The following letter is in response to the Point:Counterpoint “Active venoconstriction is/is not important in maintaining or raising end-diastolic volume and stroke volume during exercise and orthostasis” that appeared in the October issue.

To the Editor: Hainsworth and Drinkhill (4) argue against the importance of active venoconstriction in raising right atrial pressure (RAP) during exercise based in part on a low reserve of blood volume that can be mobilized by baroreflex-induced increases in sympathetic activity. Because the arterial baroreflex is largely a resistance-raising reflex and not a flow-raising reflex (1, 2), the low reserve is perhaps not surprising. In sharp contrast, the pressor response to muscle ischemia (termed the muscle chemoreflex or muscle metaboreflex) during dynamic exercise is produced almost entirely by an increase in cardiac output, the maintenance of which likely requires substantial venoconstriction (5, 6). Accordingly, we have demonstrated that the muscle chemoreflex is threefold more potent in raising right atrial pressure (5) than are the arterial baroreflexes (1). Moreover, when arterial pressure is maintained constant by ventricular pacing, strong activation of the muscle chemoreflex can reflexly raise RAP by 8 mmHg (5), a substantial fraction of which is likely due to extrasplenic venoconstriction (3). Therefore, it appears that the reserve capacity for central blood volume mobilization in conscious dogs during exercise is much greater than suggested by Hainsworth and Drinkhill (4). However, the extent to which the muscle chemoreflex is active during normal exercise remains a pressing issue.

REFERENCES


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