The optimal hematocrit increases during exercise

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TO THE EDITOR: We appreciate the approach of Drs. Stark and Schuster (13) to elaborate a systematic foundation of the optimal hematocrit concept on physics. However, some corrections and additional information are necessary.

It is important to note that the empirical description of Pries et al. (7)—in contrast to the theoretical approaches for bulk viscosity—is based on experiments for tube flow of blood and thus does include the radial red cell concentration profile for a given combination of tube diameter and hematocrit. For this situation, a complete theoretical treatment is still missing, but some approaches have been developed [reviewed in (11)]. Optimal hematocrit values for bulk viscosity approaches (experimental or theoretical) will only apply to vessels with diameters above 300 μm. However, most of the flow resistance in the circulation is located in microvessels below that diameter range. In the critical microvascular range, additional effects may be elicited by the blood/endothelium interface, which is covered by the endothelial surface layer (8). Recently (9) it was shown that this layer is influenced by high intravascular hematocrit levels, further complicating the situation in vivo.

The concept that an increased hematocrit value at altitude is an advantage has been questioned. Humans genetically well adapted to altitude since at least 20,000 years such as part of the Ethiopians and Tibetans do not possess high hemoglobin concentrations (2).

Astonishingly both Stark and Schuster as well as Pasipoularides (6) in his editorial do not really treat the influence of physical exercise. We have reviewed these aspects recently (3). The optimal hematocrit increases in perfused dog muscles (4) from 33% at rest to 50–60% during exercise. The latter fits to the value of 60% for capillary size vessels obtained with Pries et al.’s (7) formula, because the number of perfused small vessels increases in the working muscle. Animals such as dogs and horses inject erythrocytes stored in their spleen into the active circulation during running, thus bringing the hematocrit in line with the optimal value.

In humans, who do not possess a storing spleen, the hematocrit also rises during exercise [to ~50%, e.g., (5)]. The cause is a water shift from plasma to tissues because of increased arterial blood pressure and increased osmolality in the working muscle. Because the selection drive for survival (danger of thrombosis and hypertension) probably has favored a low hematocrit, the human value is not optimal for exercise. The same mechanism is presumably the cause for the low hematocrit in the best adapted highlanders. Turning this argument, doping with blood is effective but dangerous.

Additional important factors might be the hematocrit-influenced transport of NO from erythrocytes to the arterial wall (12) and the variation in red cell size between 35 and 100 fluid liters (1). Finally, if animals are excited during blood sampling, catecholamine secretion causes spleen contraction and the resting hematocrit increases in various species (e.g., dogs, horses, camelids); also body position (10) exerts an influence. This might explain part of the variability in Table 1.

REFERENCES