Letters To The Editor

Last Word: Point:Counterpoint author responds to commentaries on “The classical Guyton view that mean systemic pressure, right atrial pressure, and venous resistance govern venous return is/is not correct”

To the Editor: In my Counterpoint (2), space did not permit mention of the dynamics accompanying each heartbeat that Lin Wang (4) focuses on. Of course, diastolic flow is driven by elastic energy stored from previous systoles as is any discrepancy between cardiac output and venous return. But in the (Pms−Pra)/Rv concept, variables are time averaged over multiple heartbeats; this was certainly so in the experiments that introduced us to venous return curves (e.g., Ref. 3). Measurements were made during steady states—brief ones, but steady in the sense that time-averaged flows, volumes, and pressures were not changing [“steady state” does not mean zero flow, as Mitzner (4) seems to suggest]. The energy dissipated against vascular resistance by steady flow passing through the constant volume vascular compartments was supplied by the heart (and pump), not by shrinking elastic vessels. The experiments from which the (Pms−Pra)/Rv concept arose (3) were not about pulsatile dynamics nor venous return:cardiac output imbalances.

Rothe (5) mentions his model. It represents the peripheral vasculature as two resistors in series with two capacitors connected at the nodes. I regret that he represents the venous system with a single resistor (Rsv) and capacitance, thus not showing the redistribution of volume within the venous system that accompanies flow change (and I hope he will expand the model to represent at least 2 parallel organ vasculatures). Nonetheless, like the model I used (1), Rothe’s predicts an inverse flow:Pra relationship. By separating his peripheral vascular components from the heart at the upstream end of the arterial resistor and maintaining total volume constant, one can vary flow and generate the inverse relationship with Pra familiar from venous return curves. One would not interpret this as indicating that venous return is driven from a compartment at fixed Pms acting as the pressure head on the upstream end of Rv, especially because the apparent Rv does not equal his Rsv.

Also, his model of the vasculature will not yield the segment of a venous return curve where flow is constant despite progressively reduced Pra. To do so would require nonlinear resistance elements with properties similar to collapsible blood vessels.

Permutt (4) focuses on this flow limitation phenomenon, undeniably of fundamental importance. I would defer to him for discussion of the far-reaching significance of the finding revealed in the original studies of Guyton et al. (3) that central venous pressures fall toward zero when high levels of flow redistribute volume upstream. However, flow limitation by nonlinear vessel properties is not what (Pms−Pra)/Rv describes; it is about the apparent linear increase in flow with progressive reduction in Pra. That is what led to the misconcept that fixed Pms drives venous return. Although Mitzner may think it trivial to debate what (Pms−Pra)/Rv means, even in this 21st Century it is still associated with confusion and absurdities such as bathtub models for the vasculature and flow driven by elastic chambers at constant volume. I have no intention of throwing out the baby, only the bathtub.

REFERENCES


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