Cerebral call for justice: My cognitive aging problems are not merely the passive result of arteriole dilation; raised RBC rigidity plays the major role

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

Sofia, Bulgaria

TO THE EDITOR: The excellent study of Dr. Barnes and coworkers (1) confirmed that aerobic exercise improves cognitive function in older adults, ameliorating cerebral blood flow. We appreciate the importance given to the middle cerebral artery blood velocity (MCAv) reactivity and agree that this may encourage adequate behavioral interventions. At the same time, we categorically cannot accept that the authors stick to one of the most destructive physiological dogmas (the arteriolar regulation), assuming that CO₂ and prostaglandins work by dilating arterioles. In total discrepancy with preserved physical cognition, this deleterious dogma imposes that arterioles are “taps” “irrigating” the highest biological organization (human brain), whereas MCAv is instantly governed by the mentally generated electric currents, exciting the capillary pumps (CPs) (3). For science, this dogma is the guillotine that in 1794 ended the life of Antoine-Laurent Lavoisier (mistakenly convicted), shortly after discovering the principle of the production of CO₂, finding the likeness of life to fire. It is the same “guillotine” that forced the brilliant proponent of this gas, Y. Henderson, to change his mind, writing in 1940-Cyclopedia of Medicine: “differently from fire, the oxygen consumption in the body is regulated by independent from CO₂ factors” (2). CO₂ is not proven as a vasodilator. Prostaglandins are inflammatory agents released in the microvasculature, and our concept is that they enhance the work of the CPs by displacing CO₂ [2 (Ref. 4 therein)]. Their vasodilatory action measured in vitro is quantitatively inversely dependent, turning into contraction in higher concentrations. Is it not absurd that such dependence is taken seriously as a regulator of MCAv? Adding to this, prostaglandins’ hypothetical extravasation should occur from the postcapillary venules [2 (Ref. 4 therein)]; should they travel upstream to the supplying arteriole, endangering the vulnerable brain cell thermodynamic homeostasis, the picture becomes even more “apocalyptic” than in the muscle [3 (Ref. 4 therein)]. Exercising subjects regulate instantly their MCAv by task-produced electric currents (3), which depend on the applied efforts. When writing emotionally on this dogma, we produce the strongest proof of the electrical regulation of the CPs by simply sensing our MCAv. It is a similar sensation when applying strenuous physical effort. In our view, it is a minus that the unknown mental conditions were not incorporated as a limitation.

With the above in mind, we offer the following: first, the CPs (2, 3), which work depends on both CO₂ and prostaglandins; second, the increased RBC rigidity with age and their decrease in athletes, as a result of their slower and faster turnover, respectively. Increased RBC rigidity directs more blood to the arteriovenous shunts, where the flow is not CO₂ dependent, from which comes the correlation of MCAv reactivity with the maximal aerobic fitness in older adults. Exercising 12 wk [1 (Ref. 17 therein)], which approximates the RBC life span, seems enough to manifest improvement in their average deformability by diminishing blood diversion to the shunts, thus increasing MCAv resting reactivity in young and aged, whereas MCAv was not changed, because resting CP work feebly and the blood diverted from them is entirely undertaken by the shunts and conversely.

DISCLOSURES

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Address for reprint requests and other correspondence: A. V. Pancheva, Bulgaria, 1324 Sofia, j. k. Ljulin 10, bl. 134, ap. 27 (e-mail: adelina @vam-panchev.com).