“Functional sympatholysis” in the present concept does not exist:
arteriovenous pumping, supplied by capillary pumps, explains immediate exercise hyperemia

Adelina V. Pancheva, Vladimir S. Panchev, and Marieta V. Pancheva
Sofia, Bulgaria

TO THE EDITOR: We read with great interest Dr. Casey and colleagues’ (1) investigations aimed at discovering the causes of substantial reduction in forearm blood flow under hyperoxia. In our opinion, they concluded quite rightly that “functional sympatholysis” (one of the most cited, yet half a century old notion) is far from explaining the observed reduction and that it does not have a vasoconstrictive origin. In 1962, in the Handbook of Physiology, Wiggers (5) warned that venous return-mechanism is open. We were driven to write this letter with the hope that these results will motivate the authors to join us in demonstrating that the half-century failure to find what lies at the root of this vague term is good evidence that its introduction and use masks the failure to adequately explain immediate exercise hyperemia with the theory of arteriolar regulation. As we already noted (3), this theory is one of the greatest fallacies in animal physiology, and its continuing dominance and teaching at school, together with the fallacy that venous muscle pump is the principal driver of venous return in orthostasis (2), produce new generations for whom these two misconceptions are dogmas.

In our opinion, Dr. Casey et al.’s very useful results are easily explainable with the Haldane effect of oxygen displacing CO2 from hemoglobin. Thus CO2 reduction weakens the action of capillary pumps (CPs) (3). Supportive of the authors’ findings for a nonsympathetic origin of hyperoxia-induced blood flow reduction are reports showing the same effect in organs not subjected to sympathetic vasoconstriction, such as the brain, retina, and heart. Supportive of our explanation is that adding CO2 to the breathed gas diminishes the blood flow reduction caused by hyperoxia in the brain and retina. This was not explored by the authors.

The introduction of “functional sympatholysis” deepened the fallacy of arteriolar regulation by introducing a new fallacy that sympathetic action is counteracted. Evolution has not created actions to “lyse” them concomitantly, which is extravagant. In the resting muscle, blood flow is governed by the arteriovenous shunts, which are under sympathetic control. In the exercising one it is governed by CPs, mechanically driven directly by the muscle contractions (muscle CPs) and thermodynamically by CPs (3). Arteriolar regulation has secondary, blood flow-related NO dilatory action, which could be named “sympatholysis.” The mass sympathetic outflow at exercise onset has the following positive effects, neither of which is “lysed” initially in the contracting muscle.

1) Supporting arterial pressure during the instant action of CPs until the blood supplied by them is returned to the heart by the AVPs (2);
2) “Injecting” blood in the capillary pumps at exercise onset (as the accelerating pump injects fuel in the gasoline engine by pressing the accelerator pedal) causing the peak in the resting area (Figs. 2 and 4 of Ref. 4);
3) Increasing arterial pulsations driving AVPs;
4) Increasing venous ton, supporting the action from point 3; and
5) Redirecting blood to the active organs.
The small increase of O2 uptake during the initial strong blood flow increase, shown by Remensnyder et al. (Fig. 2 and pp. 376–377 of Ref. 4), we explain with the mechanical and anaerobic action of CPs. This reveals convincingly the fallacy of the O2 demand regulation.

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

AUTHOR CONTRIBUTIONS
Author contributions: A.V.P., V.S.P., and M.V.P. conception and design of experiments; A.V.P., V.S.P., and M.V.P. prepared figures; A.V.P., V.S.P., and M.V.P. analyzed data; A.V.P., V.S.P., and M.V.P. interpreted results of experiments; A.V.P., V.S.P., and M.V.P. prepared figures; A.V.P., V.S.P., and M.V.P. drafted manuscript; A.V.P., V.S.P., and M.V.P. edited and revised manuscript; A.V.P., V.S.P., and M.V.P. approved final version of manuscript.

REFERENCES