Letters To The Editor: I have argued (1–3) against interpreting Guyton’s findings as revealing that steady-state venous return (Q) is driven through the resistance of the venous system, Rvs, by the difference between mean systemic pressure (Pms) and right atrial pressure (Pra). Despite previous opposition to this argument (4), Magder et al. (5) in their model analysis show Q driven not by (Pms − Pra) but equal to (Pvs − Pra)/Rvs. Both Pvs (the pressure in the compliant compartment representing the venous system) and Pra are variables that settle in the steady state at levels dependent on various specified parameters1 and specified values for blood volume and the pump properties defined in Eq. A11–A15. Nonetheless, in their discussion and in their appendix C, the authors still speak of steady-state flow driven by elastic recoil.

In the sentences just prior to their Eq. 1 and in their Eq. 3, Magder et al. (5) focus on a time constant, tau, and monoequational emptying of elastic structures. Indeed, their mathematical model is built with differential equations and is thereby capable of describing dynamic situations, during which compartment volumes change, for which time constants are useful parameters. But what they call “venous drainage in the steady state” is not about transient volume changes—in the steady state, the various compartment volumes remain fixed and therefore neither release nor take up elastic energy2. Steady-state flow through the system, i.e., Q, is the same through all the resistive elements. Steady-state pressure gradients, such as (Pvs − Pra), have settled to the level equal to the product of Q and the associated resistance.

True, what determines Q has to do with how fixed total blood volume distributes between the various compliant compartments during the approach to the steady state. The eventual equilibrium is the outcome of the interaction between the properties of the cardiopulmonary and systemic vascular subsections of the system model shown in their Fig. 1. However, the (Pvs − Pra) level shown in Eq. 1 of Magder et al. (5) has nothing to do with ongoing emptying of compliant compartments. None of the energy dissipated in forcing Q through Rvs comes from the elastic recoil of venous compartments. It comes from the steady-state rate of energy output from the cardiac pump in accordance with their Eqs. A11–A15 for ventricular pressures.

In the discussion associated with their Eqs. 1 and 2, Magder et al. slip into the notion that that Q is caused by the (Pvs − Pra) gradient. Complicating their Eq. 1 by dividing top and bottom by Cvs (i.e., venous system compliance) to arrive at Eq. 3 and introduce a time constant parameter only obscures the issue.

The Q = (Pms − Pra)/Rvs formulation criticized in Refs. 1–3 is a simplification of an expression obtained through algebraic manipulation of steady-state relationships. Magder et al. (5) use a similar development to arrive at their Eq. C4 and proceed to improve on it, arriving at their Eq. C10. It describes an obligatory steady-state inverse relationship between the two variables, Q and Pra; all the other terms, specifically including mean systemic filling pressure (MSFP; a proxy for total volume and compliance), are constants. In words, in their model of the peripheral vasculature, under the constraint that total volume remains fixed, Pra must fall with increase in Q. This is the counterpoise against the tendency in the cardiac pump for Q to increase with increase in Pra.

Further algebraic manipulation brings the development of Magder et al. (5) to Eq. C14 and to the accompanying statement that it “...indicates that cardiac output is determined by...” followed by a list of the parameters of the peripheral vasculature included in Eq. C14. But the equation does not show what determines Q. What it shows is the obligatory Pra for a given steady-state Q (and vice versa). What determined the equilibrium levels of both Pra and Q predicted by their model was not just the parameters of the systemic vasculature but also the pump properties coded in their equations for ventricular pressures.

Although Magder et al. (5) include me among those who argue that the drainage characteristics of the veins are not “a significant factor in the regulation of cardiac output,” I certainly do not dispute the importance of resistive and elastic properties of the venous vasculature in the complex interplay between pump and vasculature that determines steady-state cardiac output. Clearly the volume and pressure profiles throughout the system must settle at levels consistent with the properties of the pulmonary and peripheral vasculatures and of the cardiac pumps. What I object to is perpetuating the notion that elastic recoil in the peripheral vasculature generates steady-state venous return.

REFERENCES