Pulmonary Hemodynamics during a Strenuous Intermittent Exercise in Healthy Subjects

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ABSTRACT

LONSDORFER-WOLF, E., RICHARD, S. DOUTRELEAU, V. L. BILLAT, M. OSWALD-MAMMOSSER, and J. LONSDORFER. Pulmonary Hemodynamics during a Strenuous Intermittent Exercise in Healthy Subjects. Med. Sci. Sports Exerc., Vol. 35, No. 11, pp. 1866–1874, 2003. Purpose: It has been suggested that an intermittent work exercise test (IWET) is as efficient but better tolerated than continuous exercise for rehabilitation. Although systemic and pulmonary cardiovascular adjustments have been investigated for continuous exercise, it has not been done for IWET with exercise bouts near maximal work rate. Methods: In seven healthy subjects, the pulmonary hemodynamics have been studied by the aid of heart catheterization during a strenuous 30-min bicycle IWET where a 4-min work set at the first ventilatory threshold (VT1) alternated with a 1-min work set at the second ventilatory threshold (VT2). Results: During the IWET, cardiac output increased then remained stable with decreasing stroke volume and increasing heart rate, which became near maximal at the end of the test. Mean pulmonary arterial pressure increased from rest to the fifth minute of exercise and decreased significantly thereafter (P < 0.01). An identical evolution was observed for mean systemic arterial pressure (SAP). Conclusion: Pulmonary hemodynamics adapt well in healthy subjects during a strenuous IWET despite the performance of exercise bouts of near maximal intensity. Key Words: INTERMITTENT WORK EXERCISE TEST, MEAN PULMONARY ARTERIAL PRESSURE, CARDIAC OUTPUT, STROKE VOLUME, VO2MAX

Physical training of an aerobic or endurance type is the most appropriate kind for improving a subject’s ability to sustain physical activity (2,5). Although there is a consensus on the duration and frequency of the training sessions, two types of training are usually proposed: continuous submaximal exercise or intermittent exercise (also referred to as interval exercise). The latter involves repeated bouts of hard work alternating with relief consisting of a rest or a light working period. It could thus be preferred because the relief period has been shown to delay fatigue (3) and allow higher expenditure during the hard work periods (3). This allows the achievement of greater amounts of total work with reduced metabolic demands (2–4).

Interval training has been performed with efficiency in heart transplant recipients in our laboratory (10,17–19). The “tailored” 6-wk endurance training program, derived from the square wave endurance exercise test of Gimenez et al. (12) led to significant improvements, with an increase of aerobic work capacity (19), lactate removal (18), and changes in muscular ultrastructural characteristics that were similar to age-matched controls under the same relative intensity training (17). Such exercise rehabilitation is also proposed to patients with chronic obstructive pulmonary disease (COPD) (11). In the latter patients, there remains nevertheless the question of whether repeated bouts of hard work might not induce a dramatic increase in mean pulmonary arterial pressure (PAP), especially when they are chronically hypoxic. In the present study, we report the pulmonary hemodynamic behavior during interval exercise in healthy subjects in order to document the normal pulmonary vascular response to an intermittent work exercise test (IWET) to gain better understanding of that response during a similar test in COPD patients.

METHODS

Subjects

Seven healthy nonsmoking male volunteers (38 ± 5 yr) participated in the study after having given their written informed consent. None of the subjects had cardiac or pulmonary disease and none was on any medication. Two of them trained in aerobic sports to the amateur level. The local Ethics Committee approved the experimental protocol.
Exercise Tests

All subjects underwent two types of exercise: an incremental exercise test (IET) and an intermittent work exercise test (IWET). These two tests were performed on the same day, in the same conditions, i.e., with pulmonary and arterial catheterization and in a constant temperature air-conditioned room. The IET was performed in the morning 2 h after a light breakfast, and the IWET in the afternoon after...
a 3-h rest during which they had a light lunch (Fig. 1A). The IET was performed first, in order to determine the ventilatory thresholds used to determine the work rates of the IWET.

For right heart catheterization, a floated fluid-filled catheter (Flexopulmocath 4F, Plastimed, Saint-Leu-la-Forêt, France) was inserted into the pulmonary artery under local anesthesia through a brachial vein, and in the supine position. Monitoring the pressure curves (Physiogard SM 785, Brucker, France) permitted to follow the progression of the catheter. Mean PAP was recorded, and mixed venous blood samples could be obtained through the catheter. To measure mean systemic arterial pressures (SAP), a pediatric arterial catheter (Seldinger catheter 20G-8 cm, Vygon, Ecouen, France) was inserted under local anesthesia into a radial artery. Arterial blood was sampled in this way.

After insertion of the catheters, the subjects rested for half an hour and were then seated upright on an electronically braked cycle ergometer (Medifit 1000 S, Medifit, Maarn, The Netherlands). In this position, the reference point for the pressure transducers was set laterally midway between the lower line of the axillary and the last rib. Seat and handlebar settings were adapted to each subject for the two tests. The pedaling frequency was 70 rev-min⁻¹ and was maintained constant during the tests.

Incremental Exercise Test

Each subject performed an IET up to exhaustion to determine his maximal tolerated power and the ventilatory thresholds, VT₁ and VT₂ (see below). After a 3-min warm-up period (52 ± 7 W), charge increments of 20 or 30 W·min⁻¹ were used, depending on the subject’s weight and training level, in order that the exercise duration not exceed 20 min. Hydration was estimated from the blood volume (BV) and plasma volume (PV) calculated using the Dill and Costill’s method (8).

Intermittent Work Exercise Test

After a 3-min warm-up set at the same power as for the IET, the 30-min IWET was performed. It consisted of six stages of 5 min, with 4 min of cycling at a power output corresponding to the first ventilatory threshold (VT₁) called “base,” alternating with 1 min of cycling at a power corresponding to the second ventilatory threshold (VT₂) called “peak.”

Measurements

Ventilatory parameters. During the entire investigation, minute ventilation (VE), O₂ uptake (VO₂), and CO₂ output (VCO₂) were measured on a breath-by-breath basis by means of an open-circuit metabolic chart with rapid O₂ and CO₂ analyzers (breath-by-breath metabolic measurement, SensorMedics MSE, Yorba Linda, CA). The pneumotachograph was calibrated with a 3-L calibration syringe (Model 5530 series, Hans Rudolph, Kansas City, MO), and the gas analyzers were calibrated with reference gases. The breath-by-breath data were averaged over 20-s periods.

VT₁ and VT₂ were determined graphically, using all the VO₂, VCO₂, and VE curves (Fig. 1B). For VT₁, the determination was confirmed by the Beaver et al. method (1) based on a computerized regression analysis of the slopes of the VCO₂ versus VO₂. VT₂ was determined by the breakpoint of the VE/VCO₂ slope (32), where the increase in VE overwhelms the CO₂ output because of the ventilatory drive due to excess acidosis (Fig. 1B).

Metabolic Parameters

Blood lactate. Lactate was measured on 2-mL samples of whole arterial blood by the lactate oxidase reaction (Chiron-Diagnostics S 800, Bayer, Puteaux, France) with reference to a calibration curve.

Cardiovascular Parameters

Cardiac output. Cardiac output (Q) determined with the direct Fick method, was obtained as:

\[
Q(\text{L·min}^{-1}) = \frac{\text{VO}_2\left(\text{mL·min}^{-1}\ STPD\right)}{\left[\text{CaO}_2\left(\text{mL·L}^{-1}\right) - \text{CO}_2\left(\text{mL·L}^{-1}\right)\right]},
\]

where STPD stands for standard temperature and pressure dry and VO₂ for measured O₂ consumption. CaO₂ is the systemic arterial blood O₂ content and VO₂ the mixed venous blood O₂ content. Blood gas analyses (pH, PaCO₂, PaO₂) were performed with a Ciba-Corning 270–278 CO-oxymeter (Bayer), which permits measuring (and not calculating) hemoglobin saturation. A manual calibration using medical gases was performed in the morning. Thereafter, two commercially available ampuled quality control solutions (one with pH 7.382, PaCO₂ 35 mm Hg, PaO₂ 85 mm Hg, and the other with pH 6.838, PaCO₂ 70 mm Hg and PaO₂ 0 mm Hg) were used to control the gaseous calibration. This allowed plotting a reference slope for the subsequent measurements. An automatic calibration was made every 30 min during the day using the first solution. In addition, a two-point calibration was automatically performed every 2 h. The blood gases were not the subjects’ temperature corrected.

During the IET, Q was measured every 2 min from rest to peak effort. During the IWET, Q was measured at rest, at the end of the warm-up, at the last 20 s of the first and the third minutes of the “bases” (charge corresponding to VT₁). VO₂ was measured, and arterial and mixed venous blood was sampled during these 20-s periods.

Heart rate (HR) was monitored continuously with a 12-derivation electrocardiograph (Cardiovit CS-200, Schiller AG, Baar, Switzerland). Stroke volume (SV) was calculated as the ratio of Q to HR.

Vascular pressures. PAP and SAP were recorded by means of the fluid-filled catheters. Measurements were made regularly from rest to peak effort during the IET, every 2 min, alternatively with the sampling of mixed venous and arterial blood. During the IWET, measurements...
were obtained at rest, during the warm-up, and during the last 20 s of the second and fourth minutes of the “bases” and during the “peak” (charge corresponding to \( VT_2 \); Fig. 1C).

**Vascular resistances.** Systemic total vascular resistance (STVR) and pulmonary total vascular resistance (PTVR) were calculated as the ratio of mean pressure to \( Q \).

**Statistical Methods**

Data are reported as means ± SD. Individual values are also given. Statistical analysis was performed using SIGMASTAT statistical software for Windows (version 2.0.3). Analysis of variance for repeated measurements was performed on all variables registered during the IWET. When significant differences were found, Student-Newman-Keul’s method further evaluated these differences. Student’s \( t \)-test was used for maximal paired data to assess differences between the IET and the IWET. The level of significance was set at \( P < 0.05 \).

**RESULTS**

The anthropometric data for the subjects are presented in Table 1.

**IET**

**Cardiac and respiratory parameters.** All subjects satisfied the maximal incremental exercise test by fulfilling at least three of the following four criteria: a VO\(_2\) increase of less than 150 mL·min\(^{-1}\) compared with the previous step, a high level of lactate during the early recovery period, a respiratory exchange ratio (RER) > 1.1, and the attainment of at least 90% of the theoretical maximal HR. Individual rest and maximal exercise parameters are reported in Table 2.

\( VT_1 \) and \( VT_2 \) determined during the IET (Table 3) correspond respectively to 62.2 ± 10.4% and 90.4 ± 21.2% of VO\(_{2\max}\).

**Pulmonary and systemic arterial pressure parameters.** PAP could be measured in only six subjects. In the remainder, the catheter did not progress further than the right ventricle, and for safety of the investigation, the catheter was removed and maintained into the right atrium. No pressure of interest could be measured in this patient, but by maintaining the catheter in the right atrium, mixed venous blood could be sampled, allowing the calculation of \( Q \). The mixed venous blood saturation and oxygen pressure of this subject were not different from those measured for the six other subjects, where gas sampling was performed in the pulmonary artery. At rest, PAP was 13.5 ± 2.0 (range 11–17) mm Hg and reached 26.5 ± 5.6 (range 20–35) mm Hg at peak exercise (Table 2). SAP increased from 100.8 ± 6.4 mm Hg at rest to 133.8 ± 10.2 mm Hg at peak exercise (Table 3).

**IWET**

**Ventilatory and metabolic parameter.** VO\(_2\) increased during the first stage of the IWET, with a first peak value at the sixth minute of exercise (\( P < 0.05 \)). After this initial increase, VO\(_2\) rose during the peak cycling and decreased during the base cycling, but with no significant change until the end of the 30-min exercise. The maximal VO\(_2\) reached during the IWET (21st min) did not significantly differ from the VO\(_{2\max}\) of the IET (2871.0 ± 596.0 mL·min\(^{-1}\)) or 38.0 ± 6.9 mL·min\(^{-1}\)·kg\(^{-1}\) vs 3081.3 ± 507.8 mL·min\(^{-1}\) or 40.5 ± 6.9 mL·min\(^{-1}\)·kg\(^{-1}\), respectively; Fig. 2A). VE increased from rest to a maximal value at the sixth minute of exercise and then remained stable until the end of the IWET. The maximal value of VE measured during the IWET was significantly lower than the maximal VE of the IET (83.2 ± 24.6 vs 107 ± 31.7 L·min\(^{-1}\), respectively; \( P < 0.05 \); Fig. 2B). VCO\(_2\) paralleled VE during the 30-min IWET (not shown).

Lactate increased significantly during the IWET until the sixth minute (6.6 ± 1.0 mmol·L\(^{-1}\)) and then oscillated around this value until the end of the 30-min exercise (Fig. 2B). Maximal lactate (21st min) tended to be lower during the IWET than during the IET (7.9 ± 1.9 vs 9.6 ± 2.4 mmol·L\(^{-1}\), respectively; Fig. 2B), but the difference did not reach the level of significance.

**Hydration parameter.** Variation of PV calculated with hematocrit and hemoglobin did not exceed 5.4 ± 7.3% at rest before the IET and IWET. BV and PV were, respectively, 5.5 ± 3.7% and 11.5 ± 8.4% lower at the end of the IWET than at rest before the exercise.

**Hemodynamic parameters.** \( Q \) increased during the first 5-min stage of the exercise, reached a first peak value at the sixth minute after the warm-up, and remained stable until the end of exercise (Fig. 3A). The maximal value measured during the IWET (21st minute) did not significantly differ from the maximal value observed during the IET (19.6 ± 5.6 vs 20.0 ± 3.3 L·min\(^{-1}\), respectively; Fig. 3A). During the IWET, HR increased progressively and significantly from the beginning to the end of the exercise (\( P < 0.01 \); Fig. 3B). SV increased during the first 6 min, then decreased until the end of the IWET (\( P < 0.05 \); Fig. 3B).

During the IWET, PAP increased immediately and values were maximal during the first 5-min stage for all subjects. No difference was found between maximal PAP during IET and IWET (26.5 ± 5.6 vs 24.0 ± 5.0 mm Hg, respectively; Table 3; Fig. 4A). After the initial increase, PAP decreased significantly throughout the IWET (\( P < 0.001 \); Fig. 4A).

After the warm-up, SAP increased to a maximal value at the fifth minute of the IWET and decreased thereafter significantly (127.2 ± 10.7 vs 111.7 ± 5.5 mm Hg, \( P < 0.001 \); Table 2. **TABLE 1. Anthropometric data of the subjects.**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>BMI (kg·m(^{-2}))</th>
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<td>178</td>
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<tr>
<td>7</td>
<td>32</td>
<td>55</td>
<td>182</td>
<td>25.7</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>38 ± 5</td>
<td>77 ± 7</td>
<td>178 ± 3</td>
<td>24.3 ± 2.0</td>
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</table>

BMI, body mass index.
Fig. 4A). PTVR and STVR decreased respectively from 183 ± 31 to 91 ± 14 dyn·cm⁻²·s⁻¹ (P < 0.001) and from 1319 ± 417 to 557 ± 176 dyn·cm⁻²·s⁻¹ (P < 0.001) from rest to the end of exercise with the main decrease being observed during the first minutes of the exercise (Fig. 4B).

**DISCUSSION**

The main result of the present study is that our intermittent exercise in healthy young subjects induces no dramatic increase in PAP during the first minutes of exercise nor does it prevent the decrease of PAP with the prolongation of exercise, although higher work rates (peaks) could have pushed the pulmonary pressures higher. Moreover, this result accounts for a high-intensity exercise because HR was near maximal at the end of the 30-min exercise.

The intermittent exercise at VT₁ and VT₂. Training close to VO₂max has proven its efficacy in athletes (2). Nevertheless, in elite runners, Billat et al. (3) showed that with a repetition of 30 s of running at a velocity correspond-

TABLE 2. Individual values for the different parameters measured at rest and at MTP of IET.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Power (W)</th>
<th>VO₂max (mL·min⁻¹·kg⁻¹)</th>
<th>Lactate (mmol·L⁻¹)</th>
<th>SaO₂ (%)</th>
<th>HR (% pred)</th>
<th>SV (mL)</th>
<th>Q˙ (L·min⁻¹)</th>
<th>PAP (mm Hg)</th>
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<tr>
<td>1</td>
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<td>440</td>
<td>1.06</td>
<td>98</td>
<td>40</td>
<td>102</td>
<td>7.3</td>
<td>17</td>
</tr>
<tr>
<td>Rest</td>
<td>320</td>
<td>358</td>
<td>12.76</td>
<td>97</td>
<td>100</td>
<td>133</td>
<td>24.0</td>
<td>56</td>
</tr>
<tr>
<td>MTP</td>
<td>360</td>
<td>395</td>
<td>29</td>
<td></td>
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</tr>
<tr>
<td>2</td>
<td>—</td>
<td>314</td>
<td>0.56</td>
<td>98</td>
<td>49</td>
<td>72</td>
<td>6.5</td>
<td>14</td>
</tr>
<tr>
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<td>3103</td>
<td>8.49</td>
<td>99</td>
<td>99</td>
<td>128</td>
<td>23.0</td>
<td>28</td>
</tr>
<tr>
<td>MTP</td>
<td>325</td>
<td>3578</td>
<td>7.55</td>
<td>97</td>
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<td>17.9</td>
<td>28</td>
</tr>
<tr>
<td>MTP</td>
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<td>1.09</td>
<td>98</td>
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<td>48</td>
<td>3.7</td>
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<td>Rest</td>
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<td>2672</td>
<td>11.17</td>
<td>98</td>
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<tr>
<td>Rest</td>
<td>270</td>
<td>2890</td>
<td>7.23</td>
<td>97</td>
<td>95</td>
<td>115</td>
<td>20.6</td>
<td>—</td>
</tr>
<tr>
<td>MTP</td>
<td>276 ± 50</td>
<td>3081 ± 508</td>
<td>9.6 ± 2.4</td>
<td>97 ± 0.5</td>
<td>43 ± 5.0</td>
<td>69 ± 20</td>
<td>5.4 ± 1.5</td>
<td>13.5 ± 2.0</td>
</tr>
</tbody>
</table>

Predicted HR, 220 — age (1); VO₂, O₂ uptake; SaO₂, arterial hemoglobin saturation; SV, stroke volume; Q, cardiac output; PAP, mean pulmonary arterial pressure; IET, incremental exercise test; MTP, maximal tolerated power.
recovery periods were of lower intensity. In the present study, the 30-min intermittent exercise allowed, at the end of exercise, the subjects to reach their maximal HR and was well tolerated by all of them. There was no large increase in lactate, which remained below 8 mmol·L$^{-1}$ during the 30-min exercise. Q$\dot{\mathbf{v}}$ and VO$_2$ rose during the “peaks” and decreased during the “bases” but remained stable as a mean, from the sixth minute to the end of the IWET. This reflects a constant mean metabolic demand, throughout the 30-min exercise, despite the performance of 1-min bouts of heavy exercise.

**Q adjustments.** The constant Q observed in our study is in accordance with previous studies for constant load exercises (6,9,14), although an increase of Q over time has also been observed (29). This discrepancy might be explained by the differences in work intensities among the studies. In the present work, a stable Q resulted from an increase in HR and a decrease in SV.

One of the factors leading to a decreased SV is peripheral vasodilation and the shift of BV from central to peripheral circulation. It has been suggested that skin vasodilation and increased skin blood flow result from an increase in body temperature (28). Vasodilation in skin and working muscles has been reported to cause a decrease in SAP (7), as was clearly seen in our study.

A decrease in PV is another factor that could explain the SV decrease, as reported by Hamilton et al. (15), for a continuous 120-min exercise, although these authors allowed their subjects to drink freely the night before and up to 1 h before testing. This was not the case in our study and might explain our more marked decrease in PV (11.5% in the 30-min exercise in our study vs 6.7% in the 20-min exercise in Hamilton et al.’s study (15)).

From the above-mentioned studies, it can be suggested that skin vasodilation with a shift of BV from central to
peripheral circulation and dehydration with decreased PV lead to a decreased venous return with a subsequent decrease in ventricular filling and in SV. This gives in turn an increased HR with decreased ventricular filling time (31) and a decrease of end-diastolic heart volume (13,31), which explains also a decreased SV.

Left ventricular dysfunction is an other factor that can be considered in the SV decrease, but the results of the different studies (22,35) devoted to this aspect suggest to us that left ventricular dysfunction in healthy subjects probably appears only for exercise durations that exceed 90 min, which was not the case in our study.

The increase in HR observed from the beginning to the end of the IWET is a general phenomenon of cardiovascular adjustment in prolonged exercise. In addition to thermoregulatory and hydration responses, the HR increase in prolonged exercise is probably also due to increased circulating catecholamines (21,27). With our intermittent exercise, HR reaches the maximal value observed during the IET at the 30th minute. This was generally not the case in previous studies assessing prolonged exercise, either because the intensity was rather low (6,13,24) or because the exercise consisted of alternating heavy work with rest (20) or was continuous. In contrast to these studies, HR was maximal at the end of the IWET in our study, and we think that this type of interval exercise produces near maximal stimulation in healthy subjects.

**Pulmonary hemodynamics.** The main result of the present study is that PAP, after its initial increase, does not increase further during our IWET, although high-intensity exercise bouts could have led to pushing the pulmonary pressures higher. Our present work is a part of a larger study of estimating peripheral and central hemodynamics, with special attention to the pulmonary circulation during IWET in healthy subjects but also in COPD patients. It is well known that the PAP level is a prognostic factor for survival in COPD patients (34) and that exercise may worsen their hypoxia and increase PAP to an abnormally high level (16). Whether short repeated bouts of high-intensity work rate may lead to a dramatic increase in PAP in such patients is still in question. Indeed, in COPD patients, PAP may increase during exercise more than two- or threefold the normal increase of 1 mm Hg·L⁻¹·min⁻¹ increase of Q (23). Moreover, in the long term, pulmonary hypertension may lead to right heart failure in some patients (33). Whether long-term training with our IWET may lead to worsening pulmonary hypertension is another still unanswered question. But before trying to get an answer, it is important to know what can be considered as a normal response during an IWET.

To our knowledge, this study is the first that reports the behavior of the pulmonary circulation during intermittent prolonged exercise. In previous studies, this aspect was investigated either during incremental exercise (25,27) or during continuous prolonged exercise at varying intensities (9,29,30), most of them having been performed during constant light- to moderate-intensity exercise (9,30). Like these previous studies (9,29,30) devoted to the behavior of PAP during continuous prolonged exercise, we observed an initial increase of PAP, which did not attain the maximal value observed at the end of the IET, followed by a significant decrease despite 1-min heavy-exercise bouts. Such behavior is in part explained by the fact that during exercise, there is an opening of resting unperfused vessels. The 12% increase in diffusion lung capacity of carbon monoxide in the study
of Ekelund (9) between the seventh and 30th minutes of work is in accordance with such a pulmonary vascular recruitment, which can also explain, in part, the progressive decrease in PTVR during the IWET in our study. We observed an increase of PAP of less than 1 mm Hg for a 1 L-min\(^{-1}\) increase in Q during the IET. Similar results for the PAP/Q slope have been reported by Palevsky (25) during an incremental exercise test. Moreover, in our study, the slope of the ratio of PAP to Q decreased from the beginning to the end of the IWET, which underlines changes in the pulmonary vascular bed during prolonged intermittent exercise. Whether vascular recruitment alone or additional changes in vasomotor tone at the pulmonary vascular bed level lead to the decrease in PTVR cannot be inferred from our study. Local metabolic, myogenic, or hormonal factors may affect the pulmonary vasomotor tone. Atrial natriuretic peptide (ANP) and brain natriuretic peptide have been studied during prolonged exercise (21,22,26). The increase in ANP is probably due to atrial distension, especially during the first minutes of exercise (21), but also to catecholamines and body temperature (21). Whether increased ANP has a direct vasodilative effect on pulmonary vasomotor tone or acts as an inhibitor of the vasoconstrictive effect due to other factors like endothelin-1 (ET-1) still remains in question. The role of ET-1 during exercise in healthy subjects is probably of low importance, but in patients, this hormone may have a substantial vasoconstrictive effect (36). Further studies have to be performed to clarify the effect of the different factors involved in the pulmonary vascular adaptation during exercise.

**Limitations of the study.** Q was measured at the first minute after the “peak,” but \(\dot{V}O_2\) and the arteriovenous \(O_2\) difference at this time probably reflect what happened during the 1-min peak exercise.

**REFERENCES**


The study would have been improved if the same subjects had also performed continuous exercise. Because of the invasive nature of the investigation, this would have made this ethically difficult. It still remains in question whether during a continuous exercise PAP would have decreased more than during an IWET performed with bouts of very high work rates.

Another limitation is that our subjects were younger than the general population of COPD patients. This was also the case for the earlier invasive studies, which were performed in young healthy subjects. We nevertheless think that in healthy older subjects, there probably would be no dramatic increase in PAP during an IWET, although the decrease of PAP might be less than in younger subjects.

**CONCLUSION**

In conclusion, during an IWET with near maximal stimulation (HR near maximal at the end of the 30-min exercise), we observed an initial increase in Q, which remained stable thereafter, with an increasing HR and a decreasing SV. PAP rose during the first minutes of the IWET then decreased significantly to the end of exercise. To our knowledge, our study is the first devoted to the behavior of the pulmonary circulation during prolonged intermittent exercise with bouts of near maximal work rate. We can conclude that such high-intensity exercise bouts do not lead, in healthy subjects, to push the PAP high. Whether the same behavior can be seen in COPD patients (decreasing PAP during a prolonged IWET) who are often chronically hypoxic with resting or exercising pulmonary hypertension needs further evaluation.


