Letter to the editor: Comments on “Value and determinants of the mean systemic filling pressure in critically ill patients”

George L. Brengelmann
Department of Physiology and Biophysics, University of Washington, Seattle, Washington

TO THE EDITORS: In their recent article published in the American Journal of Physiology-Heart and Circulatory Physiology, Repessé et al. (8) recently reported on intravascular pressures [mean systemic filling pressure (Pmsf)] recorded shortly after death in patients in intensive care. With a broad dispersion around an average near 12 mmHg, the only clear influence on Pmsf that could be seen was elevation when norepinephrine had been infused before death.

Their motives for focusing on Pmsf were “its crucial role as the upstream pressure of systemic venous return” and “physiological understanding of venous return may help intensivists to improve management of critically ill patients.” Although I would heartily support the latter, I believe the former stands in the way of understanding.

This pressure, Pmsf, found when flow through the vasculature is zero is appropriately viewed as the distending pressure for the aggregate vascular container. Notwithstanding the complexity of the vascular architecture and of the elastic state of its vascular segments, we see Pmsf as the distending pressure of a single elastic container filled with the blood volume. As observed by Repessé et al. (8) in the presence of a vasoactive substance that increases tone in vascular smooth muscle, we certainly expect a higher Pmsf, other things remaining constant, especially total intravascular volume.

As Repessé et al. (8) emphasize, the measurement of Pmsf is a snapshot taken in a dynamic situation; active tension in vascular smooth muscle will decline as will pressure. The assumption is that when measured soon enough after cessation of flow, intravascular pressure will reflect something important about the peripheral vasculature.

But what? The driving pressure for venous return?

In the definitive work of Guyton et al. (5, 6), Pmsf, revealed as the zero-flow intercept of a given venous return curve, was shown to increase when total blood volume was increased. It was not right atrial pressure (Pra) that they varied to obtain varied levels of flow through the vasculature, but the height of a Starling resistor that throttled flow to bring about a desired Pra; that is, they set the flow through the vasculature to the level that would produce the desired Pra in the obligatory inverse relationship between steady-state flow and Pra for a given blood volume (3).

Guyton et al. (5) reproduced the form of venous return curves with mathematical models with resistors representing vascular segment resistances and capacitors representing vascular segment compliances. But, in their model, no capacitance representing an elastic compartment remained at Pmsf while flow (cardiac output/venous return interchangeably) varied.

At any condition other than zero flow, the distribution of the volume among compliant segments of the vasculature changes in relation to the altered distending pressures that develop with altered flow-related pressures [for a lucid description of this process, see Chapter 3 in Rowell’s monograph (7)]. Pmsf, as recorded by Guyton et al. (5, 6), was simply a proxy for total volume.

For clinical assessments, why not estimate actual volume directly (readily done with dye dilution), rather than seek to estimate Pmsf? Better yet, think not of dropping Pra to improve venous return but of improving cardiac output and/or the maldistribution of volume [with the obligatory consequence of lower Pra (3)].

The present study highlights the error in thinking that Pmsf represents the steady-state pressure head that drives venous return, opposed by the “back pressure” at the right atrium, Pra. The only steady state at which Pmsf is meaningful is constant volume, zero flow. If Repessé et al. had opened the right atrium to atmospheric pressure, say by disconnecting the venous catheter from the pressure transducer, there would have been a brief period of flow, dwindling with the decline of pressure as volume left the vasculature, not steady-state flow driven by Pmsf.

The argument against my assertion that Pmsf cannot be the upstream pressure driving steady-state venous return might be that Pmsf depends on the volume within the vasculature. For steady-state flow, blood volume would have to be returned to the vasculature to keep total circulating volume, and thus Pmsf, constant.

Indeed, maintaining steady-state flow through the vasculature with fixed total volume requires a pump. It is the mechanical energy expenditure of the pump, the heart, reflected by the difference between aortic pressure and Pra, which maintains steady-state flow. Just how the total volume is distributed among the myriad segments of the vasculature varies with flow (7), so does the point at which the pressure profile along a particular pathway from aorta to right atrium intersects Pmsf.

We need to get rid of the idea that some large compartment which determines venous return but of improving cardiac output and/or the maldistribution of volume among compliant segments of the vasculature changes in relation to the altered distending pressures that develop with altered flow-related pressures [for a lucid description of this process, see Chapter 3 in Rowell’s monograph (7)]. Pmsf, as recorded by Guyton et al. (5, 6), was simply a proxy for total volume.

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AUTHOR CONTRIBUTIONS
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REFERENCES

Address for reprint requests and other correspondence: G. L. Brengelmann, Dept. of Physiology and Biophysics, Univ. of Washington, Box 357290, Seattle, WA 98195 (e-mail: brengelm@u.washington.edu).

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