Volume Management Using Dynamic Parameters

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is probably a more realistic end point to be sought. Treatment failure in severe HAP may be found in ≥ 50% of cases[15] and is also associated with higher mortality, length of hospital stay, and costs. However, death is not observed in all patients presenting treatment failure. Future studies should include a reduction in treatment failure as a desirable end point to be achieved on the implementation of guidelines.

In summary, at least concerning respiratory infections, guideline implementation is necessary to provide the best medical care for our patients and to improve their outcome. Studies such as that published in the present issue of *CHEST* on guideline validation are necessary. Nonetheless, the optimal methodology to carry out these studies still requires standardization.

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The Good, the Bad, and the Ugly

In the August issue of *CHEST*, Hofer et al[1] place another brick in the wall of clinical studies by demonstrating the superiority of dynamic parameters over static indicators of cardiac preload in predicting fluid responsiveness.[2]

The Good

The concept of fluid responsiveness has become very popular over the last few years, likely because this is a very pragmatic approach to fluid therapy. Indeed, we have a clear idea of the normal total blood volume (70 to 80 mL/kg), intrathoracic blood volume (800 to 1,000 mL/m^2), and right and left ventricular end-diastolic volumes (90 to 110 mL/m^2 and 60 to 80 mL/m^2, respectively) in healthy subjects. However, it is much more difficult to determine which level of preload is optimal in an "abnormal" situation (*eg*, vasodilation induced by anesthetic agents or sepsis). Therefore, to determine fluid therapy, a very practical approach consists in detecting patients who will turn fluid loading into a significant increase in stroke volume and cardiac output. Of course, clinical end points of fluid therapy are usually different (*eg*, increasing BP, or urine output) but will be achieved only if the physiologic effect (an increase in stroke volume and cardiac output according to the Frank-Starling mechanism) first occurs. If not, fluid administration is useless or even potentially harmful (worsening in pulmonary edema).

In the study by Hofer et al,[1] left ventricular end-diastolic area (LVEDA) assessed by echocardi-

ography, global end-diastolic volume (GEDV) assessed by transpulmonary thermodilution, stroke volume variation (SVV) assessed by pulse contour analysis, and arterial pulse pressure variation (PPV) were collected before and immediately after a standardized volume challenge in patients undergoing off-pump coronary artery bypass grafting. Measurements were performed before sternotomy in patients sedated, paralyzed, and receiving mechanical ventilation with a tidal volume of 10 mL/kg. Both LVEDA and GEDV behaved as indicators of cardiac preload: they increased with fluid loading, and their changes were proportional to changes in stroke volume. However, preinfusion LVEDA and GEDV values were not related to the hemodynamic effects of fluid administration. These findings are consistent with previous reports[3–5] and with cardiac physiology: an increase in stroke volume as a result of an increase in preload depends more on the slope of the Frank-Starling curve than on baseline cardiac preload.[6] In contrast, the higher PPV and SVV before fluid administration, the more marked was the increase in stroke volume as a result of fluid administration. Both PPV and SVV reflect the sensitivity of the heart to phasic changes in loading conditions induced by mechanical inspiration[6] and have already been shown to be accurate predictors of fluid responsiveness.[8–13] However, as far as I know, the study by Hofer et al is the first comparing PPV to SVV.

The pulse pressure (systolic minus diastolic pressure) depends not only on stroke volume but also on arterial compliance.[14] In other words, the same PPV may theoretically result from large swings in stroke volume in compliant arteries or smaller swings in stroke volume in stiff arteries. Therefore, PPV is expected to be a less accurate predictor of fluid responsiveness than SVV. Interestingly, Hofer et al did not report any significant difference between the value of PPV and SVV in predicting fluid responsiveness but regrettably did not comment on this issue. One can reasonably assume that the physiologic weakness of PPV (its dependency on arterial compliance) may be compensated by the precision in measuring this parameter. Indeed, measuring systolic and diastolic pressures (and hence calculating pulse pressure) is much more simple than measuring stroke volume from pulse contour analysis.[15] It may result in a better precision when assessing PPV than when assessing SVV, which may explain, at least in part, why SVV was not found to be a better predictor of fluid responsiveness than PPV.

The Bad

Dynamic parameters have some limitations that prevent their use in all circumstances. First, because in clinical practice the arterial pressure curve is obtained from fluid-filled catheters, several factors (air bubbles, kinks, clot formation, compliant tubing, excessive tubing length) may distort the signal. This problem can be ruled out by a "fast-flush test"[16] but requires first careful examination of the BP curve by a caregiver before relying on PPV and SVV values. Second, in patients with cardiac arrhythmia, the beat-to-beat variation in stroke volume and hence in BP may no longer reflect the effects of mechanical ventilation. This is particularly true in patients with atrial fibrillation or frequent extrasystoles. In patients with few-and-far-between extrasystoles, the arterial pressure curve can still be analyzed if the cardiac rhythm is regular during at least one respiratory cycle. However, it definitely rules out the possibility of a continuous and automatic monitoring of this phenomenon, like done in the study by Hofer et al.[1] Third, if pleural pressure changes are small over a single respiratory cycle, inspiration will not induce any significant change in left ventricular stroke volume, even in fluid-responsive patients. Small variations in pleural pressure may be observed in patients with spontaneous breathing activity, in patients receiving mechanical ventilation with small tidal volumes (*eg*, 6 mL/kg), or in patients with increased chest compliance (*eg*, open chest). In this context, caution should be exercised before concluding that a patient will not respond to a fluid challenge because PPV or SVV are low.

The Ugly

First, when limitations mentioned above are not understood or/and respected, the assessment of dynamic parameters may become more harmful than helpful in the decision-making process regarding volume expansion. Mistakes and misinterpretations in measuring or using dynamic parameters should be avoided by education and teaching. The pulmonary artery catheter has been used for > 30 years, and studies have clearly shown that physicians' knowledge of the device is quite insufficient.[17] We should use lessons from the past and not waste the clinical value of "new" parameters (like PPV and SVV) by improper use. In the near future, the development of interactive monitoring systems could also help the caregiver to check that all conditions are met to correctly use and interpret the parameters displayed on bedside monitors. Second, fluid responsiveness does not mean that fluid is needed. We are all—as healthy subjects—fluid responsive.[18] Fortunately, it does not mean that we need volume expansion! I have still in mind a patient with hiccups (and hence significant swings in pleural pressure) who did re-

ceive a lot of fluid because his PPV was elevated. Patients should not receive fluid simply because the PPV or SVV is high. Indeed, before using dynamic parameters, the first question must be, "does my patient need an increase in stroke volume or in cardiac output?" Definitely, dynamic parameters will never answer this question. The answer may lie in clinical examination (mottling and oliguria), biological tests (*eg*, renal failure and high lactate level), or/and other hemodynamic parameters (*eg*, low cardiac output and low mixed venous oxygen saturation). However, when the answer to this question is "yes," and if there is no contraindication to fluid loading (severe pulmonary edema and hypoxemia), dynamic parameters can be very useful to discriminate between patients who may benefit from volume loading and patients in whom inotropic support is a more logical approach to improve hemodynamics.

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