The following is the abstract of the article discussed in the subsequent letter:

P. A. Wilkins, R. D. Gleed, N. M. Krivitski, and A. Dobson. Extravascular lung water in the exercising horse. J Appl Physiol 91: 2442–2450, 2001.—Seven standardbred horses were exercised on a treadmill at speeds (~12 m/s) producing maximal heart rate, hypoxemia, and a mean pulmonary arterial pressure of ~75 mmHg. Extravascular lung water was measured by using transients in temperature and electrical impedance of the blood caused by a bolus injection of cold saline solution. Lung water was ~3 ml/kg body wt when standing but did not increase significantly with exertion. We conclude that any increase in fluid extravasation from the pulmonary hypertension accumulates in the lung at a level that is less than that detectable by this method. At maximal exertion, the volume of blood measured between the jugular vein and the carotid artery increased by ~8 ml/kg, and the actively circulating component of the systemic blood volume increased by ~17 ml/kg with respect to corresponding values obtained when walking before exertion. These volume increases, reflecting recruitment and dilatation of capillaries, increase the area for respiratory gas exchange and offset the reduced transit times that would otherwise be imposed by the approximately eightfold increase in cardiac output at maximal exertion.

Exercise, extravascular lung water, and gas exchange

To the Editor: The article by Wilkins et al. (3) contains much data of interest, prompting two comments.

First, the lack of increase in detectable lung water is entirely compatible with the essential preservation of ventilation-perfusion (V\textsubscript{A}/Q\textsubscript{A}) relationships with exercise reported in the horse (1, 2). It would be of interest to measure lung water during exercise with reliable methods in species known to worsen V\textsubscript{A}/Q\textsubscript{A} relationships on exercise (pigs, humans), because the hypothesis that increased lung water during exercise causes more V\textsubscript{A}/Q\textsubscript{A} mismatch has not yet been excluded. The current equine data should not be argued as evidence against exercise causing interstitial edema in other species in which the gas-exchange response differs. Parenthetically, that a species developing very high pulmonary vascular pressures during exercise does not develop measurable interstitial edema or V\textsubscript{A}/Q\textsubscript{A} mismatch, whereas other species show worsening V\textsubscript{A}/Q\textsubscript{A} relationships during exercise despite lower vascular pressures, is of great interest.

Second, the authors must be taken to task for overinterpreting their own data. Both the abstract and text (p. 2448) state that the 56% increase in central blood volume on exercise offsets the eightfold increase in cardiac output in regard to transit times. The clear implication is that diffusion limitation should not occur. However, Table 1 shows that transit time falls fully fivefold from 13.5 to 2.6 s between rest and maximal effort. The offset is clearly only partial. Moreover, the transit time in question is between jugular vein and carotid artery (p. 2448). Only a fraction of this represents pulmonary microvascular gas-exchange time. The authors also claim a plateau in transit time between 75% and maximal effort (p. 2449), but in fact, from Table 1, there is a 0.4-s further reduction. If this occurred mostly in the exchange vessels, it could have a large negative effect on diffusion equilibration. Similarly, whether the increase in central blood volume was in exchange or conduit vessels is unknown, further complicating the argument. Because it is therefore difficult, if not impossible, to know how actual gas-exchange contact time changed from rest to exercise, it is suggested that the conclusions about mechanisms of hypoxemia (abstract and p. 2449) need to be scaled back commensurate with what the data actually show.

REFERENCES


Peter D. Wagner

Divisions of Physiology and Pulmonary and Critical Care Medicine

University of California, San Diego
La Jolla, California 92093
E-mail: pdwagner@ucsd.edu

REPLY

To the Editor: We agree with Professor Wagner that the failure to detect a substantial increase in lung water during exercise should be examined carefully in other species. However, it is conceivable that ventilation-perfusion ratios are independent of changes in lung water.

In interpreting our use of the term “offset” as “completely offset,” Professor Wagner may be committing a solecism similar to that of which he accuses us. We thought it obvious from the numbers given that the offset is partial but agree that we would have been better to include this modifier. We do not wish to imply that diffusion limitation cannot occur.
We too would like to think the transit time at 100% effort is less than at 75%; however, the data over this range appear asymptotic (Fig. 4), and the mean fall (0.35 s) is well within the scatter of the measurement (see Table 2, not Table 1). Hence, we cannot use these data to infer that the significant decrement in arterial PO\textsubscript{2} (PaO\textsubscript{2}) is due to decreased transit time. We did, however, observe a significant, contemporaneous, 17% increase in the blood volume between the pulmonary and carotid arteries. If some of this increase occurred in the pulmonary capillary bed, then increased diffusion distance could play a role in the etiology of the decrease in PaO\textsubscript{2}. Of course we cannot exclude other mechanisms for the decrease in PaO\textsubscript{2}.

Pam Wilkins
Robin Gleed
Alan Dobson
College of Veterinary Medicine
Cornell University, Ithaca, New York 14853
E-mail: rdg2@cornell.edu

Nikolai Krivitski
Transonic Systems, Ithaca, New York 14850