Maximal rowing has an acute effect on the blood-gas barrier in elite athletes

Birgitte Hanel,1 Ian Law,1,2 and Jann Mortensen1

1Department of Clinical Physiology and Nuclear Medicine, Copenhagen University Hospital, Rigshospitalet, and 2Department of Clinical Physiology and Nuclear Medicine, Copenhagen University Hospital, Hvidovre Hospital, DK-2100 Copenhagen, Denmark

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Address for reprint requests and other correspondence: B. Hanel, Dept. of Clinical Physiology and Nuclear Medicine, 4011, Copenhagen University Hospital, Rigshospitalet, Blegdamsvej 9, DK-2100 Copenhagen Ø, Denmark (E-mail: bhanel@mobilixnet.dk).

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THere is evidence to suggest that the integrity of the lung blood-gas barrier (BGB) in elite athletes is altered after short-term maximal exercise (19). The finding of red blood cells and protein in the fluid of bronchoalveolar lavage suggests that this effect may be caused by mechanical stress on the alveolar capillary membrane. In contrast, sustained submaximal exercise [77% of maximal O2 uptake (VO2 max)] in elite athletes did not elicit the same ultrastructural changes (18). Hence, only extreme levels of exercise in elite athletes may disrupt the integrity of the BGB, with possibly secondary development of interstitial edema. During maximal exercise, the BGB is subjected to conflicting requirements. A thin barrier is most optimal for efficient gas exchange by diffusion, whereas a strong barrier is needed to withstand the increased pulmonary capillary pressure and the longitudinal tension in the alveolar wall associated with lung inflation (42). If any injury should occur, it may cause these layers to become leaky, thereby increasing permeability.

A very sensitive method to evaluate the BGB permeability is measurement of the pulmonary clearance rate using 99mTc-labeled diethylenetriaminepentaacetic acid (DTPA) (22). Nonpolar molecules, most of which are gases, are lipophilic and penetrate the BGB very quickly. The clearance of such solutes from the lungs is limited by perfusion (4), with the exception of carbon monoxide, which is diffusion limited because of high binding affinity of hemoglobin (3). Polar molecules such as the hydrophilic 99mTc-DTPA solute do not pass the BGB so easily and are normally limited by passive diffusion (33) through the intercellular tight junctions of the alveolar epithelium and the capillary endothelium. The permeability of the alveolar epithelium to solutes is 10 times less than that of the capillary endothelium, regardless of the molecular weight (38). The alveolar epithelium is therefore the rate-limiting membrane, preventing fluid and solute transport from the capillary to the alveolar space. This is due to the pore radius, which has been reported to be 0.8–1.0 and 4.0–8.0 nm for the alveolar epithelial and capillary endothelium tight junctions, respectively (39). 99mTc-DTPA, with a molecular radius of 0.6 nm, is able to pass through the pores of the alveolar epithelium.

At rest, pulmonary clearance is increased if the epithelial cell barrier is damaged, as seen in a variety of lung diseases (43), including acute asthma (24), interstitial lung diseases (33), and acute respiratory distress syndrome (21) and in cigarette smokers (20). However, pulmonary diseases and toxic exposure are not the only factors influencing pulmonary clearance. During exercise, increased tidal volume leads to increased inflation of the alveoli and increased pulmonary epithelial permeability (30). The increase in capillary pressure and inflation of the lungs during maximal exercise may result in ultrastructural changes in the BGB and would likely be detected as an increased...
pulmonary clearance rate of $^{99m}$Tc-DTPA as a result of an increased permeability of the alveolar epithelia. The pulmonary clearance rate of $^{99m}$Tc-DTPA was increased by 170% 25 min after a 75-min exercise bout at 75% $\dot{V}O_2\text{max}$ (25). In contrast, Edwards et al. (9) found the pulmonary clearance rate of $^{99m}$Tc-DTPA unchanged 38 min after incremental exercise to exhaustion in highly trained male cyclists. However, inasmuch as the temporal development of BGB permeability change is unknown, increases immediately after exercise cannot be excluded.

Hence, the purpose of the present study was to evaluate the permeability of the alveolar epithelial membrane in elite athletes immediately after maximal exercise and in the late recovery phase.

METHODS

Subjects. Three female and seven male nonsmoking oarsmen with no history of cardiovascular or pulmonary diseases participated in the study. All subjects competed in rowing on an international level, five of them receiving medals. One subject won the world championship for lightweight rowers (1994, 1997–1999), one Olympic gold medal (1996), one bronze medal (2000), and one silver medal (2001). Another subject won the bronze medal at the world championship for lightweight rowers (2000) and one silver medal (2001). Three rowers won world championship silver medals (2001). The last five subjects were among the participants in the finals at the world championship (2000 or 2001).

Each subject gave informed written consent, and the procedures were approved by the local Ethics Committee of Copenhagen (KF 01-180/99).

Preliminary tests. The preliminary tests were performed to characterize lung function and to identify and subsequently exclude subjects suffering from asthma. The measurements took place on 3 separate days; the experimental protocol was executed on a 4th and a 5th day.

The subjects refrained from vigorous activity for 12 h before reporting to the laboratory. With the subject in the seated position, static and dynamic lung function and single-breath diffusion capacity (23) (MasterLab Jäger, Wurtzburg, Germany) were measured, and a histamine challenge test (6) was performed at rest. The histamine challenge test was carried out with a Wright nebulizer (Aerosol Products, London, UK). The first aerosol, a saline solution (0.9%), was followed by increasing concentrations of histamine (0.075–8 mg/ml). Each aerosol was inhaled by tidal breathing for 2 min. Nonspecific bronchial hyperresponsiveness was defined as a concentration of histamine (<8 mg/ml) causing a 20% decline in forced expiratory volume in 1 s (FEV1; Vitalograph). To exclude subjects suffering from exercise-induced asthma, the effect from rowing was evaluated on a separate day by measurement of FEV1 before and after rowing. Exercise-induced asthma was defined as a 10–15% decrease in the preexercise value of FEV1 at 5–15 min after exercise (14). The room temperature was 21–22°C, and humidity was 40–50%. $\dot{V}O_2\text{max}$ and ventilation were measured during a 6-min all-out row on a separate occasion with an automated metabolic cart (AMIS 2001, Inovision, Odense, Denmark). Heart rate was recorded on a Polar heart rate monitor (Vantage XL). Rowing was performed on a wind-braked ergometer (Concept 2, Morrisville, VT), and power output was obtained from a computer (Concept 2).

Experimental protocol. All subjects performed the rowing protocol (postexercise) and reappeared on a separate day for a control evaluation without rowing (control).

After a warm-up period at their own pace, the subjects performed a 6-min all-out row or a 2,000-m race, which corresponded to 6.1–6.5 min of maximal exercise. Immediately after termination of the rowing bout, the subject was placed in the supine position and then inhaled $^{99m}$Tc-DTPA aerosol for an average of 2.5 min. The aerosol was generated from a 400-MBq (1st inhalation) and an 800-MBq (2nd inhalation) $^{99m}$Tc-DTPA solution in 5 ml of 0.9% sodium chloride using a jet nebulizer (Swirler Nebulizer, AMICI) at a flow rate of 9 l/min. A dose of 20 or 40 MBq, respectively, was deposited in the subjects. The binding percentage of $^{99m}$Tc-DTPA was 97–99%. Radioactivity from the chest was detected in subjects positioned in the supine position above a gamma camera with a circular, low-energy, high-resolution collimator (General Electric) linked to a computer. The energy was set to 140 keV with a 20% window setting. Data were acquired as a 20-min dynamic acquisition (128 $\times$ 128 pixels) in 10-s frames (initial measurement). The time between acquisition and the completion of image acquisition was on average 9 ± 0.8 min. After 2 h, the subjects performed a repeated inhalation of $^{99m}$Tc-DTPA, which was followed by a new measurement of pulmonary clearance (late measurement). To image the ventilation distribution and the lung edges, $^{81}$Kr (500 MBq) was inhaled. $^{81}$Kr was inhaled 35 min (4 subjects) or 2.5 h (4 subjects) after rowing or on the control day (2 subjects). Because of the infrequent delivery of the Kr generator, it was not possible to perform this inhalation within the same time scale in all the subjects. $^{81}$Kr was continuously inhaled from an $^{81}$Rb$^{81}$Kr generator (produced in our Cyclotron unit). The above-mentioned camera was used to image posterior activity from the chest as a 1-min static acquisition. The energy was set to 190 keV with a 20% window setting. To compensate for the associated water loss during the 3.5-h examination with associated strenuous exercise, subjects were allowed to drink water during the experiment. Moreover, three subjects returned to the laboratory on a 6th day to perform a 6-min all-out row for evaluation of the supine recovery of the tidal volume (MasterLab Jäger).

Data analysis. For the 20-min lung clearance measurement, all frames were integrated to yield one static image. Two regions of interest (ROIs) were drawn over the peripheral one-third area from both lungs and subsequently projected back onto the dynamic image sequence for generation of the time-activity curve. A ROI was placed outside the right and left lungs to correct for the background activity arising from the chest wall tissue. For each subject, the same ROIs were applied to all dynamic data. For correction of residual background activity from the first inhalation of radio aerosol, the pulmonary clearance measurement was repeated 30 min before the second inhalation of radio aerosol. The residual radioactivity was subtracted from the late measurement. The time-activity curves obtained from the ROIs were fitted by a monoexponential function (Fig. 1), with the negative slope of the line being the rate constant of clearance, expressed in terms of percent decrease in activity (%/min). For each measurement (initial and late), lung clearance was calculated for 0–7 and 10–20 min, i.e., four time intervals (0–7, 10–20, 125–135, and 135–145 min). Scintigrams were processed from the initial 3 min of gamma camera acquisition from the initial and late measurements of $^{99m}$Tc-DTPA clearance. The homogeneity of the radio aerosol distribution during all measurements and comparisons between measurements and conditions were evaluated blindly and randomized by a nuclear
A clearances were measured, the tidal volume had to be calculated for each subject. Each curve was fitted by monoexponential functions, and lung clearance was calculated for 0–7 and 10–20 min. cps. Counts per second.

Statistical analysis. Two-tailed paired t-tests were performed for identification of changes in respiratory variables. The clearance rates were analyzed using repeated-measures ANOVA. The factors were measurement with four levels (4 time intervals) and condition with two levels (control vs. postexercise). The significance threshold was $P < 0.05$.

RESULTS

Subjects. One male subject was excluded because of technical difficulties during the measurement of $^{99m}$Tc-DTPA clearance in the control situation. The characteristics of the remaining 10 subjects are presented in Tables 1 and 2.

Preliminary tests. All ventilatory capacities and diffusion capacities were within normal limits, and bronchial hyperresponsiveness to histamine or bronchoconstriction after exercise could not be demonstrated in any subject. Thus it was not necessary to exclude subjects from the study because of this criterion.

Recovery of tidal volume. When the initial pulmonary clearance was measured, the tidal volume had returned to resting values (1.16 $\pm$ 0.19 and 1.17 $\pm$ 0.23 liters before and 10 min after exercise, respectively), indicating no active stretching of the alveolar epithelia during the measurements (12).

Pulmonary $^{99m}$Tc-DTPA clearance. The ANOVA showed a significant overall decrease of pulmonary clearance rates over time [measurement, 4 time intervals: $F(3, 27) = 20.3, P < 0.0001$]. Furthermore, the pulmonary clearance rates were overall significantly greater after exercise than in the control condition [$F(1, 9) = 11.3, P < 0.01$], and there was a significant difference in the development of the pulmonary clearance rates over time for the two conditions [$F(3, 27) = 9.2, P < 0.0005$; Fig. 2]. Two post hoc repeated-measures ANOVA showed that a significant decrease of the pulmonary clearance rates over time could be found only after exercise [$F(3, 27) = 20.6, P < 0.0001$], whereas there were no significant spontaneous changes in the control condition [$F(3, 27) = 2.7, P = 0.07$]. Furthermore, two-tailed paired t-tests at each of the four measurement intervals revealed significant increases in the pulmonary clearance rates after exercise compared with control for the two first measurement time intervals only [$t = 3.7$, degrees of freedom ($df$) = 9, $P < 0.01$ and $t = 5.6$, $df$ = 9, $P < 0.0005$, respectively], whereas analyses from the last two measurement time intervals were not significantly different ($t = 1.0$, $df = 9$, $P = 0.34$ and $t = 0.1$, $df = 9$, $P = 0.90$).

Table 1. Descriptive characteristics of subjects and maximal performance

<table>
<thead>
<tr>
<th></th>
<th>Men ($n = 7$)</th>
<th>Women ($n = 3$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>22.3 $\pm$ 3.2</td>
<td>29.4 $\pm$ 4.0</td>
</tr>
<tr>
<td>Height, cm</td>
<td>183.0 $\pm$ 4.8</td>
<td>173.5 $\pm$ 7.8</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>80.9 $\pm$ 7.6</td>
<td>69.9 $\pm$ 10.1</td>
</tr>
<tr>
<td>FVC, liters</td>
<td>6.5 $\pm$ 0.7</td>
<td>4.8 $\pm$ 0.6</td>
</tr>
<tr>
<td>FEV$_1$, liters</td>
<td>5.3 $\pm$ 0.4</td>
<td>4.0 $\pm$ 0.3</td>
</tr>
<tr>
<td>FEV$_1$/FVC, $%$</td>
<td>82.8 $\pm$ 6.0</td>
<td>82.3 $\pm$ 4.2</td>
</tr>
<tr>
<td>RV, liters</td>
<td>1.9 $\pm$ 0.4</td>
<td>1.5 $\pm$ 0.1</td>
</tr>
<tr>
<td>TLC, liters</td>
<td>8.5 $\pm$ 1.0</td>
<td>6.5 $\pm$ 0.6</td>
</tr>
<tr>
<td>$D_l$, mmol-min$^{-1}$-kPa$^{-1}$</td>
<td>14.8 $\pm$ 0.7</td>
<td>10.2 $\pm$ 1.7</td>
</tr>
<tr>
<td>$V_O_2$, l/min</td>
<td>5.9 $\pm$ 0.3</td>
<td>4.0 $\pm$ 0.7</td>
</tr>
<tr>
<td>$V_O_2$max, l/min</td>
<td>193.6 $\pm$ 15.3</td>
<td>133.0 $\pm$ 11.3</td>
</tr>
<tr>
<td>Power, W</td>
<td>384.8 $\pm$ 27.5</td>
<td>290.0 $\pm$ 70.7</td>
</tr>
</tbody>
</table>

Values are means $\pm$ SD. FVC, forced vital capacity; FEV$_1$, forced expiratory volume in 1 s; RV, residual volume; TLC, total lung capacity; $D_l$, pulmonary diffusing capacity; $V_O_2$max, maximal $O_2$ consumption; $V_E$, maximal minute ventilation.

![Fig. 1. Time-activity curves from initial diethylenetriaminepentaacetic acid (DTPA) measurement in control (A) and postexercise (B) conditions from 1 subject. Each curve was fitted by monoexponential functions, and lung clearance was calculated for 0–7 and 10–20 min. cps. Counts per second.](image)

![Fig. 2. Pulmonary clearance during initial (0–7 and 10–20 min) and late (125–132 and 135–145 min) measurement in control and postexercise conditions. $^*P < 0.01$, $^{**}P < 0.0005$ vs. control.](image)
0.95, respectively). Hence, it is the increase of the pulmonary clearance rates during exercise of the first two measurements that primarily drives the differences. It appears as if there is a decrease in pulmonary clearance rates between 0–7 and 10–20 min after exercise (Fig. 2). However, an additional ANOVA could not distinguish this change from the spontaneous nonsignificant change in the control condition \( F(1, 9) = 2.1, P = 0.18 \).

In conclusion, the pulmonary clearance rates were increased significantly immediately after 6 min of all-out rowing compared with control and subsequently normalized after 2 h. There were no significant spontaneous changes in pulmonary clearance rates in the control condition. The decrease in the average lung clearance rates between 0–7 and 10–20 min after exercise was not significant.

**Scintigrams.** In 6 of 10 subjects, ventilation distribution on the lung \(^{99m}\)Tc-DTPA scintigrams was inhomogeneous immediately after rowing. Thus three scintigrams were classified as having small defects and three as having moderate defects. At 2 h after exercise, all \(^{99m}\)Tc-DTPA scintigrams were classified as normal, except for two that still had small defects. On the control day, all \(^{99m}\)Tc-DTPA scintigrams were normal during the initial measurement, and one had small defects during the late measurement. Among the four subjects who inhaled \(^{81m}\)Kr gas 35 min after exercise, two had no defects and two had large defects (Fig. 3). The \(^{81m}\)Kr scintigrams were normal with no defects 2.5 h (4 subjects) after rowing or on the control day (2 subjects).

**DISCUSSION**

The present study demonstrates that the pulmonary clearance of \(^{99m}\)Tc-DTPA (%/min) was doubled after 6 min of all-out rowing compared with control. At 2 h after rowing, the pulmonary clearance had normalized. Moreover, lung scintigrams in 6 of 10 subjects showed small-to-large rowing-induced ventilation defects. There was no clear relation between the individual increase in pulmonary clearance and the degree of ventilation defects (no, small, moderate, or large defects).

Evaluation of DTPA clearance is a simple noninvasive procedure that is very sensitive to acute as well as chronic changes in lung permeability (43). The precise mechanism behind the accelerated clearance of \(^{99m}\)Tc-DTPA after severe exercise cannot be explained on the basis of the present study alone, where only an increased permeability associated with alterations in the uniformity of ventilation has been demonstrated. When the epithelial permeability is increased, it does not necessarily indicate interstitial edema, but the BGB is more permeable to fluid and solute transport.

Two physiological mechanisms may explain the increased permeability after exercise: 1) increased tidal volumes and 2) elevated pulmonary arterial pressures. The increased tidal volume during exercise may have resulted in an alteration of the alveolar epithelium stretching the intercellular tight junctions. Animal studies show that volume-induced increases in lung epithelial solute permeability are not reversed immediately at lower volumes (10). Thus, despite unchanged resting tidal volume in our study, the tight junctions may still have a larger pore radius, increasing the pulmonary clearance. This is in agreement with an increased clearance 25 min after exercise, which was found to correlate with the increased tidal volume during exercise (25). Although a measurement of functional residual capacity during recovery would have provided supportive data, this could not be sampled in the supine position in the body plethysmography. Edwards et al. (9) demonstrated no changes in the pulmonary clearance of \(^{99m}\)Tc-DTPA after increased iso-capnic ventilation without exercise. On the basis of these findings, the authors suggested that the pulmonary arterial pressure is an important factor in the change in alveolar permeability during exercise and argue against a ventilation-related mechanism for increased pulmonary permeability. It is noteworthy that maximal rowing produces large increases in the pulse pressure influenced by a Vasalva-like maneuver (5). Hence, the effective pulse pressure, which was coupled...
with the stroke rate, was two to three times the magnitude of the normal pulse pressure. It seems reasonable, therefore, to attribute the increased pulmonary clearance to the increase in pulmonary arterial pressure and consequent disruption of the alveolar epithelial layer and/or the capillary endothelium (41, 42). If the injury creates “holes” large enough to leak protein, alveolar edema and hemorrhage will ensue, but if the leakage is minimal, no clinical effect can be identified (22).

In our study, we have demonstrated an increase in the pulmonary clearance of ~100% from 9 min after exercise and in the following 20 min. Manier et al. (26) found a 7.5% increase in pulmonary capillary blood volume 15 min after exercise, decreasing to resting values within 30 min. It can be speculated that the increase in pulmonary clearance is partially caused by an increase in pulmonary capillary blood volume and the associated pulmonary surface area. The size of this effect or whether it contributes at all is unknown but will maximally correspond to the size of the increase in pulmonary capillary blood volume, with the assumptions that capillaries are completely “recruited” and pulmonary clearance is present in these segments. This means that a 10 or 20% increase in pulmonary capillary blood volume maximally accounts for 10 or 20% in pulmonary clearance, with the assumption that the pulmonary clearance increases in proportion to the capillary surface area. Hence, the dynamic changes in pulmonary capillary blood volume cannot fully account for the changes in pulmonary clearance.

We found nonuniform ventilation distribution in the lungs, indicating constriction of the airways immediately after exercise, as shown by the study of Schaffartzik et al. (35), who found ventilation-perfusion inequality in the early recovery from heavy exercise. The lack of increased bronchial responsiveness excludes asthmatic disease. The exact mechanism behind these changes cannot be directly determined from our data. One possibility could be the presence of minor structural differences in the airways, which, although unimportant during rest, becomes significant during maximal exercise. However, the persistent nonuniform ventilation distribution after ventilation recovery does not support this notion. An alternative possibility could be mucus secretion from irritated airways caused by the high flow rates through the activation of broncho- or vasoactive mediators (2). Furthermore, heterogeneous ventilation may reflect airway compression by interstitial edema secondary to the increased alveolar epithelial permeability. The decreases in the membrane component of the lung diffusion capacity in the hours after exercise have been interpreted as transient interstitial edema (27, 31, 37, 40). However, other studies using chest radiography (13) or computed tomography and magnetic resonance imaging (29) have failed to provide visual evidence of such edema. The lack of effect of diuretics on lung diffusion capacity 2 h after exercise (16) suggests that the decreased membrane component reflects an acute stress failure causing damage to the BGB. Moreover, the finding of red blood cells and protein after exercise in bronchoalveolar lavage fluid is consistent with an altered BGB (19). Indirect evidence supports the theory of stress failure in the human BGB, including case studies of human athletes who have developed acute pulmonary edema as documented by chest radiography (28, 44). Additionally, acute cough and oppression in the chest after maximal exercise are well-known phenomena after maximal rowing (32).

Besides processing the 99mTc-DTPA scintigrams, we also processed scintigrams after inhalation of the 81mKr gas to obtain an image of the ventilation distribution at the lung periphery. This gas consists of atoms that are smaller than the 99mTc-DTPA droplets and is distributed to the smallest airways. The insoluble inert 81mKr gas has a short half-life (13 s), and it reflects distribution of ventilation. The deposited 99mTc-DTPA aerosol also mainly reflects distribution of ventilation, but a central deposition of radioactivity may occur in patients with airway obstructions, such as chronic obstructive pulmonary disease and asthma, or if the particle size is too large. Hence, 81mKr and 99mTc-DTPA scintigrams are rather comparable, but the distribution of ventilation is most accurately visualized by the 81mKr scintigrams. Although the time delay between the 99mTc-DTPA scintigrams (obtained 9–12 min after exercise) and the 81mKr scintigrams (obtained in 4 subjects 35 min after exercise) makes a comparison difficult, the results from the two methods were in overall agreement. Thus the same two subjects with defects on 81mKr scintigrams showed similar defects on the 99mTc-DTPA scintigrams, and the remaining two subjects did not demonstrate defects on either of the scintigrams.

The pulmonary clearance rate in the control condition in our subjects was within the limits for normal nonsmoking healthy subjects (36). Correction for recirculation has been evaluated (34, 15), and the authors concluded that because recirculation during rest never contributed to >2% of the initial radioactivity, it was without practical importance.

There are several technical issues to be considered in performing and interpreting Tc-DTPA scintigrams. The distribution of inhaled 99mTc-DTPA in the lungs is an important factor for measurement of the pulmonary clearance. If primarily centrally deposited, 99mTc-DTPA partly could have been partially cleared by mucociliary action. To avoid central distribution, the flow rate should be <36 l/min, the particles size <2 μm, and inspiratory volume slightly deeper than normal (1). All these conditions were met in our study. Theoretically, pulmonary clearance can be affected by the pulmonary perfusion, but 99mTc-DTPA is still cleared in severe pulmonary embolism and only increases modestly if the pulmonary artery blood flow is increased (34). We did not monitor heart rate after exercise during measurement of pulmonary clearance, and a higher blood flow could have been present. The alveolar epithelial membrane is the rate-limiting membrane because of the pore radius (38). Polar molecules such as the hy-
drophilic 99mTc-DTPA solute are limited by passive diffusion.

The radiochemical purity of 99mTc-DTPA is the most important quality problem, because pulmonary clearance is five times faster for free pertechnetate (99mTcO4) than for 99mTc-DTPA. Our in vitro binding of 99mTc-DTPA was 97–99%; thus <3% was 99mTcO4. We did not observe any accumulation of 99mTcO4 in the thyroid gland or stomach, which could have been the case if a large amount of 99mTcO4 was present.

The possibility of an increased pulmonary clearance due to a depletion of surfactant has been discussed in the study of Groth et al. (15). Inasmuch as the surfactant levels were not explicitly measured in this study, this factor cannot be excluded completely as a contributor to the increased pulmonary clearance.

Conflicting conclusions in studies of the recovery of the BGB may depend on the time of measurement; i.e., the disruptions of the BGB can be “closed” within a few minutes of reduction of the capillary transmural pressure (11). Therefore, Edwards et al. (9) did not see any changes ~38 min after maximal exercise in elite athletes, indicating that the membrane at that time may have already recovered. Furthermore, recovery of the BGB may depend on the magnitude of the stress failure, i.e., the intensity of exercise and, thereby, pulmonary capillary pressure. In the present study, the pulmonary clearance measurement started 9 min after termination of a 6-min all-out exercise in elite rowers able to produce very high pulmonary capillary pressures. We found that the pulmonary clearance was still increased at 10–20 min and normalized after 2 h. This suggests that resolution of the ventilation inhomogeneity begins ~30 min after exercise, in agreement with the study of Schaffartzik et al. (35). Two subjects still had small defects on the lung scintigrams 2 h after exercise, despite a normalized pulmonary clearance, indicating defects, which require more time to recover. The problem with leaky lungs in elite athletes could be an additional factor leading to the acute exercise-induced hypoxemia observed during maximal exercise in many highly fit humans (8). However, a long-lasting effect does not seem to take place. Hence, the increased alveolar-arterial O2 difference causing the exercise-induced hypoxemia was not aggravated 20 min later during a repeated 2-min maximal treadmill run, and arterial PO2 and arterial O2 saturation were increased during the subsequent exercise (7).

Likewise, in the study by Hanel et al. (16), VO2 max and work capacity were the same during two all-out rows interspersed by 2 h, with arterial PO2 and, in turn, arterial O2 saturation, being slightly higher in the 2nd min of the repeated row. This points to an exercise-induced increased permeability of the BGB that persists for a short time after exercise, in agreement with a fast remodeling of BGB and recovery of the interstitial pulmonary edema in parallel with restoration of the plasma volume (17), without causing any acute physiological consequence for performance in elite athletes.

In conclusion, we have demonstrated that the BGB is more leaky during the early recovery after a 6-min all-out rowing bout in highly trained rowers, causing ventilation defects in some subjects. These observations indicate that the alveolar capillary barrier may be disrupted with development of increased permeability and interstitial edema.

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DISCLOSURES

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