


Susan R. Hopkins
Division of Physiology
Department of Medicine
Pulmonary Imaging Laboratory
Department of Radiology
University of California
San Diego, La Jolla, California

e-mail: shopkins@ucsd.edu

COUNTERPOINT: PULMONARY EDEMA DOES NOT OCCUR IN HUMAN ATHLETES PERFORMING HEAVY SEA-LEVEL EXERCISE

The arterial partial pressure of oxygen (\(P_{aO_2}\)) can be reduced by as much as 30 mmHg and oxyhemoglobin saturation (\(S_aO_2\)) by 15% below resting levels in some aerobically trained athletes exercising at high intensities (3). Some have hypothesized that mild interstitial edema could develop and, through changes in local compliance (e.g., alveolar wall edema) or resistance (e.g., perivascular or bronchial edema), affect the distribution of ventilation or blood flow. However, the measurement of lung density in humans in vivo is not possible, so any discussion of whether transient pulmonary edema exists is dependent on the validity and sensitivity of the tools used to estimate the change in lung water. One must also consider the pathophysiology, or at least the physiology, that would lead to pulmonary edema in human athletes at sea level. Possible mechanisms in this population are limited but include 1) increased pulmonary capillary permeability secondary to leakage (starling forces) or damage (capillary stress failure); 2) increased pulmonary capillary surface area; 3) increased pulmonary capillary hydrostatic pressure; and 4) lymphatic insufficiency. Others have argued that it is unlikely that exercise at sea level would result in pulmonary edema because there are important “edema safety factors” that exist to oppose its formation (16, 19). Here we present the relevant studies that have been unsuccessful in documenting pulmonary edema.

Repeated exercise. Repeated heavy exercise does not worsen gas exchange (13, 18) in athletes. In fact, gas exchange impairment can be lessened by prior exercise (18). This shows that impairments to gas exchange are not caused by a mechanism that persists after the initial exercise period and is not aggravated by subsequent exercise, as would be expected of structural alterations to the blood-gas interface.

Transthoracic electrical impedance. Reductions in transthoracic electrical impedance (TEI) following exercise have been reported as related to the accumulation of lung water (1). The physiological importance of these findings remain unclear, because the measurement of TEI is nonspecific, it does not explain the source of hindered impedance, and may be due to elevated intravascular volume not interstitial edema. Furthermore, others (15) have reported increased TEI following maximal exercise.

Chest radiography. There are reports of pulmonary edema in two athletes competing in a 90-km running race (12). While the clinical manifestations of pulmonary edema appear present, two points should be emphasized. First, the pulmonary pressures would not be significantly elevated during this type of exercise and the possibility of “stress failure” is low. Second, ultra-distance running races occur worldwide without widespread reports of pulmonary edema, suggesting that the presence of edema was more likely to be related to undiagnosed pathology. Chest radiographs were scored following three sets of 5-min of heavy cycle exercise with an increase in a semi-quantitative edema score (20). However, the pre-exercise values were not zero (i.e., no edema present) and calls into question the meaningfulness of the scale. Changes in the magnitude of extravascular water needs to be ~35% to be detected by chest radiography (17), implying a pathology not seen in a healthy, athletic population.

99mTc-labeled diethylenetriaminepentaacetic acid. Usage of 99mTc-labeled diethylenetriaminepentaacetic acid (99mTc-DTPA) is a sensitive method for detecting lung pathology. Edwards et al. (4) found that pulmonary clearance of 99mTc-DTPA after maximal cycle exercise was unchanged. Furthermore, there was no relationship between pulmonary clearance and the fall in \(P_{aO_2}\) (~27 mmHg), suggesting that hypoxemia occurs despite maintenance of alveolar epithelial integrity. Conversely, following 6 min of maximal rowing, clearance appears to be increased (6). However, this is not direct evidence of edema. Rather, it could reflect a change in epithelial permeability, meaning that the blood-gas barrier is simply more permeable or it may be attributable to an increase in pulmonary capillary blood volume and the associated increase in pulmonary surface area.

Computed tomography. Computed tomography (CT) allows a rapid image of the lung, but because it exposes the subjects to ionizing radiation, it’s usefulness as a research tool in healthy, young athletes is limited. Following completion of a triathlon race, CT scans revealed a 19 and 21% increase in lung density and mass, respectively (2). However, heart rate was higher during post-race scans and the number of veins and venules was increased reflecting increases in pulmonary blood flow. Also, visual analysis of CT scans did not reveal an obvious image of acute pulmonary edema. We recently conducted two studies (5, 11) where the exercise protocol was
designed to maximize exertional effort. This was coupled with a hypoxic stressor that would increase the likelihood of extravascular water accumulation by heterogeneously increasing pulmonary artery pressure via the hypoxic vasoconstriction response. Despite a carefully designed protocol and objective imaging technique there was no increase in lung density.

**Magnetic resonance imaging.** Advances in magnetic resonance imaging (MRI) have opened the door to more sensitive measurements of lung physiology (8). MR scanners were developed for clinical use, and images are obtained in supine or occasionally prone positions and the effects of gravity and body position have influences on the measurement of lung water. Moreover, validity has never been established with this technology and it lacks a standard to which it can be fully compared. In addition, inherent in the measurement of lung density in a healthy athletic population is the acquisition time. In our experience, imaging is gated to heart rate and trained individuals with bradycardia take longer to scan. If pulmonary edema exists during and postexercise in this group surely it is transient in nature. MR imaging does not give a snapshot of lung density but rather an average value of the data accumulated over time (30–60 min). In a healthy young athletic group, restoration of “normal physiology” is rapid. Lymphatic clearance is increased with exercise, and any additional extracellular lung water may be gone before the MR imaging can detect the changes in lung density. We reported a 9.4% increase in extravascular lung water following 45-min cycle exercise using MRI (14). Although statistically significant, the increase in lung density was primarily driven by two subjects. In a separate study using similar imaging techniques, we failed to detect any evidence of pulmonary edema following exercise (7), emphasizing the uniqueness of these two subjects and that generalizations should be limited.

**Bronchoalveolar lavage fluid.** Red blood cells and protein have been recovered from bronchoalveolar lavage (BAL) fluid from six athletes after 7 min of heavy exercise compared with four resting sedentary controls (9). The results showed that the athletes had higher concentrations of red blood cells, total protein, and leukotriene B4 in their BAL fluid than the controls. On the surface, these findings imply a loss of blood-gas barrier integrity. However, the subjects all reported previous postexercise hemoptysis, making them a unique subgroup rather than a physiological “rule.” In addition, red blood cells were observed in one control subject from this study as well as five of eight control subjects in a separate study by the same research group (10). Despite the care taken with this invasive procedure, the presence of red blood cells in the controls probably reflects abrasion of the airways during bronchoscopy and emphasizes the likelihood of experimental artifact.

In sum, attempts to determine the presence of pulmonary edema in humans with modern, objective imaging techniques and other methods have failed to provide supportive evidence for its existence.

**REFERENCES**


