Letter to the editor: “A return to the venous return controversy: a visual aid for combatants”

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TO THE EDITOR: I have followed with interest the controversy surrounding the determinants of venous return, specifically the latest contribution by Beard and Feigl (1) published in the American Journal of Physiology-Heart and Circulatory Physiology (1–5). Since this debate has continued for several years without apparent resolution, I would like to offer the following suggestions for a common ground visual aid that may be of use in clarifying some of the points of difference. As a starting point, since the circulation is a complete closed loop, it would be helpful for any physical or conceptual model of the systemic circulation to reflect this anatomic reality. Such a visual aid and conceptual model might help to avoid errors in interpretation that can arise from analysis of only one segment of the circulation in isolation from the dynamics of a closed loop.

In addition, it would be helpful to remember how mean systemic pressure (PMS), the supposed upstream pressure driving venous return in the classic Guyton view, was defined in the first place. In 1897, Ernest Starling pointed out that, “Somewhere in the circulation there must be a point where the pressure is neither raised nor lowered and where, therefore, the pressure is independent of cardiac activity” (7). By definition, neither the pressure nor, therefore, the vascular wall tension changes as a function of cardiac activity at that point. Consequently, in the simple model depicted below (Fig. 1), neither volume nor pressure change at the point of PMS, which occurs in the peripheral venous compartment.

It may be helpful in establishing new parameters for this debate if those who interpret right atrial pressure (PRA) as a “back pressure” impeding venous return and who posit the gradient between PMS and PRA as a driving force that determines venous return would comment on the following two observations:

First, by definition, only one of the two boundaries (PRA) of that pressure gradient changes with cardiac activity and vascular flow. PMS does not change in an isovolemic situation. PRA cannot decrease until the heart moves blood from the central venous compartment (CVC) to the arterial compartment (AC). This is consistent with Levy’s observation in a right heart bypass model that central venous pressure falls as a result of an increase in the right heart pump flow setting and is not the cause of an increased flow rate (4). Venous return is only enabled by the action of the heart, not by the static elastic properties of the venous compartment. The former correlates with flow; the latter does not, since it remains unchanged with or without flow. To use an even more visual analogy, if it were possible to facilitate steady-state flow with an isovolemic and isometric elastic element, then oil pipelines could achieve increased flow rates by the simple inclusion of a static elastic element under stretch somewhere upstream in the pipeline. A

Fig. 1. A simplified 3 compartment model of the systemic circulation. AC, arterial compartment with low capacitance or compliance (CA); P A, arterial pressure; PVC, peripheral venous compartment with a greater capacitance or compliance (CV); CVC, central venous compartment with a compliance that can accommodate changing central blood volumes. The AC and PVC could just as well be drawn as closed, elastic elements with differential compliances. PMS, mean systemic (circulatory arrest) pressure; PRA, right atrial pressure; VR, venous return; RV, resistance to venous return (a more useful mathematical than anatomical concept, not depicted in the figure but included in the standard equation for venous return, Eq. 2); PATM, atmospheric pressure; V, blood volume; Vo, unstressed blood volume; SVR, systemic vascular resistance; CO, cardiac output; Ra, right atrium.

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“restorative” function of the heart or another pump operating on the upstream volume source (to use Magder’s terminology, Ref. 5) does not explain away that analogy. An isometric elastic element involves no energy exchange and can move nothing while remaining isometric. Similarly, the locus of PMS in the veins remains isometric during steady-state flow and is constrained from moving blood.

Second, since our observational study in 2001 (6), there have been 25 other studies confirming that the gradient between peripheral venous pressure (close to, if not identical with, PMS) and PRA are essentially unchanged within the same patient under widely varying clinical conditions and flow states. The model depicted here accommodates those observations, but a model where the PMS-PRA gradient changes and determines venous return does not. The model below predicts, in an isovolemic state, a fall in PRA with an increase in cardiac activity but an essentially unchanged peripheral venous compartment volume and peripheral venous pressure, corresponding to an unchanged PMS. During the transition between circulatory arrest and circulatory function, the heart moves blood from the CVC to the AC. By definition, at circulatory arrest, PMS = PRA. As blood is evacuated from the CVC by the heart, PRA falls below PMS and a gradient develops. In fact, PRA is reasonably considered as a local distortion of PMS, caused by the activity of the heart. According to the large body of clinical data available, where PRA (equivalent to central venous pressure, or CVP) and PMS (peripheral venous pressure) have been simultaneously measured, there appears to be a threshold effect where increasing cardiac activity is coupled to an increase in cardiac output and venous return but where neither PMS nor PRA changes unless blood volume changes. In accordance with clinical observations, the visual aid presented here predicts that both PMS and PRA will respond to volume changes while also accommodating such a threshold effect in the PMS-PRA difference.

DISCLOSURES
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REFERENCES