Letter to the Editor

Venous return, mean systemic pressure and getting the right answer for the wrong reason

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Berger and Takala, in their recent article in this journal (1), adhere to the view that venous return (VR), determines cardiac output (CO) because the heart can only pump out what it receives[1]. They see VR as driven by mean systemic pressure (Pms), which is opposed by the pressure at the right atrium (Pra).

These notions descend from the work of A.C. Guyton and colleagues. In 1957, they presented their data on steady-state flow (F) through the vasculature in relation to Pra in the format known as "VR curves" like the plot Berger and Takala included in their figure 1 (2). These reveal an inverse relationship between Pra and F that shifted rightward when they increased the total circulating blood volume, Vtot. It should be remembered that the desired levels of Pra were accomplished through adjustment of the output of the pump that forced F through the vasculature and that the data were recorded in steady states, meaning that dynamic adjustments of volumes and pressures following changes in F were complete.

The inverse relationship between F and Pra duplicates what would be observed if the pressure at the outflow end of a tube connected to an elastic container were varied whilst keeping the pressure within the container constant. For comparison, call the volume held in the container Vtot; the steady pressure within it, Pms; the pressure at the outflow, Pra; the resistance of the connecting tube, Rven; and the outflow rate, VR. The quantitative description of the relationship between these variables would be VR = (Pms – Pra)/Rven.

When elevated to the point that outflow pressure equaled Pms, outflow would be zero. Pms would be in proportion to the elastic stiffness of the container, higher, for a greater Vtot. With progressive reduction of outflow pressure but preservation of Pms, outflow would increase, equal to the pressure difference divided by the resistance of the outflow path. The resistance, Rven, would be revealed as the inverse of the slope of the relationship between F plotted against outflow pressure.

This formal identity between F versus backflow pressure for a single elastic compartment and the vasculature is the rationale behind views listed above. In the context of VR curves, the value of Pra recorded when F is zero is taken as revealing the driving pressure, Pms, within the vasculature. Pms is thought of as physically present within the venous vasculature, kept constant by the steady-state refilling from the cardiac output, and upstream of the effective hydraulic resistance of the venous system. The progressive increase in F associated with reduced Pra is seen as due to reducing the back pressure that opposes VR. The ratio of (Pms – Pra), to F is defined as the resistance to VR.

The (Pms – Pra)/Rven view of peripheral vasculature function has a profound flaw—violation of the principle of conservation of energy. Where does the energy come from that supplies the work done in forcing F through the resistance? An elastic container at fixed distending pressure and volume cannot supply the energy required for the work of driving outflow. It would have to shrink to release the mechanical energy stored in its walls. But, the notion that a steady VR at some fixed Pra is driven by Pms necessitates that Pms remain steady, i.e., that the elastic

[1] A focus that overlooks the fact that neither can the heart receive what it does not pump out when flow is steady.

container with internal pressure kept at Pms be supplied with inflow at exactly the rate that matches the rate at which it is being drained[2]. In other words, what drives steady F, whether regarded as CO or VR, is the work done by the left ventricle, not work from the release of elastic energy stored in distended fibers of the vascular container.

This is not to dispute that Pms reveals important information about the peripheral vasculature. Undeniably, it co-involves the total volume contained within the peripheral vasculature and the elastic properties of its individual segments. To the extent that an individual's peripheral vasculature will behave like those studied by Guyton and others, increasing the blood volume will result in greater cardiac output for a given level of Pra. Regrettably, without independent information about possible changes in segmental elastic properties versus changes in volume, changes in Pms cannot be ascribed to one or the other.

Thinking in terms of how altered pressures associated with altered F obligate altered distribution of volume affords better understanding of the reciprocal relationship between steady-state F and Pra. For example, take the case of an increase in F that occurs without change in segmental resistances or compliances, as in Guyton's experiments. We know the overall difference between arterial pressure and Pra must increase; a steepening of the pressure profile, so to speak. Any segments at higher pressure than before will contain more volume. Those increases obligate reciprocal loss of volume from segments now at lower pressure. Necessarily, therefore, Pra will be lower than at the previous, lower, F, reflecting a loss of volume now transferred to more peripheral compartments now at higher including upstream venous segments and even the arterial system.

This obligatory volume re-distribution has been a feature of physical models of the peripheral vasculature. None of the quantitative models comprised of resistances and capacitances that correctly predict proper "VR" curves includes a compartment kept at a constant pressure equal to Pms, not even the early three-capacitor model of Guyton *et al.* (3) that represented the venous system as comprising two elastic compartments. Though it is mathematically certain that the vascular pressure profile will pass through a value equal to Pms, that does not identify the locus of the drive for VR. What is creating steady F with its associated distributions of pressure and volume in the peripheral vasculature is the work done by the left ventricle.

These comments come from a physiologist who has never dealt with management of a patient in surgery or in critical life support. Perhaps clinicians in such situations have made correct interventions through rationales based on the idea that Pms drives VR. But getting the right answer for the wrong reason is a matter of intuition mixed with luck. Would it not be better to move forward armed with understanding of how blood volume is apportioned among vascular compartments in relation to their pressures?

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Footnote

Conflicts of Interest: The author has no conflicts of interest to declare.

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[2] As their defense against this criticism, Berger and Takala state that critics "*ignore that the emptying mechanics including a highly elastic venous reservoir, are central for the achievable flow... In addition, since stressed volume is present during ongoing circulation, so must be its related pressure...*". In no way do these intuitive notions address the question of conservation of energy. What emptying mechanics? Does not each vascular compartment have its own "stressed" volume in relation to its local distending pressure, dependent on flow?

