Reply to “Letter to the editor: Why persist in the fallacy that mean systemic pressure drives venous return?”

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REPLY: We thank Dr. Brengelmann for his comments (2) to our recent article (1) appearing in the American Journal of Physiology-Heart and Circulatory Physiology. Brengelmann highlights the controversies on Guyton’s concepts of venous return. He raises 10 important issues.

First, how Guyton did his experiments. We do not disagree with this description. Our study was not designed to resolve the debate on Guyton’s (4) versus Levy’s (5) interpretation. We studied venous return in a model relevant to translational application of physiology in clinical practice: a preparation with beating heart and intact vasculature exposed to transients resulting from heartbeats and intrathoracic pressure variation.

Second, whether right atrial pressure (Pra) acts as backpressure to venous return or whether it only responds passively to volume shifts when flow changes: As explicitly stated in our article, we did not try to solve this central point in the debate between proponents of Guyton and those of Levy. Tyberg acknowledged that both models are internally consistent and perhaps impossible to prove one against the other (13). In our experiment, transients in vascular resistances, shifts between the stressed and unstressed volume, intrathoracic and intrapericardial pressures, atrial compliance, biventricular performance, and Pra interact. Although we modified Pra by airway pressure, the focus was not the backpressure debate.

Third, Dr. Brengelmann claims that we believe that mean systemic filling pressure (MSFP) “exists physically within some significant subcompartment at the upstream end of the venous resistance...” (2). We never made such a claim. We explicitly state that the “MSFP is not located in any particular subdivision but represents the stressed volume of the entire systemic vasculature. We consider it as the weighted mean of elastic recoil pressures in all systemic vascular beds, as measured after venous pressure equilibration during zero flow induced by right atrial occlusion. It will change if volume shifts alter the stressed volume or if vascular elastance changes” (1).

Fourth, elastic recoil versus passive conduit: Resistance to venous return is the resistance encountered by the average element in the systemic circulation in returning to the heart (4, 12). Dr. Brengelmann’s conclusion that “...one sees MSFP as reflecting the volume within the peripheral vasculature, not a pressure physically present in some actual compartment” (2) should be modified, since MSFP is related to stressed volume. Stressed volume does not disappear under ongoing circulation but contributes to wall tension and therefore pressure in various vascular beds, even in the presence of additional energy supplied by the heart, which of course is necessary to run the circulation. Our data indicate that MSFP during ongoing circulation is subject to adaptive volume shifts from the splanchnic bed. Still, a new, linear steady-state venous return is established, representing the elastic properties of the system (1).

With a beating heart, stressed volume is maintained constant by pulsatile in- and outflow. In contrast to continuous flow from a mechanical pump, the pulsatility allows for small oscillations around MSFP. Because of large vascular compliance, these transients have marginal effect on MSFP (6–8) and may readily be offset by oscillations in right atrial pressure (RAP) during the cardiac cycle and changes in intrathoracic pressure (10, 14). This vascular buffering is also recognized by Dr. Brengelmann (2). The heart can only pump what it gets, and in an intact circulation, the heart is the overall source of energy. The constant refill need should not disguise the emptying mechanics, because they determine the maximum obtainable cardiac output (9).

Fifth, electric and hydraulic models of circulation: This goes beyond our study, but it is addressed elsewhere (8). The same concerns the sixth point: depiction of MSFP for teaching purposes. We acknowledge that such illustrations are oversimplified and may contain conceptual errors in details.

Seventh, overlay of cardiac function and venous return curves and their intercept representing the real Guyton’s model (3): “That is, the concept that peripheral vasculature and cardiac pump subsystems are counterpoised. In one, elevated Pra tends to result in increased” steady-state venous return (F); “in the other, elevated F tends to result in decreased Pra” (2). We fully agree that the intercept of these two curves is fundamental to the “Guytonian” approach to the circulation. We disagree that an increase in Pra tends to increase flow—this is true when Ppr increases as a result of increased stressed volume and a larger increase in MSFP, as in our study (1).

Eighth, the “potential value of an estimate of MSFP obtained by techniques...that do not resort to reducing F to zero” is “in indirectly estimating the volume contained within the vasculature” (2). We would like to emphasize again that the MSFP obtained by any technique reflects the stressed volume and the vascular compliance. The stressed blood volume may and does redistribute very rapidly during changes in vascular tone, and therefore the MSFP also changes independent of changes in total blood volume.

Ninth, MSFP reflects volume, not a pressure, in a specific component. As explicitly stated in our article (1), in our view the MSFP is not located in any particular vascular subdivision but represents the stressed volume and therefore the weighted or aggregate pressure of the entire systemic vasculature.

Tenth, measurement of blood volume for interpretation of “abnormal pressures in central veins could be assessed for

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evidence of maldistribution of volume.” This proposal can be seen as a very indirect way to assess the elastic driving forces in the circulation—estimation of MSFP does the same but more directly. Hence, total blood volume measurement does not provide the relevant information, as recently pointed out by Repessé et al. (11) in their response to Dr. Brengelmann.

We would like to thank Dr. Brengelmann for his constructive comments and his appreciation of our findings that also in our view shed light on how changes in volume of organ vascular compartments buffer temporary discrepancies between cardiac output and venous return.

When MSFP is seen as the aggregate pressure of the systemic vasculature—consistent with Guyton’s initial description (4)—it is perfectly possible to describe the venous return as \( \frac{\text{MSFP} - \text{RAP}}{\text{RVR}} \) during transient states of imbalances (1, 10), where RVR is resistance to venous return. What needs to be left behind is not the “fallacy that venous return is driven by \( \frac{\text{MSFP} - \text{RAP}}{\text{R}_{\text{ven}}} \)” (2), but the explicit restriction to steady states and zero flow conditions.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

D.C.B. drafted manuscript; D.C.B., P.W.M., and J.T. edited and revised manuscript; D.C.B., P.W.M., and J.T. approved final version of manuscript.

REFERENCES