, in this context, the absence of respiratory variations in arterial pulse pressure does not mean that the patient is insensitive to changes in preload (and hence would be nonresponder to a fluid challenge) but simply that preload does not vary during the respiratory cycle. In this regard, ΔPP has been validated as an accurate predictor of fluid responsiveness mainly in deeply sedated mechanically ventilated patients having a tidal volume ≥ 8 mL/kg; therefore, it cannot currently be recommended as a clinical tool in other situations.[26] Moreover, ΔPP cannot be used in patients with cardiac arrhythmias.[27] Therefore, when ΔPP cannot be used, the assessment of cardiac preload may be useful for predicting fluid responsiveness if very low or very high values of RVEDV (< 90 or > 140 mL/m^2), LVEDA (< 5 or > 20 cm/m^2), ITBV (< 7 50 or > 1000 mL/m^2) or GEDV (< 600 or > 800 mL/m^2) are observed. But it must be emphasized once again that intermediate values of preload are not useful for predicting fluid responsiveness.

Fluid therapy does not always increase cardiac preload. One might assume that a fluid challenge systematically and necessarily induces a significant increase in cardiac preload and that it is only necessary to assess the position on the Frank-Starling curve (using ΔPP) to accurately identify patients who will benefit from a fluid challenge—but this is not the case. Fluid infusion increases intravascular blood volume (at least transiently) but not necessarily cardiac preload. Axler et al[28] studied the hemodynamic effects of 159 "typical rapid volume infusions" in critically ill patients and did not observe any significant increase in left ventricular preload as assessed by the measurement of LVEDA using echocardiography. The increase in ventricular end-diastolic volumes as a result of fluid infusion depends on the partitioning of the fluid into the different cardiovascular compartments organized in series. When the heart is poorly compliant, giving fluid may not increase cardiac preload. Ventricular compliance can be decreased because of ischemic cardiopathy or simply because the ventricles are already dilated. These physiological or rather mechanical considerations are supported by several clinical studies. In critically ill patients receiving fluid, when the right ventricle is already dilated (RVEDV index greater than 140 mL/m^2), fluid infusion does not increase right ventricular dimensions. In contrast, when the RVEDV index is below 140 mL/m^2, the same fluid regimen leads to a significant increase in RVEDV.[17] Similar findings have been reported with the GEDV.[24] In patients with septic shock receiving the same amount of fluid (500 mL of colloid) over a short period of time (20-30 minutes), we observed various responses in terms of GEDV increase.[24] These findings support the notion that a standardized fluid challenge does not induce the same increase in preload in all patients. In addition to ventricular compliance and dimensions, factors like mean intrathoracic pressure (by acting on the intra/extrathoracic repartition of the total blood volume) or

venous capacitance/pooling (usually increased in sepsis) may also play a role in the partitioning of the fluid infused. Therefore, during a fluid challenge, assessing preload is definitely useful for checking whether preload effectively increases. If preload does not increase (for any reason), an increase in cardiac output cannot be expected even if the heart is operating on the steep portion of the Frank-Starling curve.

RESUMO

Lopes MR, Auler JOC, Michard F. Avaliação da volemia em pacientes críticos: nova proposta. Clinics. 2006; 61(4):345-50.

Para ser efetivo em aumentar significativamente o volume sistólico um volume de fluido precisa preencher duas condições :

1- A infusão deste fluido tem que aumentar a pré-carga
2- O aumento da pré-carga tem que promover uma elevação proporcional do volume sistólico
Em outras palavras o paciente pode ser não responsivo à infusão de volume em termos de volume sistólico, devido a quantidade de fluidos ainda não ser a necessária ou o coração já estar operando na faixa superior da curva de

Frank-Starling. Os indicadores volumétricos da pré-carga cardíaca são úteis para verificar se esta pré-carga aumenta efetivamente durante a infusão de fluido. Em caso negativo, ou seja a pré-carga não aumenta, medidas adicionais como mais fluidos, venoconstrictores para aumentar o quantidade de sangue, ou aumento do retorno venoso por redução da pressão intratorácica podem ser efetivas para atingir a primeira condição: aumento da pré-carga. Delta PP pode ser útil para verificar se o volume sistólico aumenta com a infusão de fluidos. Isto não acontecendo somente drogas inotrópicas podem aumentar o débito cardíaco. Portanto, combinando os indicadores estáticos da pré-carga (PVC, pressão capilar pulmonar) com Delta PP, consegue-se a melhor opção para monitorar a resposta aos fluidos em pacientes críticos.

UNITERMOS: Variação de pressão arterial (ΔPP). Reação a fluidos. Pré-carga cardíaca. Débito cardíaco.

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