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CHANGES IN BREATHING PATTERN AND CYCLING EFFICIENCY AS A RESULT OF TRAINING WITH ADDED RESPIRATORY DEAD SPACE VOLUME

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ABSTRACT

Purpose. The aim of this study was to evaluate the impact of training with added respiratory dead space volume (ARDSV) on changes in a breathing pattern and cycling efficiency. **Methods.** Twenty road cyclists were equally divided into an experimental (E) and control (C) group. All of them were involved in a training program that included endurance training (at moderate intensity) and interval training (at maximal intensity). During semi-weekly endurance training, ARDSV (1000cm^3 tube) was introduced in the experimental group. Respiratory parameters, including, among others, oxygen uptake (VO_2), carbon dioxide excretion (VCO_2), end-tidal partial pressure of carbon dioxide ($PETCO_2$), pulmonary ventilation (VE), tidal volume (TV) and total work done during the tests (W), were measured before and after the experiment by a progressive and continuous test. **Results.** Higher $PETCO_2$ and TV in both groups during the progressive and continuous tests were observed. VCO_2 increased in group E during continuous test, while for group C only in the first four minutes of the test. VO_2 and VE increased only in group E during submaximal and maximal exercise. Total work increased during the continuous test in both groups (significantly higher in group C than E). However, total work during the progressive test increased only in group E. **Conclusions.** Training with ARDSV improved exercise capacity at maximal effort and was associated with an increase in maximal oxygen uptake. On the other hand, this type of training lead to a decrease in cycling efficiency, reducing in effect the benefits associated with an increase in VO_2max and reducing the ability to perform submaximal effort.

Key words: added dead space, breathing pattern, cycling efficiency

Introduction

Cardiorespiratory efficiency is determined not only by the amount of oxygen supplied and used (maximal oxygen uptake) by the cardiovascular system but also by the efficiency in removing excess carbon dioxide [1, 2]. Some researchers believe that pulmonary ventilation may limit maximum performance during aerobic exercise. An increase in pulmonary ventilation during exercise allows the lungs to adjust the partial pressure of carbon dioxide in the arterial blood (P_aCO_2), while the respiratory system helps control the hydrogen ion concentration in extracellular fluids [3]. However, reduced pulmonary ventilation causes an excessive increase of P_aCO_2 in the blood during effort, where increased P_aCO_2 levels are associated with greater blood and tissue acidosis [4]. A simultaneous increase in the partial pressure of carbon dioxide and decrease in blood pH is known to adversely affect muscle contractile properties and metabolism (by limiting enzyme activity), contributing to fatigue [2, 5]. As a result, high levels of P_aCO_2 may indicate an insufficient increase in lung ventilation during effort and therefore limit the ability for maximal exercise. This may be due to mechanical respiratory constraints when reaching the upper limit of peak expiratory flow such insufficient respiratory muscle strength or even reduced chemoreceptor responsiveness. On the other hand, lower ventilation is also connected with decreased respiratory muscle work and may be the cause of decreasing blood flow to the respiratory muscles while increasing blood flow (by about 10%) to limb muscles. Such a mechanism may delay the onset of fatigue [4].

P_aCO_2 can be measured noninvasively by sampling end-tidal partial pressure of carbon dioxide ($P_{ET}CO_2$) [4, 6]. In most cases, the resting value of $P_{ET}CO_2$ is slightly lower than $PaCO_2$ as decreased blood flow distribution in the lungs causes alveolar partial pressure of carbon dioxide to be lower than arterial partial pressure of carbon dioxide. However, $P_{ET}CO_2$ is higher than P_aCO_2 at submaximal or maximal effort, causing an increase in pulmonary blood flow and an increase in the delivery rate of carbon dioxide (CO_2) to the lungs [7, 8]. During effort, the difference between P_aCO_2 and $P_{ET}CO_2$ is mainly connected to respiratory rate, as the amount of expired CO_2 does not plateau. Therefore, for a given value of alveolar CO_2, $P_{ET}CO_2$ is higher while the breathing frequency is lower [4]. According to Benallall and Busso [8], the difference between P_aCO_2 and $P_{ET}CO_2$ during exercise is influenced by both respiratory rate and tidal volume, where the greater the volume and respiration rate, the greater difference between these two measures. Additionally, a high value of $P_{ET}CO_2$ may indicate high cardiovascular and ventilation efficiency. As a result, $P_{ET}CO_2$ can serve as a marker that combines measures of performance and ventilation efficiency although it

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cannot be regarded as an independent physiological variable [4].

In order to improve the efficiency of the gas exchange system and respiratory muscles, techniques such as specific inspiratory or expiratory muscle training or hyperpnoea training have been introduced [9–12]. Several authors reported that these methods improve breathing efficiency by increasing tidal volume while decreasing respiratory rhythm, resulting in a decrease in submaximal and an increase in maximal ventilation efficiency [9, 13, 14]. Furthermore, increased respiratory muscle strength following inspiratory training may contribute to a reduction in relative tension during submaximal exercise [14, 15]. A study by the present author also found that increasing dead space tidal volume as one form of respiratory training showed increases in pulmonary ventilation (VE) and respiratory tidal volume (TV) [16]. As it was not known whether the observed changes in the breathing process affected the efficiency of carbon dioxide removal, it was assumed that the increase in TV reduced $P_{ET}CO_2$.

Therefore, the aim of this study was to evaluate the impact of inspiratory training with added respiratory dead space volume on the breathing pattern, amount of exhaled carbon dioxide, and cycling efficiency. It was hypothesized that a change in breathing pattern, characterized by an increase in TV, would enhance work efficiency.

Material and methods

The study involved a 20-person group of road cyclists (all men) who were training together. The participants were evenly divided into an experimental (E) and control (C) group. Before the experiment, no statistically significant differences were noted between the experimental and control groups for mean values of age (E = 16.5 ± 0.5 y, C = 17.1 ± 0.7 y), body height (E = 180.6 ± 3.8 cm, C = 180.2 ± 4.4 cm), body mass (E = 70.4 ± 5.4 kg, C = 69.4 ± 7.2 kg), and maximal oxygen uptake (E = 64.4 ± 4.6 ml · kg^{-1} · min^{-1}, C = 64.2 ± 4.7 ml · kg^{-1} · min^{-1}) and work capacity (C = 285.4 kJ ± 41.8, C = 274.9 ± 30.9 kJ) during a progressive cycloergometer test.

The study was approved by the Ethics Committee of the University School of Physical Education in Wroclaw, Poland and carried out in accordance with the Declaration of Helsinki. Additionally, all participants provided their written informed consent prior to testing.

The experiment lasted 10 weeks during which the participants continued their existing training program that involved both aerobic and anaerobic cycling exercises. Three types of training were employed: (1) interval training consisting of 40-second repetitions at maximum intensity followed by six minutes of active recovery by exercising at a low intensity (50–60% maximum heart rate, HRmax), (2) high intensity interval training consisting of alternating exercise, where five minutes was performed at maximum intensity (95–100% HRmax) followed by fifteen minutes of medium-intensity exercise (65–70% HRmax), and (3) steady-state endurance training performed at 70–80% HRmax. Throughout each training session heart rate was monitored using a S810 heart rate monitor (Polar Sports, Finland). One type of training was employed once a day in the order provided and was then followed by a day of rest (i.e., first day interval training, second day high intensity interval training, third endurance training, a day of rest, and the next day again interval training, etc.). Each daily training session lasted 120 to 150 min.

An apparatus increasing respiratory dead space was introduced in the experimental group. It consisted of a mask with a 1000 cm^3 tube worn over the mouth, forcing the inhalation of additional atmospheric air and thereby diluting the amount of exhaled air left in the mask and tube after each previous exhalation. This device was used only when the experimental group performed endurance training (therefore every fourth day during the 10-week period). The control group performed the same exercises as the experimental group but without any modification to their breathing pattern.

Each participant was subjected to two physiological exercise tests prior and after the 10-week experiment. The first was a progressive test (incremental load) whereas the second was a continuous test performed at constant load. The tests were separated by an interval of one week with the progressive test being the first performed. During the tests, the experimental group did not use the apparatus for increasing respiratory dead space volume.

The exercise tests were performed in laboratory conditions at the Exercise Laboratory at the University School of Physical Education in Wroclaw, Poland (PN-EN ISO 9001:2001 certified).

The progressive test was performed on an Excalibur Sport cycle ergometer (Lode B.V., Netherlands), which was calibrated before each test session according to manufacturer's instructions. The test began at a load of 50 W, which was increased by 50 W every three minutes until the participant reached exhaustion. The cycle ergometer was controlled with a computer program that also registered instantaneous power, time, and speed and used these values to calculate the total work done during the test. Measurement of respiratory parameters began two minutes before the test and continued for five minutes after it was completed (after reaching exhaustion) and included: oxygen uptake (VO_2), carbon dioxide excretion (VCO_2), end-tidal partial pressure of carbon dioxide ($P_{ET}CO_2$), pulmonary ventilation (VE), tidal volume (TV), and breathing frequency (BF). Measurement was performed by having the participants wear a mask connected to a Quark b² gas analyzer (Cosmed, Italy). The gas analyzer was calibrated before each test with a reference gas mixture of: CO_2 – 5%, O_2 – 16%, and N_2 – 79%. Oxygen consumption and oxygen consumption per kilogram of body mass were calculated for each participant

based on the composition of exhaled air and pulmonary ventilation.

The continuous test was also performed on the Excalibur Sport cycle ergometer. A 10-minute warm-up on the ergometer preceded the test, during which the load for each participant was chosen so that heart rate did not exceed the anaerobic threshold (defined during the progressive test). The test was performed at a constant load of 85% of maximal aerobic power and continued until exhaustion. Maximal aerobic power was treated as the value at which maximum power was produced for at least 90 seconds during the progressive test. Exercise was performed at a self-selected pedal frequency. The computer program controlling the cycle ergometer modulated the workload in response to changes in pedaling frequency. The same respiratory parameters (VO_2, VCO_2, $P_{ET}CO_2$, VE, TV, and BF) as in the progressive test were measured, following the exact same method. Oxygen consumption and oxygen consumption per kilogram of body mass was then calculated based on the composition of exhaled gas and pulmonary ventilation.

Respiratory parameters (VO_2, VCO_2, $P_{ET}CO_2$, VE, TV, and BF) were measured breath by breath. For later analysis, the parameters recorded during progressive test were averaged over 60-second periods. Measures of VCO_2 and $P_{ET}CO_2$ during the continuous test were also averaged over 60-second periods, while VO_2, VE, TV, and BF were averaged in the steady-state phase (5^{th}–10^{th} minute of the test). In addition, a new variable was introduced measuring the percentage of maximal oxygen uptake (%VO_2max) by the formula [VO_2/VO_2max × 100%] and also calculated in the steady-state phase. Only data up to the 10-minute mark during the continuous test were analyzed as some of the participants were unable to continue exercising after this point.

Statistical analysis was performed using Statistica ver. 10.0 (Statsoft, Poland). The arithmetic means and standard deviations of all measured parameters were calculated. Statistical significance was determined using Student's *t* test for all dependent variables. The level of statistical significance was set at $p < 0.05$. Student's *t* test was also applied to determine the significance of differences between groups.

Results

End-tidal partial pressure of carbon dioxide ($P_{ET}CO_2$) significantly increased during the continuous test in the experimental group between the 2^{nd} and 10^{th} minute of the test and in the control group at the 3^{rd} and 4^{th} minutes, and between the 6^{th} and 10^{th} minute of the test (Fig. 1). In the progressive test, $P_{ET}CO_2$ values increased in both groups, with statistically significant differences in the experimental group observed at a load of 250–350 W and in the control group at 300–350 W (Fig. 2).

The amount of carbon dioxide excreted (VCO_2) significantly increased upon conclusion of the experiment

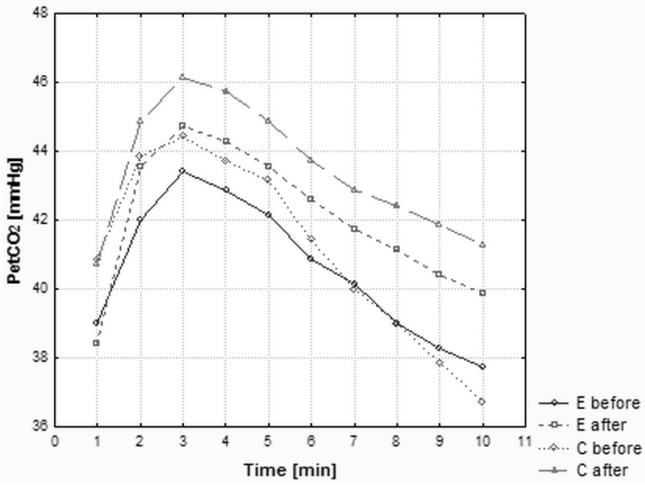

Figure 1. $P_{ET}CO_2$ measured for the experimental (E) and control (C) groups during the continuous test before and after the experiment

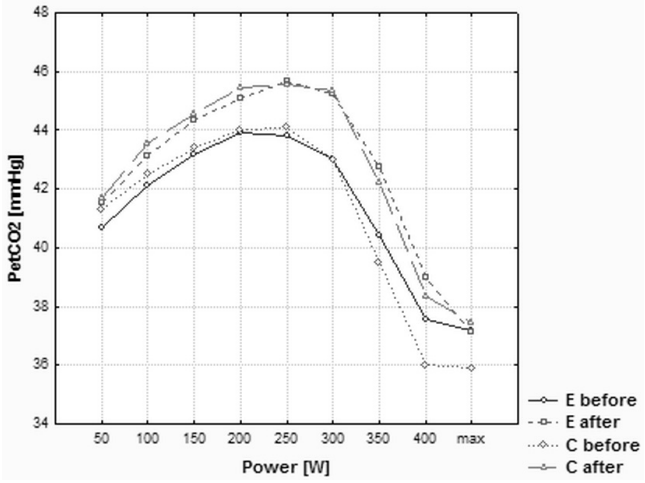

Figure 2. $P_{ET}CO_2$ measured for the experimental (E) and control (C) groups during the progressive test before and after the experiment

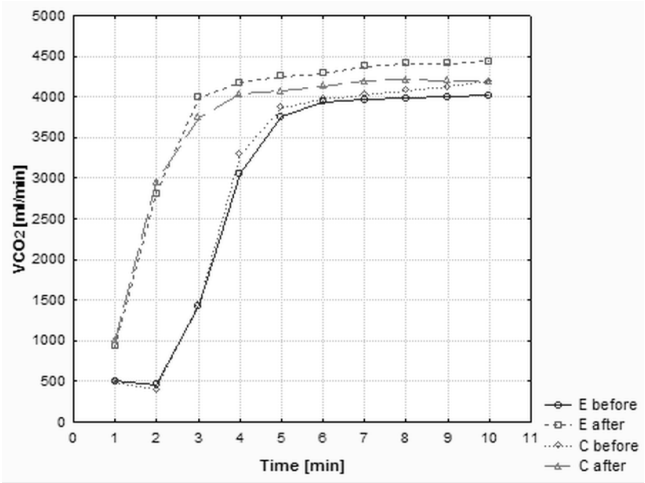

Figure 3. VCO_2 measured for the experimental (E) and control (C) groups during the continuous test before and after the experiment

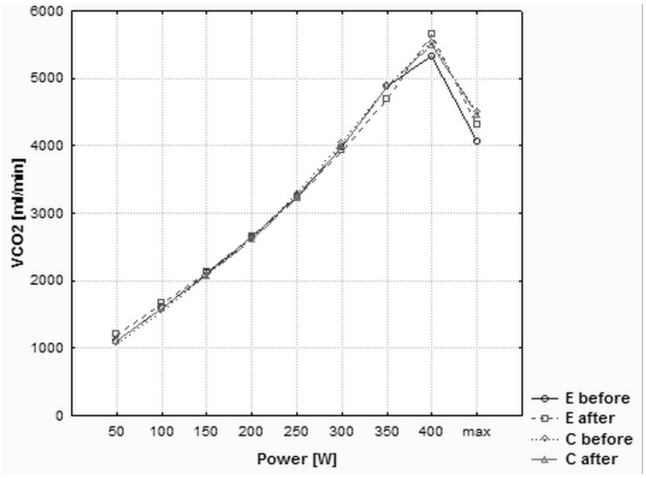

Figure 4. VCO_2 measured for the experimental (E) and control (C) groups during the progressive test before and after the experiment

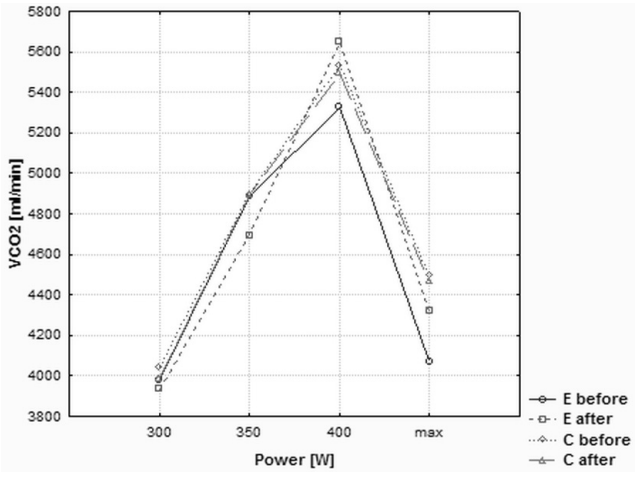

Figure 5. VCO_2 measured at maximum load for the experimental (E) and control (C) groups during the progressive test before and after the experiment

Table 1. Total work done and respiratory parameters for the experimental (E) and control (C) groups during the progressive test before and after the experiment

Parameter	Group E before	Group E after	Group C before	Group C after
	$\bar{x}$ ± SD	$\bar{x}$ ± SD	$\bar{x}$ ± SD	$\bar{x}$ ± SD
W (kJ)	285.44 ± 41.85	324.90 ± 46.10*	274.99 ± 30.94	298.99 ± 42.20
VO_2max (l/min)	4.48 ± 0.36	4.77 ± 0.23*	4.38 ± 0.3	4.58 ± 0.47
VO_2max (ml/min/kg)	64.4 ± 4.6	67.9 ± 2.9*	64.3 ± 4.7	65.1 ± 5.1
VEmax (l/min)	160.2 ± 19.9	178.4 ± 18.6*	165.3 ± 18.3	168.8 ± 23.4
TVmax (l/breath)	2.93 ± 0.2	3.22 ± 0.29*	2.89 ± 0.35	3.14 ± 0.33*
BFmax (breaths/min)	55.11 ± 5.52	54.79 ± 3.99	57.76 ± 4.30	54.02 ± 3.16*

$\bar{x}$ – arithmetic mean, *SD* – standard deviation, W – total work done, VO_2max – maximal oxygen uptake
VEmax – maximal pulmonary ventilation, TVmax – maximal tidal volume, BFmax – maximal breathing frequency
* difference before and after experiment at $p > 0.05$

Table 2. Total work done and averaged respiratory parameters (between the 5th and 10th minute) for the experimental (E) and control (C) groups during the continuous test before and after the experiment

Parameter	Group E before	Group E after	Group C before	Group C after
	$\bar{x}$ ± SD	$\bar{x}$ ± SD	$\bar{x}$ ± SD	$\bar{x}$ ± SD
W (kJ)	257.4 ± 91.1	380.4 ± 111.8*	222.7 ± 33.2	375.9 ± 121.4*
VO_2 (l/min)	3.79 ± 0.59	4.25 ± 0.24*	3.91 ± 0.25	4.01 ± 0.30
%VO_2max	83.06 ± 9.48	88.11 ± 2.42	90.86 ± 2.66	90.57 ± 6.82
VE (l/min)	120.13 ± 19.59	126.00 ± 11.73*	127.50 ± 10.30	120.76 ± 14.87
TV (l/breath)	2.63 ± 0.33	3.08 ± 0.25*	2.54 ± 0.21	2.76 ± 0.25*
BF (breaths/min)	46.40 ± 6.92	41.56 ± 5.54*	50.50 ± 3.66	44.57 ± 4.49*

$\bar{x}$ – arithmetic mean, *SD* – standard deviation, W – total work done, VO_2 – mean oxygen uptake
%VO_2max – mean percentage of maximal oxygen uptake, VE – mean pulmonary ventilation
TV – mean tidal volume, BF – mean breathing frequency, * difference before and after experiment at p > 0.05

in each minute of the continuous test in the experimental group. In the control group, a significant increase was observed only between the 1st and 4th minute of the continuous test (Fig. 3). However, during the progressive test, VCO_2 significantly decreased at a load of 350 W and significantly increased at 400 W and also at maximum load (power) in the experimental group, while no statistically significant differences for VCO_2 were observed in the control group (Fig. 4 and 5).

After the experiment, only the experimental group in the progressive test featured significantly increased values for total work done, maximal oxygen uptake, and maximal pulmonary ventilation. Maximal tidal volume (TVmax) was found to significantly increase in both

groups after the 10-week period, whereas maximal breathing frequency (BFmax) significantly decreased only in the control group (Tab. 1).

The total work done in the continuous test was found to increase in both groups. In the experimental group, increased values were observed for pulmonary ventilation and tidal volume while breathing frequency decreased. The control group featured an increase in tidal volume with a decrease in breathing frequency (Tab. 2).

Discussion

A gradual rise in $P_{ET}CO_2$ is frequently observed during exercise of increasing intensity, reaching its maximum value at the beginning of respiratory compensation for lactic acidosis after which it slightly decreases and continues to do so until maximum effort is ceased. Rarely has $P_{ET}CO_2$ been observed to continue to increase until the cessation of exercise [7]. According to Yano et al. [17], the same can be seen during exercise performed at a constant intensity (70% of maximal oxygen uptake), with $P_{ET}CO_2$ first increasing and then decreasing. Similarly, the present study also found an initial increase and then decrease in $P_{ET}CO_2$ in both the progressive and continuous tests. These changes are associated with a buildup of carbon dioxide, whose concentration rises for a few minutes and then plateaus as exercise is continued or even decreases due to increased pulmonary ventilation [2, 17].

In a study on individuals with chronic heart failure, it was found that a reduction in maximal $P_{ET}CO_2$ values was correlated with a decrease in maximal oxygen uptake during incremental exercise [7]. In the present study, measures of maximal $P_{ET}CO_2$ during the progressive test were found to have increased after the 10-week period in both the experimental and control groups. However, significant increases in maximal oxygen uptake and total work were observed only in the experimental group.

Thus, it is quite probable that other factors than $P_{ET}CO_2$ contributed to the rise inVO_2max in the experimental group. It is believed, in the case of conditioned athletes, that the most important exogenous agent that improves VO_2max is high-intensity training [18]. However, in our study where exercise intensity was monitored using heart rate monitors (i.e., they performed the same intensity training), no significant differences were found in the results between the experimental and control groups. Therefore, further studies are needed to help explain how training with added respiratory dead space volume increases exercise capacity at maximum aerobic effort.

According to Bussotti et al. [4], individuals with very low exercise $P_{ET}CO_2$ (and therefore likely low P_aCO_2) have lower exercise efficiency, as evidenced by high submaximal oxygen uptake and also exercise acidosis levels. In such cases, the authors observed a specific breathing pattern characterized by high pulmonary ventilation due to increased breathing frequency, which provoked an increase in physiological dead space and the amount of work performed by the respiratory muscles.

In the present study, $P_{ET}CO_2$ measured during the continuous test increased in both groups, although the control group reached higher $P_{ET}CO_2$ values than the experimental group after the 10-week period. The experimental group presented higher oxygen consumption and pulmonary ventilation levels during the test than before the experiment as well as higher values than those of the control group. These results indicate that training with added respiratory dead space volume caused deterioration in exercise efficiency and could have mitigated the economizing effects on their endurance training.

However, increases in pulmonary ventilation are due to tidal volume and not to an increase in breathing frequency. High pulmonary ventilation with high tidal volume and low breathing frequency is considered to be desirable, as it reduces physiological dead space and is less taxing on the respiratory muscles [4]. Our results indicate that the control group performed more total work during the continuous test despite a smaller change in tidal volume. The gains, in view of the increases in the maximal respiratory parameters, the experimental group achieved in the progressive test were reduced in the continuous test probably due to a decrease in exercise efficiency. This is the most likely explanation why this group showed less dramatic improvements in the total work done than the control group. It also can be deemed that the change in the experimental group's breathing pattern did not lead to improved efficiency.

Anderson et al. [19] believe that breathing characterized by a reduction in breathing frequency with a simultaneous increase in tidal volume, triggering an increase in pulmonary ventilation, results in a reduction in end-tidal carbon dioxide tension. These authors suggest that lower $P_{ET}CO_2$ may indicate higher ventilation efficiency. Then again, it is necessary to overcome the elastic forces of the lungs and chest wall, hence the increase in tidal volume above critical volume is associated with increased respiratory function [20]. This may be the reason explaining the lower performance of the experimental group, although this issue requires further investigation.

Exercise performed with added respiratory dead space volume from 0.2 to 2 liters and exercise performed with some form of breathing restriction, such as swimming or diving, is known to cause hypercapnia, or an increase in the partial pressure of carbon dioxide in arterial blood [5, 21–24]. This drop in the chemical balance of the system irritates chemoresponsive areas within the cardiovascular system. As a result, pulmonary ventilation increases through an increase in tidal volume [23, 24] and an increase in breathing frequency [25]. It was proven that ventilation increases as $P_{ET}CO_2$ rises and that ventilation is constant below a given $P_{ET}CO_2$ level – creating a ventilatory threshold in relation to

$P_{ET}CO_2$ [26]. The present study observed an increase in $P_{ET}CO_2$ and VCO_2 during exercise that was accompanied with an increase in pulmonary ventilation. This may have been caused by changes in chemoresponsive sensitivity. In the literature on the subject, it was shown that prolonged or frequent environmental exposure to re-inspiring expired air affects the hypoxic ventilatory response, or the increased respiratory response to changes in oxygen tension in exhaled air. This is characterized by a greater increase in pulmonary ventilation during the initial phase of hypoxia [27–29]. In the present study, it is possible that the experimental groups' sensitivity to CO_2 and hydrogen ions changed due to training with increased respiratory dead space volume. Such conditions could provoke an increase in the partial pressure of carbon dioxide and a decrease in blood pH levels even at exercise performed at the same intensity (in terms of power) [30]. These are factors that, similar to changes in the partial pressure of oxygen, may irritate the cardiovascular system's chemoreceptors.

Conclusions

Training with added respiratory dead space volume caused a drop in exercise efficiency, thereby reducing the benefits of an increase in maximal oxygen uptake and reducing the ability to perform submaximal exercise.

When performing maximal exercise (such as during a progressive test) that includes high intensity anaerobic efforts, training with added respiratory dead space volume increases exercise capacity by increasing maximal oxygen uptake.

References

1. Brown S.J., Cardio-respiratory system efficiency in trained endurance cyclists. *Med Sportiva*, 2010, 14 (4), 176–181.
2. Jones N.L., An obsession with CO2. *Appl Physiol Nutr Metab*, 2008, 33 (4), 641–650, doi: 10.1139/H08-040.
3. Wyatt F.B., McCarthy J.P., Age associated declines in exercise time to exhaustion and ventilatory parameters in trained cyclists. *J Exerc Physiol*, 2003, 6 (1), 12–17.
4. Bussotti M., Magrì D., Previtali E., Farina S., Torri A., Matturri M. et al., End-tidal pressure of CO2 and exercise performance in healthy subjects. *Eur J Appl Physiol*, 2008, 103 (6), 727–732, doi: 10.1007/s00421-008-0773-z.
5. Kapus J., Ušaj A., Kapus V., Štrumbelj B., The difference in respiratory and blood gas values during recovery after exercise with spontaneous versus reduced breathing frequency. *J Sport Sci Med*, 2009, 8 (3), 452–457.
6. Gama de Abreu M., Melo M.F.V., Giannella-Neto A., Pulmonary capillary blood flow by partial CO2 rebreathing: importance of the regularity of the respiratory pattern. *Clin Physiol*, 2000, 20 (5), 388–398, doi: 10.1046/j.1365-2281.2000.00271.x.
7. Tanabe Y., Hosaka Y., Ito M., Ito E., Suzuki K., Significance of end-tidal PCO_2 response to exercise and its relation to functional capacity in patients with chronic heart failure. *Chest*, 2001, 119 (3), 811–817, doi:10.1378/chest.119.3.811.
8. Benallal H., Busso T., Analysis of end-tidal and arterial PCO_2 gradients using a breathing model. *Eur J Appl Physiol*, 2000, 83 (4-5), 402–408, doi: 10.1007/s004210000260.
9. Tong T.K., Fu F.H., Chung P.K., Eston R., Lu K., Quach B. et al., The effect of inspiratory muscle training on high-intensity, intermittent running performance to exhaustion. *Appl Physiol Nutr Metab*, 2008, 33 (4), 671–681, doi: 10.1139/H08-050.
10. Passfield L., Dobbins T., Myers S., Reilly M., Williams E.M., Acute cardio-respiratory changes induced by hyperpnoea using a respiratory muscle trainer. *Ergonomics*, 2005, 48(11–14),1423–1432,doi:10.1080/00140130500101510.
11. Gething A.D., Williams M., Davies B., Inspiratory resistive loading improves cycling capacity: a placebo controlled trial. *Br J Sports Med*, 2004, 38 (6), 730–736, doi: 10.1136/bjsm.2003.007518.
12. Markov G., Spengler C.M., Knopfli-Lenzin C., Stuessi C., Boutellier U., Respiratory muscle training increases cycling endurance without affecting cardiovascular responses to exercise. *Eur J Appl Physiol*, 2001, 85 (3–4), 233–239, doi: 10.1007/s004210100450.
13. Romer L.M., McConnell A.K., Jones D.A., Effects of inspiratory muscle training on time-trial performance in trained cyclists. *J Sport Sci*, 2002, 20 (7), 547–590, doi: 10.1080/026404102760000053.
14. Caine M.P., McConnell A.K., Development and evaluation of a pressure threshold inspiratory muscle trainer for use in the context of sports performance. *Sports Eng*, 2000, 3, 149–159.
15. Romer L.M., McConnell A.K., Jones D.A., Inspiratory muscle fatigue in trained cyclists: effects of inspiratory muscle training. *Med Sci Sport Exerc*, 2002, 34 (5), 785–792.
16. Zatoń M., Hebisz P., Hebisz R., Respiratory changes resulting from training with enlarged respiratory dead space [in Polish]. *Sport Wyczynowy*, 2008, 4–6, 28–38.
17. Yano T., Horiuchi M., Yunoki T., Ogata H., Kinetics of CO_2 excessive expiration in constant-load exercise. *J Sports Med Phys Fitness*, 2002, 42 (2), 152–157.
18. Laursen P.B., Blanchard M.A., Jenkins D.G., Acute high-intensity interval training improves T_{vent} and peak power output in highly trained males. *Can J Appl Physiol*, 2002, 27 (4), 336–348, doi: 10.1139/h02-019.
19. Anderson D.E., McNeely J.D., Windham B.G., Device-guided slow-breathing effects on end-tidal CO_2 and heart-rate variability. *Psychol Health Med*, 2009, 14 (6), 667–679, doi: 10.1080/13548500903322791.
20. Carey D., Pliego G., Raymond R., How endurance athletes breathe during incremental exercise to fatigue: interaction of tidal volume and frequency. *J Exerc Physiol*, 2008, 11 (4), 44–51.
21. Zhao L., Lu J.B., Yang S.Q., Zhu L.H., Effect of dead space loading on ventilation, respiratory muscle and exercise performance in chronic obstructive pulmonary disease. *Chin J Tuberc Respir Dis*, 2004, 27 (11), 748–751.
22. Crosby A., Talbot N.P., Balanos G.M., Donoghue S., Fatemian M., Robbins P.A., Respiratory effects in humans of a 5-day elevation of end-tidal PCO2 by 8 Torr. *J Appl Physiol*, 2003, 95 (5), 1947–1954, doi: 10.1152/japplphysiol.00548.2003.
23. Toklu A.S., Kayserilioglu A., Unal M., Ozer S., Aktas S., Ventilatory and metabolic response to rebreathing the expired air in the snorkel. *Int J Sports Med*, 2003, 24 (3), 162–165, doi: 10.1055/s-2003-39084.

24. Sidney D.A., Poon C.S., Ventilatory responses to dead space and CO_2 breathing under inspiratory resistive load. *J Appl Physiol*, 1995, 78 (2), 555–561.
25. Mercier J., Ramonatxo M., Prefaut C., Breathing pattern and ventilatory response to CO_2 during exercise. *Int J SportsMed*, 1992, 13(1), 1–5, doi: 10.1055/s-2007-1021225.
26. Yano T., Matsura R., Arimistu T., Yamanaka R., Lian C.S., Yunoki T. et al., Ventilation and blood lactate levels after recovery from single and multiple sprint exercise. *Biol Sport*, 2011, 28 (4), 233–237.
27. Ursino M., Magosso E., Avanzolini G., An integrated model of the human ventilator control system: the response to hypercapnia. *Clin Physiol*, 2001, 21 (4), 447–464, doi: 10.1046/j.1365-2281.2001.00349.x.
28. Duffin J., Mahamed S., Adaptation in the respiratory control system. *Can J Physiol Pharmacol*, 2003, 81 (8), 765–773.
29. Sheel A.W., MacNutt M.J., Control of ventilation in humans following intermittent hypoxia. *Appl Physiol Nutr Metab*, 2008, 33 (3), 573–581, doi: 10.1139/H08-008.
30. Zatoń M., Smołka Ł., Circulatory and respiratory response to exercise with added respiratory dead space. *Hum Mov*, 2011, 12 (1), 88–94, doi: 10.2478/v10038-011-0007-9.

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