This view of Pra as the determinant of Fv in proportion to its decrement relative to Pms, which I will call the \((\text{Pms} - \text{Pra})/\text{Rven}\) concept, is an interpretation of findings of Guyton et al., presented in venous return curves like that in Fig. 2 (many publications by Guyton and his coworkers address our subject; for background citations, see Ref. 1). My argument is that the interpretation is wrong.

To begin with, the balloon model has a glaring defect. It would not generate the steady flow associated with any level of Pra below Pms in the venous return curve because outflow would remove volume from the elastic compartment. Inside pressure would fall along with volume according to the compliance of the compartment. Outflow rate would decline accordingly as the elastic energy stored in the compartment walls was expended. To keep Pms constant would require a pump, but then the drive for Fv comes from the pump, not stored elastic energy manifested as Pms.

Stored elastic energy was not what propelled the flow recorded for venous return curves like that in Fig. 2. Flow came from a pump whose output, recorded as Fv, passed into the aorta of the peripheral vasculature under study. The only way to change Fv was by manually resetting the pump rate or by throttling the pump by imposing a resistance in the connection to its inflow port.

Return flow was intercepted at the right atrium (where Pra was recorded) and fed through a Starling resistor to the input end of the pump. The Starling resistor functioned as a variable resistance that throttled the pump, thus changing pressures and volumes throughout the vasculature until Pra settled at the value consistent with the height of the hydrostatic column between the level of the resistor and the level of the right atrium. The beauty of this closed-loop design was that they could keep the volume contained within the vasculature constant while recording a range of steady-state levels of Fv and corresponding Pra by adjusting the height of the resistor (see, for example, Ref. 4).

So, Fv was certainly not the outflow of an elastic compartment shrinking in volume, it was recorded when flows, pressures, and segment volumes throughout the vasculature were steady.

Also, in no way was venous return recorded as distinct from the rate at which flow entered the aorta. In the investigators’ view, cardiac output would be the flow seen by an observer in the aorta looking upstream. Venous return would be what the observer would see if he turned around and looked downstream, the same flow, but in the opposite sense.

Nor was Fv set by adjusting Pra. It is not generally recognized that the Starling resistor circuit was the control element in a closed feedback loop and that its variable resistance, not Pra back pressure, caused Fv changes. What Guyton et al. varied as an independent variable was resistor height, not Pra.

Writers have stressed that one cannot say Pra or Fv is the independent variable in the intact cardiovascular system (e.g., Ref. 6). The same is true of the Starling resistor + peripheral vasculature + pump system. But, when we open loops, we can identify independent and dependent variables unequivocally. Remove the Starling resistor, find some other way of keeping total circulating volume constant, and you can independently set Fv at various levels in an isolated peripheral vasculature and observe what happens to Pra [as various workers have done, e.g., Levy (5)]. Obviously, you cannot do the opposite.

Fig. 2. Mean circulatory (Pms) and right atrial (Pra) pressures as the pressure gradient driving venous return. Implicit in typical discussions of the influence of Pra on venous return is a balloon-like model like the \(\text{Pms}\) shown with inside pressure at Pms; a resistive outflow path representing venous resistance, Rven; and pressure at the outflow end at Pra. Outflow from the balloon would obey the relationship \((\text{Pms} - \text{Pra})/\text{Rven}\). Provided that \(\text{Pms}\) were held constant, this expression would also describe the sloped portion of the graph of the relationship between steady-state flow and Pra, an idealized “venous return curve.”

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COUNTERPOINT: THE CLASSICAL GUYTON VIEW THAT MEAN SYSTEMIC PRESSURE, RIGHT ATRIAL PRESSURE, AND VENOUS RESISTANCE GOVERN VENOUS RETURN IS NOT CORRECT

How mean circulatory pressure (Pms) and right atrial pressure (Pra) influence venous return (Fv) in relation to resistance of the venous system (Rven) is commonly discussed in terms that imply the balloon-like physical model illustrated in Fig. 2. The model supports characterization of Pra as a “back pressure” and assertions such as pointing out that elevating Pra to equal Pms would stop venous return (6).

Fv is the independent variable in the Fv:Pra relationship in the isolated vasculature. Without a pump, you can set Pra wherever you want but you will get no steady-state flow.

My dispute is with the \((\text{Pms} - \text{Pra})/\text{Rven}\) concept, not the significance of the experimental results. Knowing how Pra changes in relation to steady-state flow passing through the vasculature as an open loop subsystem of the cardiovascular system with Fv as the independent variable enabled an important advance. Guyton put this new information together with cardiac output curves [properties of the open loop cardiopulmonary subsystem, with Pra as input and flow, Fco, as output, (2)]. By doing this graphically, he could discuss steady-state equilibrium points for the closed-loop system in terms of changes in either subsystem, such as the overall elevation of a venous return curve with increased system volume.

In this technique, both open-loop relationships are plotted on one graph. Guyton chose to put flow on the \(y\)-axis and pressure on the \(x\)-axis. That meant that the peripheral vasculature dependence of Pra on Fv ended up plotted as in Fig. 2, i.e., with the independent variable on the \(y\)-axis. Unfortunately, the apparent proportionality between \((\text{Pms} - \text{Pra})\) and Fv plus the mistaken idea that Pra was actually the independent variable launched the \((\text{Pms} - \text{Pra})/\text{Rven}\) concept.\(^1\)

Perhaps two other considerations contributed to the persistence of the concept. 1) \((\text{Pms} - \text{Pra})/\text{Rven}\) appeals to those with a Poiseuillean view who look for a pressure gradient as the cause of flow through a vascular segment and overlook the fact that pressure gradients and flow in the vasculature develop hand in hand as a consequence of pumping. 2) The elastic compartment in the physical model in Fig. 2 has an intuitive appeal because of the importance of stored elastic energy in driving venous return as understood in the following sense. The appropriate reason for a separate term for “venous return” as distinct from “cardiac output” is that the rates at which blood is pumped into the aorta and at which flow returns to the right atrium can differ temporarily. These transient discrepancies involve transfers of elastic energy and changes of vascular volumes beyond the predictive capability of a pumpless one-chamber model.

Why then does Pra fall below Pms in proportional relation to flow? Not because Pms is a fixed pressure head at the upstream end of a fixed venous resistance, but because progressively greater flow creates a progressively steeper pressure profile around the peripheral vasculature. With no flow, pressure in all segments of the vasculature is Pms. Forcing flow through the vasculature elevates arterial pressures above Pms. Total blood volume is fixed, so the volume that expands arterial segments is displaced from venous segments where pressures therefore fall below Pms. It is this progressive reallocation of total volume among the elastic segments of the vasculature that results in decline in Pra proportional to flow.

So what does drive venous return? In the isolated peripheral vasculature setting of venous return curves, it is set by a pump. In the closed-loop cardiovascular system, it equilibrates with cardiac output at a level set by variables such as total system volume, contractility, and elastic state of the vasculature that we could discuss with the aid of cardiac output and venous return curves. In stresses that disturb cardiovascular equilibrium, it changes dynamically as volumes redistribute among the organ vasculatures, conduit vessels, and heart. Neither steady-state nor dynamic venous return is properly described as driven by Pms in proportion to the back pressure from Pra.

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REBUTTAL FROM DR. MAGDER

So close yet so far apart! Dr. Brengelmann finds a “glaring” defect in the balloon model of the circulation and presumably my bathtub analogy (Brengelmann 2006). He argues that the elastic-recoil pressure in the balloon is rapidly dissipated and to “keep Pms constant would require a pump, but then the drive for Fv comes from the pump.” I agree with the first part and argued that the heart provides a “restorative” force. However the heart does not “drive” venous return just as the tap filling a tub does not “drive” emptying of the tub. His argument misses Guyton’s key point that the “working” cardiac output is determined by interaction of pump \(f\)unction (not cardiac output) and return \(f\)unction (3) and thus the pump is an integral part of Guyton’s analysis. He also fails to deal with the flow that occurs without a pump, even if only transiently and that maximum flow is defined by the ratio of stressed volume (\(\nu\)) to the time constant of its drainage, which is determined by the product of venous compliance (\(C_v\)) and resistance (\(R_v\); Ref. 8).

In the physiological range, \(C_v\) is essentially constant so that four variables define the system: flow (\(Q\)), \(\nu\), \(R_v\) [includes the distribution of \(Q(1)\)], and right atrial pressure (Pra). A change in one requires a change in at least one of the others (1, 8). The heart only controls \(Q\) and Pra. In most of Guyton’s experiments, \(\nu\) and \(R_v\) were constant and changes in Pra were related to changes in \(Q\) by a changes in cardiac function (or pump in the experiments). In other studies (2, 5), a pump held cardiac output constant, and changes in Pra equivalent required changes in \(\nu\) or \(R_v\). A physiological example occurs with the rise in Pra and fall in \(Q\) with an increase in pleural pressure.