Beware of the venous return in cardiovascular control

Soren Sondergaard and Per Werner Moller

Department of Anaesthesia and Intensive Care, Sahlgrenska University Hospital, Gothenburg, Sweden

Submitted 5 July 2012; accepted in final form 23 July 2012

TO THE EDITOR: Cannesson et al. (1) hypothesize that the effect of phenylephrine (PE) on cardiac output (CO) is dependent on the volume state of the cardiovascular system. This system may be dependent or independent of volume assessed from increase in CO by fluid bolus. In the independent state, PE decreases CO explained by increased afterload. In the dependent state, PE increases CO explained by mobilization of splanchnic capacitance, increasing right atrial pressure (RAP) and CO. Authors refer to Magder (6) in support of this duality. However, afterload of this magnitude hardly opposes CO [see Herndon and Sagawa (4)]. We suggest that COFP decreases 14 s prior to minimum IVCfp in the independent state depending on preload from pulmonary circulation. PE is a vasoconstrictor in pulmonary circulation, which, according to Thiele et al. (8), is rich in alpha(1)-receptors. The reduction in COFP coincident with increase in MAP may be due to increase in pulmonary vascular resistance (PVR). Where is flow, measured by IVCfp, deposited in the independent state during this period? What are the changes in pulmonary artery pressures? Magder’s editorial based the duality on the balance between splanchnic mobilization (preponderant in dependent state) and increase in resistance to venous return (RVR) (preponderant in independent state). Magder’s suggestion is supported by the experimental data of Cannesson et al. (1) and the concept of the venous return function (VR), detailed by Guyton (3) and further developed by Parkin and Leaning (7). Central to VR is the mean systemic filling pressure (Pms), estimated by the Parkin algorithm (7) or the intervention applied by Maas et al. (5). Calculation of RVR is approximated by (Pms - RAP)/IVCfp. In the independent state, RVR increases 5, 11.5, 21, and 32% for each step increase in PE bolus. In the dependent state, RVR increases 2, 5.5, 9, and 19%. Why, in the independent, hypervolemic state, does flow in IVC decrease and RVR increase, while the opposite is observed in the dependent, hypovolemic state?

Gelman and Mushlin (2) reviewed volume recruitment in splanchnic circulation by alpha(1)-agonism. This is effective up to a point where further alpha(1)-activity increases RVR more than it mobilizes volume. This resistance is anatomically located to splanchnic, portal, hepatic, and sinusoidal veins. These vessels, however, dilate in response to beta(2)-activity and decrease RVR. Explanation to the data of Cannesson et al. may be found in the balance between alpha(1) and beta(2)-activities. The independent, hypervolemic state, presumably, has a low endogenous sympathetic activity. The animals respond with vasoconstriction, increased Pms, and RVR when subjected to exogenous pure alpha(1)-activity. In the dependent hypovolemic state, animals with intact reflexes respond to exsanguination with higher endogenous sympathetic activity and recruit volume from splanchnic capacitance via alpha(1)-activation, facilitated by decreased downstream RVR via beta(2)-activity.

It seems plausible that the vasoconstrictive effect of exogenous pure alpha(1)-agonism is partly counteracted by endogenous beta(2)-activity.

In summary, the cause of the differential effects of PE on CO in hypo- vs. hypervolemic states is not to be sought in the effect on cardiac function, but in the action of PE on the venous return function and the interplay between the numerous factors governing splanchnic circulation (2).

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: S.S. and P.W.M. conception and design of research; S.S. and P.W.M. analyzed data; S.S. and P.W.M. interpreted results of experiments; S.S. and P.W.M. drafted manuscript; S.S. and P.W.M. edited and revised manuscript; S.S. and P.W.M. approved final version of manuscript.

REFERENCES