Acute Effects of Using Added Respiratory Dead Space Volume in a Cycling Sprint Interval Exercise Protocol: A Cross-Over Study

Natalia Danek 1,*, Kamil Michalik 2, Marcin Smolarek 1 and Marek Zatoń 1

1 Department of Physiology and Biochemistry, Faculty of Physical Education and Sport, University School of Physical Education in Wrocław, 51-612 Wrocław, Poland; marcin.cluby@interia.eu (M.S.); marek.zaton@awf.wroc.pl (M.Z.)
2 Department of Human Motor Skills, Faculty of Physical Education and Sport, University School of Physical Education in Wrocław, 51-612 Wrocław, Poland; kamil.michalik@awf.wroc.pl
* Correspondence: natalia.danek@awf.wroc.pl; Tel.: +48-3473359; Fax: +48-713473036

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Abstract: Background: The aim of the study was to compare acute physiological, biochemical, and perceptual responses during sprint interval exercise (SIE) with breathing through a device increasing added respiratory dead space volume (ARDSV) and without the device. Methods: The study involved 11 healthy, physically active men (mean maximal oxygen uptake: 52.6 ± 8.2 mL·kg⁻¹·min⁻¹). During four visits to a laboratory with a minimum interval of 72 h, they participated in (1) an incremental test on a cycle ergometer; (2) a familiarization session; (3) and (4) cross-over SIE sessions. SIE consisted of 6 × 10-s all-out bouts with 4-min active recovery. During one of the sessions the participants breathed through a 1200-mL ARDSV (SIEARDS). Results: The work performed was significantly higher by 4.4% during SIEARDS, with no differences in the fatigue index. The mean respiratory ventilation was significantly higher by 13.2%, and the mean oxygen uptake was higher by 31.3% during SIEARDS. Respiratory muscle strength did not change after the two SIE sessions. In SIEARDS, the mean pH turned out significantly lower (7.26 vs. 7.29), and the mean HCO₃⁻ concentration was higher by 7.6%. Average La⁻ and rating of perceived exertion (RPE) did not differ between the sessions. Conclusions: Using ARDSV during SIE provokes respiratory acidosis, causes stronger acute physiological responses, and does not increase RPE.

Keywords: sprint interval exercise; cardiorespiratory responses; blood lactate; added respiratory dead space; respiratory acidosis

1. Introduction

Interval training can be described as intermittent high-intensity exercises divided by periods of incomplete recovery (e.g., lower-intensity work) [1]. Among its most popular types, there is sprint interval training (SIT), which consists in performing maximum-intensity work (generating the highest possible power, the so-called “all-out” training) [2]. A single session normally consists of two to six efforts of 10–30-s, with recovery lasting longer (e.g., several minutes) and a usual total session time of 10–30-min [3]. With reference to the generally recommended moderate-intensity continuous training, SIT is considered an effective and time-efficient strategy for improving general physical capacity and cardiorespiratory capacity (e.g., maximal oxygen uptake (VO₂max)), as well as lowering the risk of cardiometabolic diseases in the healthy population [4]. A study by Hazell et al. [5] based on a 2-week training program revealed that 10-s efforts (with 4-min recovery) efficiently improved cardiorespiratory capacity as compared with the “classic” SIT protocol, involving 4–6 × 30-s efforts with 4-min rest. However, solutions are still being sought that will increase the training effects without additional time expenditure.
Several studies have analyzed, during single effort or regular training, the effects of applying modifications of inhaled air composition by using various types of gas mixtures, e.g., increasing the amount of carbon dioxide (CO₂) in the inhaled air [6], training masks increasing respiratory resistance [7,8], or added respiratory dead space volume (ARDSV) [9,10]. Compared to the Elevation Training Mask® (ETM) (Training Mask LLC, Cadillac, Michigan) or an airflow restriction mask (ARM) [7,8], a device used to increase respiratory dead space volume has an additional corrugated pipe of a certain length, but it has no valves to increase breathing resistance [9,10]. With reference to this latest area, research is available in which ARDSV was used during continuous effort of constant intensity [11] and during progressive effort [12]. Several experiments have confirmed the effectiveness of 1000-mL ARDSV in developing physical capacity (as reflected in VO₂max) in regular high-intensity interval training among swimmers [13] and triathletes [14]. On the other hand, 30-min training at a level of 60% VO₂max performed for 6 weeks (twice a week) with ARDSV of 1000–1200-mL did not improve VO₂max in young physically active males [15] or in swimmers [9]. This suggests that there may be a specific effort intensity that conditions achieving the desired adaptive changes during ARDSV application. Thus, it is necessary to examine acute physiological, biochemical, and psychological responses during a single sprint interval exercise (SIE) session and to compare the results with those obtained under standard conditions to determine whether this approach can provide a stronger training stimulus. This will help plan regular training, especially for individuals who make intensive efforts in conditions of increased CO₂ concentration in the inhaled air, e.g., divers, firefighters, miners, or astronauts.

Breathing with ARDSV leads to CO₂ accumulation and an increase in its partial pressure in arterial (PaCO₂) and venous blood (pCO₂) [11,16]. It was found that PaCO₂ could be determined in an ergospirometry test by using end-tidal partial pressure of CO₂ (PETCO₂) [17]. Increased partial pressure of CO₂ in blood (>45 mm Hg) results in high concentrations of hydrogen ions (H⁺), low pH (<7.35), and raised bicarbonate (HCO₃⁻) concentration (>30 mm Hg), which is a state referred to as respiratory acidosis or hypercapnic acidosis [18]. In response to increased blood CO₂ levels, chemoreceptors provide respiratory feedback [19] by raising respiratory ventilation (VE) through its components: tidal volume (VT) and respiratory frequency (RF) [20]. Higher VE is required to maintain PaCO₂ and H⁺ regulation with any metabolic rate [16]. It has been proved that higher VE leads to increased oxygen uptake (VO₂) as a result of higher respiratory muscle activity [6,8]. In turn, an increased effort of the respiratory muscles can lead to their fatigue. It has been suggested that changing the ventilation pattern can support respiratory muscle training (RMT). Moreover, increased VE, especially through a rise in RF, can increase the rating of perceived exertion (RPE) [21]. From a psychological point of view, understanding these responses is important, as protocols that are better perceived (lower RPE) are more frequently chosen during regular training. Additionally, the lowering of blood pH influences the rate of glycogenolysis, glycolysis, and lactic acid production, as well as the functioning of monocarboxylate transporter 1 (MCT1) across sarcolemma [22]. It is suggested that hypercapnia can reduce lactate (La⁻) release from muscles to blood [23,24].

So far, no study has compared actual responses during a single SIE session with ARDSV breathing. Therefore, the aim of this research was to determine the physiological and biochemical responses and RPE during a single SIE session consisting of 6 × 10-s bouts with an active 4-min rest interval with 1200-mL ARDSV breathing, as well as to compare the responses with those obtained during a session performed under standard conditions without breathing impediments. We assumed that the application of ARDSV and inhaling increased CO₂ concentrations would cause hypercapnic acidosis, which, in turn, would initiate deeper changes in the acid–base balance and reduction of blood La⁻ concentration. According to another hypothesis, this would increase the respiratory system activity, as reflected in higher VO₂. In addition, we tested a hypothesis that higher exercise VE would provoke greater respiratory muscle fatigue.
2. Materials and Methods

2.1. Participants

The study involved 11 healthy, physically active males who volunteered to participate. Each of them declared a minimum of 5 h per week of physical exercise (sports classes at a university, gym, volleyball, football, running). No participant practiced sport at a professional level or was classified in a risk group for respiratory, cardiovascular, or metabolic diseases. They did not have experience with regular cycling training. There were no smokers among the participants. All became familiar with the study procedure and provided a written informed consent to participate. The study was approved by the University Research Ethics Committee (1/2019) and followed the tenets of the Declaration of Helsinki (PN-EN ISO 9001:2001 certificate). Detailed characteristics of the respondents are presented in Table 1.

Table 1. Participants’ characteristics (x ± SD).

| Variables          | Values       |
|--------------------|--------------|
| Age (years)        | 22.4 ± 3.9   |
| Body height (cm)   | 181.0 ± 7.9  |
| Body mass (kg)     | 77.1 ± 10.8  |
| Physical activity  | 7.5 ± 1.5    |
| Systolic blood pressure (mm Hg) | 124 ± 10 |
| Diastolic blood pressure (mm Hg) | 70 ± 8 |
| FVC (l)            | 6.9 ±1.0     |
| FEV₁ (l)           | 5.1 ± 0.9    |
| FEV₁ · FVC⁻¹ (%)   | 74.1 ± 9.9   |
| PIF (l · s⁻¹)      | 3.2 ± 1.5    |
| PEF (l · s⁻¹)      | 9.4 ± 1.8    |

FVC—forced vital capacity, FEV₁—forced expiratory volume in 1 s, FEV₁ · FVC⁻¹—Tiffeneau index, PIF—peak inspiratory flow, PEF—peak expiratory flow.

2.2. Study Design

The research included 4 visits to a laboratory with an interval of at least 72 h. During the visits, sessions of exercises on a cycle ergometer were conducted. All sessions were supervised by the same investigators and performed in the morning, 2 h after breakfast. During the experiment, the participants maintained physical activity patterns and were to refrain from exercise, alcohol, and caffeine for 24 h before each laboratory session. During the first visit, body mass (kg) and body height (cm) were measured with WPT 200 medical scales (Radwag, Radom, Poland); resting arterial blood pressure was evaluated with an aneroid sphygmomanometer (Riester, Jungingen, Germany); also, a spirometry test and an incremental exercise test (IET) were performed to determine cardiorespiratory capacity. The second visit included familiarization with the cycling SIE protocol and with ARDS₉ breathing. During the third and fourth visits, cross-over SIE sessions were conducted in a random order: the participants were breathing under standard conditions in one session and with ARDS₉ in another session, and subsequently the other way round.

2.3. Spirometry Test

The spirometry test was performed by using a Quark b² ergospirometer (Cosmed, Milan, Italy). It involved an inspiration with a maximum volume preceded by 2, 3 quiet breaths and ended with an intense exhalation with a maximum airflow, resulting in a minimum volume of residual air. In the course of the respiratory test, the following parameters were recorded: peak expiratory flow (PEF), peak inspiratory flow (PIF), forced vital capacity (FVC), and forced expiratory volume in 1 s (FEV₁). Each participant took 3 trials, with the first one for familiarization. Tiffeneau index (FEV₁ · FVC⁻¹) was calculated by the dedicated software.
2.4. Incremental Exercise Test (IXT)

This was performed on an Excalibur Sport cycle ergometer (Lode BV, Groningen, Netherlands) in accordance with a ramp incremental test protocol with a linear load pattern. The test started with a load of 0 W, which increased by ca. 0.28 W every second [25]. The pedaling frequency of above 60 rpm was maintained. The tested person was breathing through a mask, and the expired air was analyzed by a Quark b² device (Cosmed, Milan, Italy). Before the examination, the device was calibrated with atmospheric air and a gas mixture composed of 5% CO₂, 16% O₂, and 79% N₂. Breathing parameters were recorded breath by breath. VE, Rf, VT, and VO₂ were measured, and the results were averaged every 30-s and converted to minute values. Heart rate (HR) was determined with a S810 sport tester (Polar Electro, Kempele, Finland) and recorded by the Quark b² analyzer software. VO₂max was registered as the highest 30-s average value at a plateau of VO₂ < 1.35 mL·kg⁻¹·min⁻¹ despite the increasing load or if at least 2 of the following criteria were met: (1) volitional exhaustion, (2) predicted HRmax ≥95% (220—age), (3) respiratory rate ≥1.15. Maximal workload (Wmax) was determined as power at the end of the test. Capillary blood was collected from a hand fingertip to heparinized capillaries in the third minute after the test to determine La⁻ concentration in a photometer (LP 400, Dr. Lange, Berlin, Germany). RPE was evaluated with the use of the Borg scale [26] immediately after the test.

2.5. Cycling Sprint Interval Exercise Sessions

Both SIE sessions were performed on a cycle ergometer (Ergomedic Monark 894, Vansbro, Sweden) in accordance with the protocol described by Danek et al. [27] and presented in Figure 1. Each session was preceded by a 10-min warm-up at 60% VO₂max obtained in the incremental test; during the warm-up, two 5-s all-out accelerations were performed in the third and sixth minute. The warm-up was followed by a 5-min rest in a sitting position. In the main study part, the participants conducted 6 × 10-s bouts with an individual load of 7.5% of the body mass and an active break of 4-min with a load of 50 W and frequency of 50 rpm. Standardized verbal encouragement for each participant was provided throughout the during the bouts. During one SIE session, ARDS₉ (SIEARDS) (Figure 2) was applied after warm-up, two min before the first bout, and removed after the cool-down. The total time of ARDS₉ application equaled 27-min. The second session was performed under standard conditions without ARDS₉ (SIESTD).

![Figure 1. Cycling sprint interval exercise (SIE) protocol SIESTD—standard protocol, SIEARDS—protocol with the added respiratory dead space volume.](image)

The Quark b² analyzer (Cosmed, Milan, Italy) measured VE and its components: Rf and VT, VO₂, expired CO₂ (VCO₂), fraction of inspired oxygen (FiO₂), fraction of inspired CO₂ (FiCO₂), end-tidal partial pressure of oxygen (PₑTₒ₂), PₑT₁₆CO₂, time of inspiration (Ti), time of expiration (Te), total time of the respiratory cycle (Ttot), and the ratio of inspiration time and respiratory cycle time (Ti · Ttot⁻¹). The software also calculated ventilatory equivalents for oxygen (VE · VO₂⁻¹) and for CO₂ (VE · VCO₂⁻¹), as well as oxygen pulse (VO₂ · HR⁻¹). HR was determined with a S810 sport tester (Polar Electro, Kempele, Finland). The results were averaged every 30-s and converted to minute values.

Before and after SIE, inspiratory muscle strength (maximal inspiratory pressure, PImax) and expiratory muscle strength (maximal expiratory pressure, PEmax) were measured with a Micro RPM device (CareFusion, San Diego, CA, USA). To assess PImax, the tested person, in a standing position,
performed a maximum inspiration from the level of a maximum expiration. Then, to evaluate PE\text{max}, the individual exhaled starting from the maximum inspiration level. In both cases, a special nose stopper was fitted. Each participant took 3 trials each time, and the highest recorded values were selected for further analysis.

![Image](image_url)

**Figure 2.** One of the participants with the added respiratory dead space volume (ARDS\text{V}) device.

Capillary blood was collected from a hand fingertip to heparinized capillaries in the third minute after each bout to determine blood acid–base balance: pH, pCO$_2$, current HCO$_3^-$ concentration, and blood oxygen saturation (SaO$_2$) with the use of a RapidLab 348 analyzer (Bayer, Germany), as well as La$^-$ concentration in a photometer (LP 400, Dr. Lange, Berlin, Germany).

### 2.6. Performance

The results obtained during both SIE sessions were analyzed with the consideration of peak power output (PPO), mean power output (MPO), and total work (W\text{tot}). These parameters were calculated with the MCE 2.0 software (MCE, Wroclaw, Poland) for 6 repetitions. Fatigue level was estimated by calculating the fatigue index (FI) with the following formula: \[100 \times (\text{total sprint MPO} \times \text{ideal sprint MPO}^{-1})\]—100; where total sprint MPO—sum of sprint MPO from all sprints, ideal sprint MPO—number of sprints (6) \times \text{the highest sprint MPO}. This formula has been recognized as the most valid and reliable method for assessing fatigue in multiple sprint tests [28].

### 2.7. Device Added Respiratory Dead Space Volume (ARDS\text{V})

ARDS\text{V} was created by a single-valve ambu-type mask and an attached 2.5-cm diameter ribbed snorkel to provide of 1200-mL total volume. Dead space volume was identical for each participant and measured by filling the snorkel with water and then transferring the volume to a graduated cylinder, as described by Szczepan et al. [9].

### 2.8. Statistical Analysis

The sample size was established a priori by using G*Power 3.1 software (v3.1.9.2, Kiel, Germany) [29]. The expected effect size (ES) was set at (Cohen’s $f$) 0.85, the $\alpha$ level was set at 0.05, and the power (1-$\beta$) was set at 0.8 [30]. The 11 participants in the group were necessary and finally recruited.

The average values of cardiopulmonary parameters in both SIE sessions were calculated for 25-min (1-min of work, 20-min recovery, and 4-min of cool-down). The mean values of pH, La$^-$, current HCO$_3^-$ concentration, and RPE were determined on the basis of measurements taken after each of the 6 bouts.

The statistical analysis of the data was performed with the Statistica 13.3 software (StatSoft Inc., Tulsa, OK, USA). All of the results are presented as arithmetic means ± standard deviations (\[\overline{x} \pm \text{SD}\]). The Shapiro–Wilk test was applied to assess the normality of the tested characteristics distribution, and the Levene’s test evaluated the equality of variances. The Student’s t-test for dependent samples served to
evaluate the differences of selected variables between the SIE protocols. A two-way (protocol × number of bouts) analysis of variance (ANOVA) with repeated measures was used to compare PPO, pCO₂, and RPE. When a significant F ratio value was obtained, the Bonferroni post-hoc test was performed. The level of \( p < 0.05 \) was assumed statistically significant. ES, or Cohen’s d, was calculated in order to show the practical effect, with the following criteria: 0.1—trivial, 0.2—small, 0.5—medium, 0.8—large [30].

3. Results

In the incremental exercise test, the studied individuals achieved the following results: Wmax: 336.9 ± 40.9 W, VEmax: 148.8 ± 22.1 L · min⁻¹, Rfmax: 51.0 ± 7.9 L · min⁻¹, VTmax: 3.4 ± 0.5 L, VO₂max: 52.6 ± 8.2 mL · kg⁻¹ · min⁻¹, HRmax: 193 ± 7 beats · min⁻¹, La⁻peakIET: 12.9 ± 1.8 mmol · L⁻¹, RPEIET: 19.0 ± 0.9.

PPO did not differ statistically significantly between the bouts in the SIE protocols (Figure 3A). In both protocols, the values in the fifth and sixth bout were statistically significantly lower than those in the first repetition (Figure 3A). The mean power turned out statistically significantly higher in SIEARDS (787.6 ± 139.1 W) as compared with SIESTD (754.8 ± 132.7 W) (\( p < 0.01, t = 3.98, ES = 0.24 \)). The amount of work performed differed statistically significantly between the protocols (\( p < 0.01, t = 3.98, ES = 0.24 \)) and equaled 45.3 ± 8.0 kJ and 47.3 ± 8.3 kJ, respectively, in SIESTD and SIEARDS. No statistically significant difference was observed with regard to FI (\( p = 0.10, t = 1.82 \)).

Peak La⁻ concentration amounted to 13.9 ± 1.9 mmol · L⁻¹ in SIESTD and 12.8 ± 1.5 mmol · L⁻¹ in SIEARDS and did not significantly differ between the sessions (\( p = 0.09, t = 1.89 \)). Mean La⁻ concentration after 6 bouts did not differ between the protocols, either (\( p = 0.08, t = 1.95 \)); it equaled 11.0 ± 1.2 mmol · L⁻¹ and 10.2 ± 1.2 mmol · L⁻¹, respectively, in SIESTD and SIEARDS. Both of them (peak and mean La⁻ values) were close to assuming the threshold equal 0.05 to reject the null hypothesis. The value of pH did not differ statistically significantly in subsequent bouts between the protocols. In SIEARDS, starting with the second bout, it was statistically significantly lower in each subsequent bout than in the first one (\( p < 0.001 \)); in SIESTD, the same was observed starting with the third bout (\( p < 0.001 \)) (Figure 3B). Mean pH was statistically significantly lower (\( p < 0.01, t = 3.54, ES = 0.77 \)) in SIEARDS (7.26 ± 0.04) than in SIESTD (7.29 ± 0.04). The blood partial pressure of CO₂ differed statistically significantly after the fifth (\( p < 0.05 \)) and sixth bout (\( p < 0.01 \)). The value of pCO₂ lowered in the subsequent bouts in both protocols, beginning with the second one in SIESTD and with the third one in SIEARDS (Figure 3C). Mean current HCO₃⁻ concentration differed between the protocols and was statistically significantly higher (\( p < 0.05, t = 3.09, ES = 0.74 \)) during SIEARDS (15.6 ± 1.6 mmol · L⁻¹) as compared with SIESTD (14.5 ± 1.5 mmol · L⁻¹). SaO₂ did not differ statistically significantly between the investigated conditions, remaining within the physiological norm of 95–98%.
The highest RPE equaled 17.6 ± 1.4 in SIE_{STD} and 18.0 ± 1.4 in SIE_{ARDS} and did not differ statistically significantly (\( p = 0.42, t = 0.84 \)). Mean RPE for the 6 bouts did not differ between the protocols (\( p = 0.40, t = 0.89 \)) and totaled 15.2 ± 1.0 in SIE_{STD} and 14.9 ± 0.7 in SIE_{ARDS}.

The inspiratory or expiratory muscle strength did not change in either SIE protocol. In addition, it did not differ statistically significantly between the applied conditions (Figure 4).

![Figure 4](image_url)  
**Figure 4.** Changes in maximal inspiratory (A) and expiratory (B) muscle strength before and after the SIE protocols.

The mean physiological parameters in both SIE protocols are compared in Table 2. The average values of VE, FiO₂, FiCO₂, and P_{ET}CO₂ kinetics during both SIE protocols are shown in Figure 5.

![Figure 5](image_url)  
**Figure 5.** The average values of (A) FiO₂, (B) FiCO₂, (C) P_{ET}CO₂, and (D) ventilation (VE)—two minutes before, during and four minutes after last bout in SIE_{STD} and SIE_{ARDS} (27 min total).
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The application of ARDS power in subsequent bouts is important for long-term training adaptations, as reported by Hazell presented. We found that during SIE.

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the work performed was statistically significantly higher when breathing with ARDS et al. [5]. Although the maximum power did not di

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The results of our own research indicate that breathing with air of altered composition increased blood $P_{ET}CO_2$ and $pCO_2$, but no hypercapnia occurred. The higher mean $P_{ET}CO_2$ (ca. 15%) maintained during the main part of SIE$_{ARDS}$ and the higher mean $pCO_2$ after the fifth and sixth bout caused a decrease in the average blood pH and an increase in its $HCO_3^-$ concentration. In other experiments where ARDS$_{VT}$ was applied, higher $P_{ET}CO_2$ values were obtained than in own research [33,34] despite lower volumes (500–600-mL). This may be due to the different nature of the effort, the individual tolerance to the investigated individuals to $CO_2$, vital lung capacity, and lower sensitivity of chemoreceptors in the stimulation of ventilation response and $CO_2$ elimination [35]. It is not surprising that blood pH was lower during ARDS$_{VT}$ breathing; it proves the occurrence of respiratory acidosis, as reported by Smołka et al. [15]. Breathing through a much smaller ARDS volume (350-mL) induced by the airflow restriction mask with special adaptation piece to gas analyzer, also triggered respiratory acidosis (lower blood pH) by inhaling $CO_2$ [8]. Respiratory acidosis probably also affects the acid–base balance at the muscular level, which may result in metabolic changes. Higher $HCO_3^-$ concentration was observed by Woorons et al. [36] during hypoventilation exercises, in which partial $CO_2$ pressure increases. This phenomenon can be a source of adaptation, leading to delayed acidosis and improved buffering capacity; it can also be beneficial for pH regulation and the development of the ability to produce energy via anaerobic metabolism [37]. This is especially important because the reduction of energy available from anaerobic glycolysis, muscular $H^+$ accumulation, and the increase in extracellular potassium are major fatigue regulators during high-intensity exercises such as maximum sprinting effort [28]. Detailed analysis requires further investigation at the muscular level, which we did not perform in this study.

The increased blood partial $CO_2$ pressure and $H^+$ concentration induced higher mean VE during SIE$_{ARDS}$. This was probably due to the stimulation of peripheral and central chemoreceptors by $CO_2$ [38,39]. However, no respiratory alkalosis was observed. Higher VE occurred through higher VT, which is in line with other studies [11,16,33,34]. The higher mean VE in SIE$_{ARDS}$ resulted in increased mean $VO_2$. In a study by Jensen et al. [16], no $VO_2$ increase was reported during effort with 500-mL ARDS$_{VT}$. In turn, other studies inform that the energy cost of respiratory muscle work was higher when breathing with ARDS$_{VT}$ [34], with a training mask [8], and with air enriched with $CO_2$ [6], which resulted from greater muscle involvement. Similar interpretation was provided by Woorons et al. [40], who used hypoventilation during exercise. Another factor contributing to higher $VO_2$ in SIE$_{ARDS}$. This may be related to the right-shift of the hemoglobin dissociation curve during acidosis, which is in accordance with the Bohr effect [41]. This increases the $O_2$ diffusion gradient between capillaries and muscle cells, leading to a greater use of $O_2$ in the cellular metabolism. The phenomenon confirms the statistically significantly lower $P_{ET}O_2$ during SIE$_{ARDS}$. We are aware that measuring hemodynamic parameters of heart performance such as stroke volume and cardiac output would provide more evidence to explain the impact of ARDS$_{VT}$ on oxygen transport and $VO_2$ during a single SIE session. Differences are likely to exist as mean HR was similar in both protocols. Therefore, other factors may also have been responsible for higher $VO_2$ and should be assessed in further studies.

Among the main VE regulators during exercise, there is the so-called central neural drive (central command) [21]. Moreover, it has been suggested that the central command preferentially regulates Rf and not VT [21]. Rf is also a sensory signal for RPE [42] and provides its neurophysiological explanation. Breathing through the aforementioned training mask limiting the air flow with special valves and increasing ARDS by 350-mL resulted in lower Rf; however, the authors did not report RPE [8]. In our study, we did not identify any differences in Rf or RPE when ARDS$_{VT}$ breathing was applied. Thus, the SIE$_{ARDS}$ session was not perceived as more difficult, which is of great importance for its implementation in regular training in a variety of populations. Similar findings were reported by Jung et al. [43], where RPE did not differ between normal breathing and respiration with ETM during continuous cycling (50 and 70% $VO_2$max). Unfortunately, we did not measure other psychological characteristics, such as the sense of pleasure. Nonetheless, protocols perceived as more pleasant are more likely to be chosen and performed by practitioners [44].
It is interesting that after both SIE protocols, there was no respiratory muscle fatigue as observed after performing maximum effort, e.g., a progressive test [45]. This suggests that ARDSV breathing is not a limiting factor for respiratory system effort despite the changed ventilation pattern—all the more so because the average ventilation did not exceed 60% of the maximum value. The applied snorkel of 1200-mL volume and 2.5-cm diameter does not seem to be a stimulus that could affect additional RMT. It is consistent with the last opinion by Shei [46], who explains recommendations to stimulus respiratory muscle training adaptations. A tube with a smaller diameter can increase resistance and engage the respiratory muscles to a greater extent. This corroborates the postulates by Illi et al. [47] indicating that increased respiratory muscle work induced by additional respiratory resistance improves endurance performance through RMT. This should become a subject of separate studies on long-term adaptation due to SIT with ARDSV.

Higher VO$_2$ may have influenced changes in cellular metabolism. However, we did not notice any differences in La$^-$ concentration between the SIE protocols, although there is a tendency for it to decrease in SIEARDS. Several studies have demonstrated that increased blood partial pressure of CO$_2$ can lower the release of La$^-$ from muscles [23,24]. On the other hand, Smołka et al. [15] did not report differences in La$^-$ concentration when the study participants breathed through 1200-mL ARDSV during a 30-min exercise at 60% VO$_2$max. We cannot entirely rule out the possibility that SIERADSV led to a lower muscle La$^-$ production than that during SIESTD. It is well known that blood La$^-$ concentration does not mirror muscle La$^-$ concentration. Solving this problem demands tissue analyses, which were not performed in this study.

It can be considered that the outcome of this study was limited by the fact that the participants were not blinded and that the differences in the amount of work performed and the lack of differences in RPE resulted from the placebo effect. Similar observations were earlier reported by Woorons et al. [37,40], who investigated the impact of hyperventilation. A corresponding problem refers to ARDSV, as it is impossible to conduct single- or double-blind trials. However, although a psychological effect cannot be ruled out in this study, it should be noted that the participants were not aware of or received no information on the potential impact of the tested method. It also seems very interesting to examine whether ARDSV breathing modifies the contribution of particular energy systems in satisfying the metabolic needs of working muscles during maximum sprinting efforts.

5. Conclusions

The application of 1200-mL of ARDSV during SIE is a simple method to induce acute stronger physiological responses and changes in the acid–base balance. SIEARDS sessions are not perceived as more difficult and can provide an alternative to the currently known training protocols. To stimulate RMT, other parameters of the applied device should be considered, e.g., increasing of the ARDSV snorkel diameter.

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