Neurologically generated electric currents of myocytes are the most rapid stimulus for the capillary pumps, even preceding muscle contraction

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TO THE EDITOR: We appreciate Drs. Villar and Hughson’s (5) excellent experiments and their scientific approach and diligence in trying to explain exercise phenomena in the cardiovascular system with the two widely accepted theories: for the local blood flow regulation and for the venous return. We find their results exceptionally important and regret that the strictly followed tradition has not allowed for the more rational use of their results. In the cited Ref. 32, recognizing that exercise hyperemia mechanism remains unresolved, it is concluded that both functional venous muscle pump and 2-s rapid vasodilatation contribute to the early exercise hyperemia. Recently (see Ref. 5 in Ref. 2 listed below) we “called” a little bird to testify that it needs 160 times more rapid vasodilation to support its 80/s wing beats. Experimenting on ourselves we proved that trained subjects can easily make, without load, 5/s rotations of their forearm. On the other hand, Villar and Hughson (5, Table 1) report ~20-fold baseline reactive hyperemia, which (if all other resistances in the conduit are zero) would require ~4.5-fold increase of the diameter of resistance arterioles.

Involving the cell thermodynamic homeostasis (a term that we first used), the picture with the arteriolar regulation “in bulk” (2) looks much more “apocalyptic.” Here we would like to draw attention to the fact that our capillary pumps (CPs) theory is based on the metabolic heat creating thermal gradient toward the capillary blood and the newly proposed CO2 pressure elevation in the capillaries, which have nothing in common with the published experiments with elevated ambient temperature and the supposed (but not confirmed) CO2 arteriolar dilation, respectively, as our letter was incorrectly interpreted (1). Is it not paradoxical that in most studies on exercise hyperemia the terms capillaries, heat, temperature, and CO2 cannot be found? One hundred thirty-two years ago it was observed that capillaries belonging to the same arteriole simultaneously behave oppositely and that capillary pressure gradient had “barely appreciable effect on capillary flow” (4). The latter is increased by electrical stimulation (even without muscle contraction), by gentle massage, or by strong light (see Ref. 3 in Ref. 2 listed below). That hints that capillary pumps are stimulated, except by the heat (produced by massage), by light (which was one of our experimental designs, when we discovered the anomaly of CO2 solubility in saline and also in vivo on ourselves) or by electrolysis, which also produces CO2 nanobubbling. Thus we extend the function of our capillary pumps with the direct action in the capillary blood of the myocyte-generated electric currents by their neurological stimulation, which must be the most rapid impact, even preceding muscle contraction.

Thus Villar and Hughson’s reactive hyperemia (5, Table 1) is caused by the heat- and CO2 metabolite-driven CPs supplying the arteriovenous pumps (AVPs) (3). The latter work is strongest in head-up tilt (strongest vein tone) (see Ref. 2 in Ref. 3 listed below), feeble in horizontal (feeble vein tone), and most feeble in head-down tilt (without vein tone). The same reasoning for the AVPs’ action should be applied for the exercise protocols. The mentioned perfusion pressures cannot be correct because they do not account for the venous pressures, which are AVP dependent and are not known. They are not needed for the explanation.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

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