pressure range. In addition, the failure to find correlations between CVP and indexes of fluid responsiveness might be influenced by ongoing fluid losses or gains that were not necessarily easily measurable. Finally, in many clinical situations, the success or failure of fluid resuscitation measures is not determined by preset goals in terms of cardiac output or other indexes of cardiac performance but by criteria such aspressor requirements or blood lactate concentrations. While the current recommendations guiding fluid replacement use CVP as only one of a number of clinical parameters to follow, and while dynamic parameters may have an increasing role in guiding fluid replacement, it may be premature to discard CVP measurements in ICU patients.

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Central Venous Pressure as Popular Resuscitation Surrogate

Not Totally Unjustified

To the Editor:

Marik and colleagues, using statistical analysis and data from controlled studies, show that isolated central venous pressure (CVP) measurements are a poor guide to the assessment of volume status and fluid responsiveness. They called attention to a common myth that CVP is a good guide to fluid management. It is an interesting reminder of related misconceptions in classical works of cardiovascular physiology. For instance, the experiments of Guyton and Starling are commonly misunderstood. The pump is commonly seen as the main orchestrator of output. Guyton’s experiments demonstrated the role of peripheral vasculature in controlling cardiac output. Physicians commonly mistake venous inflow to the atria as the determinant of left ventricular stroke volume. Starling, working on denervated hearts, showed that atrial filling and thus “ventricular wall stretch” was the primary determinant of stroke volume. Perhaps these misconceptions are a result of confusion between the cause and the consequence.

CVP is affected by a myriad of intrinsic and extrinsic factors including IV fluids, positioning, intrathoracic pressures, heart rate, contractility, and myocardial and venous compliance, among others. Further confusing the issue is discrepancy between normal physiology and disease state pathology. As an example, sepsis alters total effective vascular compliance. Thus while CVP reflects mean right atrial pressure, it is not expected to show an independent correlation with effective circulatory blood volume.

Apprently, it is the intuitive simplicity of CVP that attracts a new learner as compared to the lack of receptiveness encountered with terms such as upstream and downstream resistance, flow limitation, and capacitance. The CVP-centric practice may also be widespread because it comes with convenience and there are no controlled studies that prove definite disadvantage related to the method use for fluid management. The 2008 Surviving Sepsis Campaign guidelines say “… recommend fluid resuscitation initially target a CVP of at least 8 mm Hg.” Justification of this statement is mostly based on a single study. The attempt at getting at least one of the determinants (CVP in this case) of cardiac output right, ie, achieving physiologic limits, may be a reasonable approach toward attempting to optimize perfusion in critically ill patients.

To conclude, we feel that the findings reported by Marik and colleagues are not unexpected. CVP remains meaningful along with the other parameters (BP, heart rate, urine output, extra heart sounds, cool extremities, fractional excretion of sodium) that are used by experienced clinicians to assess volume status.

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Response

To the Editor:

We thank Drs. Cole and Singh and colleagues for their thoughtful comments. We agree on most points. Decisions regarding fluid management are among the most difficult in clinical medicine in general and in the ICU in particular. We believe that this decision should be based on an indepth understanding of the disease process and treatment strategy, with a review of fluid balance, oxygenation index, urine output, chest radiograph, and renal function (and