Role of CVP to Guide Fluid Therapy in Chronic Heart Failure
Lessons From Cardiac Intensive Care

We read with great interest the article by Qian et al. (1) on central venous pressure (CVP)-guided fluid administration for the prevention of contrast-induced nephropathy (CIN). The authors used the initial and subsequent change in CVP to guide fluid administration before elective coronary angiography in heart failure (HF), calling CVP “a safe and accurate assessment of intravascular volume status.” Higher volumes of administered fluid were associated with greater urine output and lower CIN risk, without an increase in HF exacerbation. Irrespective of CVP-guided hydration, patients with a baseline CVP >12 cm H2O received the same amount of fluids and had the highest CIN rates, implying that pre-existing congestion promotes CIN and that fluid volume, rather than CVP monitoring per se, produces the benefit. This leaves uncertainty as to whether patients in both groups would have done equally well if they had received equal amounts of fluids, independent of the CVP, which could have served to encourage a fluid-liberal strategy.

Use of CVP to guide fluid resuscitation is fraught with inconsistencies, with accumulating data suggesting that CVP is a poor reflection of volume status, fluid responsiveness, and pre-load in cardiac intensive care patients (2,3). CVP, a reflection of right atrial pressure (RAP), is subject to variability from intrathoracic structures, valvular abnormalities, and pulmonary vascular disorders (2,3). Physiological studies have demonstrated that RAP actually opposes venous return (the difference between mean systemic filling pressure and RAP). Although the CVP generally correlates with the pulmonary capillary wedge pressure and/or left ventricular end-diastolic pressure in HF, this relationship requires normal biventricular compliance, integrity of atrioventricular valve function, and normal pulmonary hemodynamics (4). Pulmonary hypertension and severe mitral and tricuspid regurgitation are noted in nearly 60% to 90%, 50%, and 30%, respectively, of patients with HF with reduced ejection fraction (5). HF patients frequently have dilated right atria with abnormal chamber compliance affecting the pressure-volume relationship (3). The present study does not comment on these confounding factors to the assumption of volume status or fluid responsiveness using the CVP.

There are no standardized cutoffs for static CVP measurements in patient management, as noted in a meta-analysis of studies evaluating CVP in different settings (2). The classic “5-2 rule” using CVP to predict fluid responsiveness has been challenged over the years. Therefore, the use of arbitrary CVP cutoff values of 6 and 12 cm H2O by the authors needs further definition of the strategy and rationale to arrive at these numbers.

Given these considerations, we believe that the conclusions of the authors (1) that CVP predicts fluid status and guides strategies to prevent acute decompensation of HF need further elaboration and investigation before establishing causation. In critically ill patients, the role of static measures of fluid status and/or responsiveness (such as CVP and pulmonary capillary wedge pressure) are increasingly being replaced by dynamic measures. Importantly, given the risk associated with invasive CVP measurement, we wonder whether a noninvasive measure such as echocardiography could have yielded similar results without the potential for vascular access complications.
An Effective and Safe Hydration Method for the Prevention of Contrast-Induced Nephropathy

We appreciate the comments of Vallabhajosyula et al. on our clinical trial. Although there are some confounding factors when evaluating volume by central venous pressure (CVP), low CVP always indicates hypovolemia, which is affected by few confounding factors. In our study, 73.9% of patients (195 of 264) had initial CVP less than 12 cm H₂O, and 19.7% of patients (52 of 264) had initial CVP less than 6 cm H₂O. We tried to improve insufficient blood volume status using aggressive hydration. Because hypovolemia is an essential risk factor for contrast-induced nephropathy (CIN), patients with the lowest CVP (<6 cm H₂O) received the greatest benefit of CIN prevention from CVP-guided vigorous volume expansion (1). Rapid infusion guided by CVP could help avoid dehydration and maintain stable blood volume.

Patients with heart failure usually do not receive adequate hydration in routine clinical practice, because of concern for pulmonary edema. We aimed to explore a “safe” hydration method to avoid fluid overload. Fluid infusion rate was dynamically adjusted by CVP in our clinical trial. Although there are some disputes regarding CVP-guided fluid therapy, recent researches have indicated that lower and higher CVP values had positive and negative predictive value, respectively, for fluid responsiveness (2). In our study, 52 patients in the CVP-guided hydration group showed obvious increases in CVP and thus reductions in infusion rate. Aggressive volume expansion did not increase the incidence of pulmonary edema in our trial. To prevent acute heart failure, the fluid infusion rate should be determined by comprehensive indicators (such as dyspnea, pulmonary edema, change in brain natriuretic peptide, etc.), rather than a single hemodynamic index.

Invasive central venous catheterization is extensively applied in clinical practice for rapid fluid resuscitation and hemodynamic monitoring. Left ventricular end-diastolic pressure-guided hydration seems to be effective in preventing CIN, but it could not dynamically monitor the change in left ventricular end-diastolic pressure during hydration (3). Noninvasive dynamic measure technologies have not been carried out widely in clinical application. Hydration guided by noninvasive dynamic measures may be a good approach to the prevention of CIN, which requires further investigation in a prospective randomized controlled trial.

REFERENCES

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REFERENCES