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Is Exercise-Induced Arterial Hypoxemia in Triathletes Dependent on Exercise Modality?

Abstract

To determine whether exercise modality affects arterial hypoxemia (EIAH) during training-intensity exercise, 13 triathletes performed 20 min of cycling (C) followed by 20 min of running (R); C-R, and two weeks later, 20 min of R followed by 20 min of C: R-C. Each trial was performed at an intensity slightly above the ventilatory threshold and close to the daily training intensity (75% of V̇O₂max). Ventilatory data were collected continuously using an automated breath-by-breath system. Partial pressure of oxygen in arterial blood (PaO₂) was measured after each C and R segment and arterial oxyhemoglobin saturation (SpO₂) was monitored continuously via pulse oximetry. The metabolic rate was similar across modalities and trials, i.e., C-R (53.8 ± 3.8 vs. 51.1 ± 5.3 ml·min⁻¹·kg⁻¹) and R-C (52.2 ± 4.5 vs. 53.2 ± 4.6 ml·min⁻¹·kg⁻¹). EIAH showed significantly greater severity for R compared to C irrespective of the order (p < 0.05 for both trials). R values of PaO₂ (and SpO₂) for C-R and R-C were 88.7 ± 6.0 mmHg (93.0 ± 0.6% SpO₂) and 86.6 ± 7.3 mmHg (93.5 ± 0.6% SpO₂) and C values were 93.7 ± 8.4 mmHg (95.4 ± 0.4% SpO₂) and 91.4 ± 5.4 mmHg (94.8 ± 0.3% SpO₂). R ventilatory data described a significantly different breathing pattern than C, with higher respiratory rate (35.9 b·min⁻¹ vs. 31.1 b·min⁻¹ for C-R, p < 0.01; and 50.0 b·min⁻¹ vs. 41.5 b·min⁻¹ for R-C, p < 0.01) and lower tidal volume (2636 ml vs. 2282 ml for C-R, p < 0.02 and 2272 ml vs. 2472 ml for R-C, p < 0.05). We concluded that EIAH was greater during running than cycling for a similar metabolic rate corresponding to training intensity and that EIAH could thus be considered dependent on exercise modality.

Key words
Pulmonary gas exchanges · EIAH · DLCO · endurance training · breathing pattern · triathlon

Introduction

Exercise-induced arterial hypoxemia (EIAH) is frequent in highly trained male endurance athletes with a maximal oxygen uptake (V̇O₂max) greater than 55 ml·min⁻¹·kg⁻¹ [5,24]. Different endurance activities are well known to generate EIAH, particularly cycling, running, swimming, rowing, and the triathlon [3,11,24, 29,32]. Recent studies [10,30] have shown a greater drop in the partial pressure of oxygen in arterial blood (PaO₂) or in arterial oxyhemoglobin saturation (SpO₂) at both maximal [10] and submaximal exercise [30] during treadmill running compared with ergometer cycling, which suggests that EIAH is dependent on exercise modality. However, a concomitant higher V̇O₂max for treadmill running than for ergometer cycling has also been observed, with V̇O₂ values inversely related to SpO₂ level during maximal and submaximal exercise [28,34]. Unfortunately, these latter studies did not clearly indicate whether the difference in SpO₂ between the two activities was associated with the difference in
VO₂ or with the activity per se, thereby leaving the question of EIAH’s dependency on exercise modality open.

EIAH is a multifactorial phenomenon [6,27] and in both cycling and running, it is usually attributed to two main mechanisms: a relative hyperventilation [5] and/or an alteration in pulmonary gas exchange, i.e. ventilation/perfusion mismatching and/or diffusion limitation [4]. Relative hyperventilation has been suggested to be the predominant factor at submaximal exercise intensity and may be present in all highly trained athletes [6,20]. In addition, the higher ventilatory components observed in triathletes during running compared to cycling have systemically been related to the higher EIAH of running [8,9,18].

This study aimed (1) to determine whether EIAH is dependent on exercise modality and (2) to evaluate the role of the two principal mechanisms, particularly relative hyperventilation, in regard to modality. Triathletes, who are skilled at both cycling and running, performed a constant submaximal cycle-run succession, as well as an inverse run-cycle succession. Both trials were conducted at the same metabolic rate, which was close to the athletes’ daily training intensity. We hypothesised that running would induce greater EIAH than cycling, whatever the modality order, as previously reported for maximal exercise.

**Methods**

**Subjects**

Thirteen male competitive triathletes participated in this study. All were students at the School of Physical Education of the University of Montpellier, France, as well as members of the University athletic team, which has been French national champion in the triathlon for five consecutive years. Some were on the national federation team. All had been competing in the triathlon for 6.3 ± 2.8 years and were in the competitive period at the time of the study. They were 22.3 ± 3.2 years old, weighed 67.5 ± 6.3 kg and measured 176.3 ± 6.8 cm in height. All were non-smokers with normal lung function and no prior history of pulmonary or cardiovascular disease. These triathletes were familiar with both the treadmill and cycle ergometer. They gave written consent to participate in this study after the purpose, design, and risks had been described to them. The study protocol was in accordance with legal requirements and the Declaration of Helsinki, and it was approved by the regional ethics committee.

**Testing protocol**

An incremental cycle test was first performed on an electromagnetical cycle ergometer (Monark 864, Monark-Crescent AB, Värburg, Sweden) to assess maximal oxygen uptake (VO₂max) and ventilatory threshold (VT). Three to seven days later, the triathletes performed two constant submaximal exercises consisting of a cycle-run (C-R) succession and an inverse run-cycle (RC) succession of 40 min each; these successions were performed within a two-week period. The incremental test and the trials were conducted in an air-conditioned laboratory with a mean room temperature of 21.9 ± 0.2 °C and a barometric pressure of 777.5 ± 4.5 mmHg. Trials were randomised and performed at the same time of day and on the same day of the week to minimise the effects of circadian rhythms and personal training. The triathletes were asked to maintain their training schedule for the duration of the study but they were not allowed to compete in a triathlon during the testing period. The triathletes were also asked to refrain from training on experimental days. The protocol of the incremental cycle test started with a 3-min warm-up at 30 W. The power was then increased by 30 W every minute until each subject reached volitional fatigue. All triathletes reached their maximal oxygen uptake with: (1) a levelling-off of VO₂ despite increasing load, (2) a respiratory exchange ratio (RER) greater than 1.10, (3) attainment of age-predicted maximal heart rate (HR) ± 5% [210 – [0.65 × age]], and (4) the inability to maintain pedalling frequency despite maximum effort and verbal encouragement. VT was determined using the V-slope method of Beaver et al. [1], which analyses carbon dioxide production (VCO₂) as a function of VO₂ and assumes that VT corresponds to the breakpoint in the VCO₂-VO₂ relationship. Because our group has demonstrated that incremental cycling and running VO₂max are similar in young triathletes without prior athletic specialisation [19] – which was the case of the triathletes of the present study – the cycling VO₂max value was used to express the metabolic rate of both the cycling and running segments of the two trials, i.e. about 75% of VO₂max is an intensity that is higher than VT. During C-R and R-C, the R and C segments were managed in order to maintain this level for the duration of the trials. The triathletes used their own cycles on a home trainer (Pro Training, Pro Trainer, Milan, Italy), and each cycle was equipped with a profiled handlebar commonly used during the cycle segment of a triathlon. The VO₂ level was progressively reached by the end of the third minute and then maintained by increasing or decreasing the cycling speed by 1 km·h⁻¹ every minute. Distances were recorded with a bike odometer (Top Bike, Tokyo, Japan). After the cycle segment, the triathletes had 1 min 30 s to change their shoes and get on the treadmill. During running (Gymroll 1800, Gymroll, Roche la Molière, France), the VO₂ level again rose progressively and was attained by the end of the third minute. It was then maintained by increasing or decreasing the running speed by 0.5 km·h⁻¹ every minute. Distances were recorded with the treadmill odometer. After the running segment of R-C, the triathletes had 1 min 30 s to change their shoes and get on their bicycle.

**Complementary experiment**

Two complementary randomised trials were performed by three volunteer triathletes. The first consisted of 10 min of cycling with a spontaneous breathing pattern (C₁), followed by 10 min of cycling with an imposed breathing pattern corresponding to that of running (CR) during C-R and R-C, then 5 min of cycling with a spontaneous breathing pattern (C₂). The second trial consisted of 10 min of running with a spontaneous breathing pattern (R₁), followed by 10 min of running with an imposed breathing pattern similar to that of cycling (RC) during C-R and R-C, then 5 min of running with a spontaneous breathing pattern (R₂). During C and R, RR was imposed on the triathletes using an electronic metronome as auditory feedback. In contrast to RR, VT was not monitored. These two trials were performed at the same metabolic rate and in the same general and ambient conditions as C-R and R-C.
Percent SpO₂
Arterial oxyhemoglobin saturation (SpO₂) was assessed continuously and recorded every minute during the incremental cycle test and the C-R and R-C successions, using an ear pulse oximeter (Ohmeda 3740 Pulse Oximeter, Louisville, USA). This method has been reported to deliver high precision, reproducibility, and validity for O₂ saturation above 85% when compared to SaO₂ measured from arterial blood gases in highly trained athletes [21], with 0.52 ± 1.36% (standard deviation) of the difference in mean % SaO₂ - % HbO₂ and a standard error of the estimate = 1.32% at submaximal exercise. Moreover, the Ohmeda 3740 Pulse Oximeter is highly accurate when placed on the ear lobe at maximal exercise, as well as during submaximal exercise [21]. The instrument’s conducting cable was secured to prevent movement artefacts, especially during running, and the oximeter was calibrated before each experiment using an internal calibration. Before the probes were placed, the sites were vigorously cleaned with alcohol and gauze pads. The oximeter’s poor signal alarm and pulse waveform were closely monitored by the recording investigator to ensure that spurious data were not included. Athletes were considered to have developed EIAH when SpO₂ decreased by at least 4% from baseline value [27].

Blood gas measurements
In order to confirm the SpO₂ data and to calculate alveolar ventilation, arterialised blood samples were obtained from the ear lobe at rest and at the 20th and 40th minutes of C-R and R-C. Before sampling, the ear lobe was warmed up by massage to activate arterialised circulation. Arterialised blood samples are known to give reliable values, as was demonstrated in recent works of our group [8,18]. These samples were immediately analysed for PaO₂, the partial arterial pressure of carbon dioxide (PaCO₂), and pH at 37 °C using an appropriate electrode (Il Meter 1306, Milan, Italy). The instrument was calibrated prior to each measurement with the same gas mixture as for the expired gas analysers. Given the variability in PaO₂ measurement, resting hypoxemia should be defined as a drop of 5 mmHg in PaO₂ [25]. During exercise, the decrease in PaO₂ may be overestimated because of a rise in body temperature. Our arterialised blood gas values are presented for a temperature of 37 °C because we assumed that the 40-min exercise duration would not raise rectal temperature since (1) the laboratory was air-conditioned (21.9°C) and (2) when exercise in triathletes was performed at 70% of VO₂max with an ambient temperature of 29°C, the increase in rectal temperature was 0.2 and 0.5°C after 20 and 40 min of cycling and 0.5 and 1.0°C after 20 and 40 min of running [20]. Therefore, to minimise the risk of overestimating EIAH due to an increase in body temperature estimated to be about 0.5°C, we added 3 mmHg to the 5 mmHg drop in PaO₂ commonly used to define hypoxemia during exercise [25], i.e., a drop of 8 mmHg in PaO₂. Lastly, to account for the error caused by the lag between rectal temperature and blood temperature, i.e., 2 mmHg [15], we defined EIAH as a minimum drop of 10 mmHg in PaO₂. Arterialised blood gases were drawn and carbon dioxide production (VCO₂) was computed during the last 20 s of treadmill running and ergometer cycling during the trials. This allowed us to calculate alveolar ventilation (VA) using the Bohr equation, where \( VA_{\text{STPD}} = 863 \cdot VCO₂_{\text{STPD}} / PaCO₂ \). In this equation, VCO₂ was calculated with the mean values at the 19th minute of each segment, i.e., C and R.

Pulmonary diffusing capacity
All subjects rested in the seated position for 10 min before measurement of pulmonary diffusing capacity for carbon monoxide (DLco). The DLco was measured during breath-holding (single breath method) while seated, using an automated transfer apparatus before and 10 min after C-R and R-C (Transfer Test, Morgan, Chatham, Kent, UK). After a full exhalation, subjects inhaled a gas mixture (10% He, 21% O₂, and 2.800 ppm CO in N₂), held their breath for 9 s, and finally executed a rapid exhalation (American Thoracic Society 1987). DLco measurements were made by the same operator in triplicate.

Gas exchange measurements
During incremental cycle testing, as well as the C-R and R-C successions, ventilatory data were computed every minute using a mass spectrometer breath-by-breath automated system (MGA 1100, Marquette, NY, USA): minute ventilation (VE), oxygen uptake (V̇O₂), V̇CO₂, respiratory exchange ratio (RER), respiratory equivalents for O₂ (V̇E/V̇O₂) and CO₂ (V̇E/V̇CO₂), respiratory rate (RR), and tidal volume (VT). The V̇D/V̇T ratio was also calculated (from the equation V̇D/V̇T = V̇A/(V̇E/R)), and the analyses were calibrated before each trial with standard O₂ and CO₂ gases of known concentration using a certified commercial gas preparation. Heart rate (HR) was recorded using a telemetry system (Polar Sport Tester, Polar Electro Oy, Kempele, Finland).

Statistical analysis
The results are expressed as means ± SD. Running and cycling distances during C-R and R-C were compared using a paired Student t-test. After verification of a normal distribution (Gaussian graphical distribution), SpO₂, PaO₂, PAO₂, PaCO₂, VA, (A-a)DO₂, pH, and the cardioventilatory data (V̇O₂, VE, RER, VE/V̇O₂, VE/V̇CO₂, VT, V̇D/V̇T, RR, and HR), which had been recorded throughout the two trials and complementary trials, were compared using a two-way analysis (exercise-time) of variance (ANOVA) with repeated measures. When significant results were obtained with ANOVA, post-hoc comparisons were made using Scheffe’s post-hoc test [35]. DLco measurements were compared after versus before the two trials to analyse the influence of specific exercise on DLco using a one-way ANOVA. Statistical analysis was performed using a statistical software package (SYSTAT). Statistical significance was accepted at the p < 0.05 level.

Results
Exercise intensities
The incremental exercise test revealed a V̇O₂max of 69.9 ± 3.3 ml·min⁻¹·kg⁻¹ for the triathletes. C-R and R-C showed no significant difference in running and cycling distances: 12.6 ± 0.9 km versus 11.9 ± 1.9 km for cycling and 6.5 ± 0.2 km versus 6.5 ± 0.2 km for running, respectively (Tables 1 and 2). Metabolic rate (and % of V̇O₂max) during C-R and R-C were similar, with 53.8 ± 3.8 ml·min⁻¹·kg⁻¹ (77 ± 5.4% of V̇O₂max), 51.1 ± 5.3 ml·min⁻¹·kg⁻¹ (73 ± 5.6% of V̇O₂max), 52.2 ± 4.5 ml·min⁻¹·kg⁻¹ (74.6 ± 6.4% of V̇O₂max), and 53.2 ± 4.6 ml·min⁻¹·kg⁻¹ (76.1 ± 6.1% of V̇O₂max), respectively (Table 1).
Table 1 SpO₂ and cardioventilatory parameters measured during C-R and R-C

<table>
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<tr>
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<th>C</th>
<th>R</th>
<th>R</th>
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<tbody>
<tr>
<td>SpO₂ (%)</td>
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<tr>
<td>VO₂ (ml-min⁻¹-kg⁻¹)</td>
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<td>51.1</td>
<td>52.2</td>
<td>53.2</td>
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<tr>
<td>% VO₂max Cycling</td>
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<td>73.0</td>
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<td>76.1</td>
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<td>VE (l-min⁻¹)</td>
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<td>113.5</td>
<td>106.9</td>
<td>100.8</td>
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<tr>
<td>VE/VO₂</td>
<td>21.7</td>
<td>28.6</td>
<td>26.1</td>
<td>24.0</td>
</tr>
<tr>
<td>VE/VCO₂</td>
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<td>30.2</td>
<td>27.3</td>
<td>25.0</td>
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<tr>
<td>RER</td>
<td>0.94</td>
<td>0.94</td>
<td>0.96</td>
<td>0.96</td>
</tr>
<tr>
<td>RR (breath-min⁻¹)</td>
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<td>51.1</td>
<td>50.0</td>
<td>41.5</td>
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<tr>
<td>VT (ml)</td>
<td>2636</td>
<td>2282</td>
<td>2272</td>
<td>2472</td>
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<tr>
<td>HR (b-min⁻¹)</td>
<td>154</td>
<td>171</td>
<td>166</td>
<td>167</td>
</tr>
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</table>

Values are mean ± SD. * p < 0.05 significantly higher compared to C inside trials. ** p < 0.05 significantly lower compared to C within C-R and R-C.

Percent SpO₂

During incremental exercise, SpO₂ decreased from 99 ± 0.2% (resting value) to 93.4 ± 1.8% (range 90–95%), thereby demonstrating EIAH. During C-R and R-C, all triathletes presented a significant drop in SpO₂ compared with the resting values (Fig. 1). The mean values presented in Table 1 show a significant drop from rest values throughout the entire C-R and R-C trials, with a p < 0.05. Moreover, this drop was significantly more pronounced during R when compared to C within each trial. The amplitude of the drop was – 6 ± 2.2% and – 5.8 ± 2.0% during running (p < 0.01) and – 3.9 ± 1.1% and – 4.1 ± 1.0% during cycling of C-R and R-C, respectively (Fig. 1).

Blood gas measurements

A significant drop from resting values in PaO₂ and PaCO₂ was noted after C-R and R-C (Table 2). The drop in PaO₂ was significantly greater during running (– 15.1 ± 3.2 mmHg and – 20.3 ± 11.0 mmHg) than during cycling (– 10.2 ± 2.2 mmHg and – 16.0 ± 11.2 mmHg) in the C-R and R-C trials, respectively. Moreover, the significantly greater drop in PaO₂ noted during running was concomitant with significantly lower VA values (Table 2).

PaO₂, and (A-a)DO₂ increased during C-R when compared to rest values, whereas only (A-a)DO₂ increased during R-C (Table 2). Moreover, a significant difference was observed between C and R of C-R, with a higher (A-a)DO₂ during successive R. The pH values only decreased during C of C-R, showing a significant drop compared to both rest and successive R.

Respiratory parameters

The two trials were characterised by higher VE, VE/VO₂, VE/VCO₂, VD/VT, and RR and lower VT during running compared with cycling (Tables 1 and 2). RER showed no significant difference throughout the trials. HR increased significantly during successive R of C-R.

Dlco measurements

Dlco measured 10 min after the end of C-R and R-C decreased significantly when compared to rest values, p < 0.01, Table 2.

Table 2 Arterialised blood gas components (PaO₂, PaCO₂, PAO₂, [A-a]DO₂, and pH), alveolar ventilation (VA) and VD/VT at rest and during C-R and R-C. Dlco measured at rest and 10 min after each trial

<table>
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<tr>
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<tr>
<td>PaCO₂ (mmHg)</td>
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<td>36.7</td>
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<tr>
<td>VA (l-min⁻¹)</td>
<td>13.3</td>
<td>95.2</td>
<td>84.1</td>
<td>#</td>
<td>11.6</td>
<td>71.9</td>
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<tr>
<td>VD/VT (%)</td>
<td>37.6</td>
<td>16.2</td>
<td>32.3</td>
<td>#</td>
<td>38.2</td>
<td>36.9</td>
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<tr>
<td>PaO₂ (mmHg)</td>
<td>115.8</td>
<td>119.4</td>
<td>121.2</td>
<td>116.8</td>
<td>117.1</td>
<td>118.2</td>
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<tr>
<td>(A-a)DO₂ (mmHg)</td>
<td>11.4</td>
<td>24.6</td>
<td>33.2</td>
<td>#</td>
<td>9.2</td>
<td>30.5</td>
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<tr>
<td>pH</td>
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<td>7.38</td>
<td>7.41</td>
<td>7.43</td>
<td>7.44</td>
<td>7.42</td>
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<tr>
<td>Dlco (ml-min·mmHg⁻¹)</td>
<td>37.6</td>
<td>33.3</td>
<td>39.1</td>
<td>32.4</td>
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</table>

Values are mean ± SD. * p < 0.05 significantly different to rest. # p < 0.05 significantly different to precedent exercise.
Table 3 SpO₂ and ventilatory parameters recorded during the two complementary trials in three triathletes. Cycling consisted of 10 min of cycling with a spontaneous breathing pattern (C₁), followed by 10 min of cycling with an imposed breathing pattern corresponding to that of running (C₃), then 5 min of cycling with spontaneous breathing pattern (C₃). Running consisted of 10 min of running with a spontaneous breathing pattern (R₁), followed by 10 min of running with a breathing pattern similar to that of cycling (R₃), then 5 min of running with spontaneous breathing pattern (R₃).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Cycling</th>
<th>Running</th>
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<tbody>
<tr>
<td></td>
<td>C₁</td>
<td>C₃</td>
</tr>
<tr>
<td>SpO₂ (%)</td>
<td>95.2</td>
<td>95.7</td>
</tr>
<tr>
<td>VO₂ (ml·min⁻¹·kg⁻¹)</td>
<td>51.5</td>
<td>54.7</td>
</tr>
<tr>
<td>VE (l·min⁻¹)</td>
<td>72.8</td>
<td>95.0</td>
</tr>
<tr>
<td>VT (ml)</td>
<td>2292</td>
<td>1821</td>
</tr>
<tr>
<td>RR (breath·min⁻¹)</td>
<td>32.0</td>
<td>52.3</td>
</tr>
</tbody>
</table>

Values are mean ± SD. # p < 0.05 significantly higher to R

Complementary experiment

Exercise intensities and respiratory parameters

Similar VO₂, VE, VT, and RR were observed during this complementary experiment when compared with the values of the C-R and R-C trials, as shown in Table 3 in comparison with Table 1. In addition, no statistical differences were observed when the VT and RR of running was imposed during C₃, or inversely when the VT and RR of cycling was imposed during R₃.

Percent SpO₂

During the entire complementary experiment, SpO₂ showed a significant decrease from rest values (p < 0.05). Moreover, when the breathing pattern of cycling was imposed during running, the arterial hypoxemia was partially reduced, whereas the breathing pattern of running failed to worsen arterial hypoxemia during cycling. Thus, within the running trial, R₃ presented a significant increase in SpO₂ compared with the R₁ and R₂ values (Table 3; Fig. 2).

Discussion

The present study showed that constant submaximal running induced greater arterial hypoxemia than cycling performed at a similar metabolic rate. The breathing pattern of running was characterised by a higher respiratory rate and lower tidal volume than that of cycling. Exercise-induced arterial hypoxemia is multifactorial and is known to involve relative hypoventilation and/or altered pulmonary gas exchanges. Our results strongly suggest that relative hypoventilation caused the higher EIAH during running, while the ventilation-perfusion ratio acted as a protective mechanism for the reduced EIAH noted during cycling; both mechanisms were accompanied with an alteration in pulmonary diffusing capacity.

The VO₂ monitoring during the trials eliminated O₂ demand as a cause of the greater drop in SpO₂ during running, as previously reported by Rasmussen et al. [28]. In fact, these authors reported
higher EIAH during running and rowing compared with arm cranking in a 6-min “all-out” test, and they linked the difference in SpO₂ with the amount of recruited muscle mass – and thus with O₂ demands which varied between ergometers. Recently, other studies [10, 17, 30] have reported higher EIAH during treadmill running compared with ergometer cycling at maximal and submaximal effort. Because of the heterogeneity of the populations (runners, cyclists, triathletes, soccer players, and swimmers) and/or the use of ergometers that were either more or less familiar to the athletes, the authors did not observe similar VO₂max [10] or significant EIAH [17]. In the present study, we selected a homogeneous population of highly trained triathletes (some of them were on the French national team) and we used constant load exercise and the same metabolic rate during both running and cycling.

According to Dempsey and Wagner [6], the absolute individual SpO₂ of the present triathletes corresponded to moderate (88–93%) EIAH during incremental testing on a cycle ergometer, and thus to mild (93–95%) and moderate EIAH during the cycling and running segments of the trials, respectively. These results agree with recent studies of our group that showed similar drops in SpO₂ during running and cycling [8, 9]. It should be noted that the two trials consisted of 40 min of steady-state exercise and that they showed low variability in SpO₂ (Fig. 1). This was in contrast with the reports on SpO₂ of fast-increment or short-maximal protocols of other studies in the literature [13, 30] and led us to assume that EIAH was present throughout the entire trials.

Whatever the exercise modality, EIAH has mainly been attributed to relative hypoventilation [5] and/or an alteration in pulmonary gas exchange, i.e., ventilation/perfusion mismatching and/or diffusion limitation [4]. Alterations in pulmonary diffus-

VA/Q mismatching may have contributed to the aggravation of EIAH, mainly during the R segments. Indeed, (A–a)DO₂, which reflects VA/Q mismatching [27], greatly increased during and within each trial, resulting in higher (A–a)DO₂ for R of C-R and a trend for R of R-C. Unfortunately, we cannot totally explain the differences in EIAH observed in the present study by VA/Q mismatching, despite its presence.

Relative hypoventilation has recently been suggested as a predominant factor at submaximal metabolic rate. It was noted in all highly trained athletes [4, 26], though in some athletes it was corrected at maximal intensity [7].

The finding of greater EIAH with higher VE during treadmill running than cycling at submaximal intensity did not agree with previous studies that showed relative hypoventilation concomitant with low VE [7, 10, 27, 30]. In our study, running was characterised by a specific breathing pattern: high RR and low VT. Such a breathing pattern, frequently termed rapid shallow breathing [3, 33], has been reported to increase the VT/VE ratio, and therefore to induce a relative hypoventilation [3]. In contrast to running, cycling was characterised by a lower VE, which has also been described as a possible cause of relative hypoventilation. Running and cycling would thus both be characterised by a very suspicious increase in VT/VE and a decrease in VA, probably in relation with a different breathing pattern. We therefore hypothesised that running performed with a breathing pattern similar to that of cycling (VT and RR) would increase SpO₂, whereas ergometer cycling performed with a breathing pattern similar to that of running (VT and RR) would decrease it.

To test this hypothesis, two complementary randomised trials were performed by three volunteer triathletes, as detailed in the Methods section. As expected, running performed with a breathing pattern similar to that of cycling, i.e., R₂, induced a significant increase in SpO₂ up to that reported for cycling during C-R and R-C (Fig. 2). This result showed that a reduction in RR and a rise in VT during running improved SpO₂, probably by increasing VA. We thus assumed that the breathing pattern of running was responsible for the greater decrease in SpO₂ rather than running per se.

In contrast, the cycling performed with the breathing pattern similar to that of running, i.e., C₀, failed to worsen the decrease in SpO₂ (Table 3: Fig. 2). We thus speculate that the “crouched position” [14] of triathletic cycling, using a specific handlebar, served to protect from moderate EIAH. A recent study in fact showed increased homogeneity of the VA/Q distribution in the prone position compared with the supine position [23]. In addition, the authors found an increase in PaO₂ with a decreased coefficient of variation of VA in the prone position. The “crouched position” may have pathophysiological effects quite similar to
those of the prone position – particularly improved VA/Q – and thus may maintain SpO₂ in spite of an unfavourable breathing pattern.

From a methodological point of view, the measurement of SpO₂ via ear oximeter had the great technical advantage of non-invasiveness. EIAH is now commonly studied with a pulse oximetry system because of its high reliability and non-invasiveness, as described in the Methods section [10,22,27], although some studies do claim that pulse oximetry is not reliable for this use [2,12,29,34]. Our group demonstrated the accuracy of pulse oximetry when used in highly trained athletes, with a minimum drop of 4% from rest values in addition to a persistence of SpO₂ values for 3 min throughout a steady-state exercise [22,27]. Pulse oximetry did, however, limit the interpretation of our results so in the present study we completed ear oximetry with blood gas measurement. To our knowledge, only two studies have investigated EIAH in athletes during cycling and/or running in short submaximal (5 min) and maximal exercises using arterial catheterisation and a rectal thermocouple [13,30]. The present experiment already required two 40-min trials and a change in apparatus so we did not want to further impose an invasive method on our subjects; we instead chose to use pulse oximetry and arterialised blood gas measurements, as previously reported in triathletes [18]. Because of the relationship between PaO₂ and rectal temperature, the succession of running and cycling could have aggravated the PaO₂ decrease during the last segment of C-R and R-C. Interestingly, PaO₂ and SpO₂ relative to running and cycling were similar whatever the position of the modalities within the trials, suggesting that the blood temperature increase throughout the trials was not sufficient to have an impact on the PaO₂ and SpO₂ results. We are aware that only three triathletes for the complementary trials pose a drawback to interpreting the results. However, the triathletes’ high training and high level of compliance need to be taken into account and suggest that the bias was probably minimal. Moreover, the results are presented here as descriptive data.

The findings of the present study confirmed the trend of previous works [10,17,30] and demonstrated that EIAH is dependent on exercise modality. The similar variation in PaO₂ and SpO₂ exhibited during running and cycling, whatever the position in the C-R and R-C successions, suggested that they each induced specific pathophysiological mechanisms for EIAH development. Pulmonary diffusion alterations and/or VA/Q mismatching probably contribute to the overall mechanism, while the relative hyperventilation observed during running appeared to be linked to breathing pattern and likely had the major effect on the EIAH observed. In contrast, the breathing pattern noted during cycling did not appear to be linked to the drop in SpO₂. We suggest that the better VA/Q distribution due to the “crouched-position” on the bicycle may have prevented further EIAH development.

In conclusion, the present study showed that exercise-induced arterial hypoxemia is dependent on the exercise modality. High level triathletes performed cycle-run and run-cycle trials at a similar metabolic rate that was close to their training intensities. Running induced greater arterial hypoxemia than cycling, whatever the position of the modality during trials, and a specific ventilatory response to each modality was noted as well. Based on these results, practical advice for triathletes and coaches would be to pay particular attention to breathing pattern during the run segment of training and races in order to limit the potentially deleterious effect of exercise-induced arterial hypoxemia.

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Author: please check if $\bar{V}_s$ and $\bar{V}_d$ have been assigned correctly in the different expressions.