IMPACT OF TRAINING WITH ADDITIONAL RESPIRATORY DEAD SPACE ON SPIROMETRY AND EXERCISE RESPIRATORY PATTERN IN CYCLISTS

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Abstract. The aim of our 10-week-long experiment was to investigate the impact of training with additional dead space (DS) on spirometry and exercise respiration. Respiratory muscle training is applied to the development exercise capacity. Twenty cyclists were assigned to two groups: the experimental (E) and the control (C). All of them carried on with their initial training programme. During endurance trainings (twice per week) group E used additional DS (1000 cm^3). Immediatelly before and after the experiment each participant was submitted to a spirometry and a continuous test. The spirometry test measured peak inspiratory (PIF) and expiratory (PEF) flows, forced expiratory volume in one second (FEV1) and forced vital capacity (FVC). The continuous test measured tidal volume (TV), respiratory frequency (RF), along with inspiration and expiration times. Our experiment demonstrated TV increase and RF decrease in both groups. In addition, the TV value was significantly higher in group E than in C. The PIF value also increased significantly, PEF and FEV1 upward trend was observed in group E only. We concluded the additional respiratory DS used in the experimental group provoked an increase of airflow observed in the spirometry tests at rest and during intensive aerobic exercise.

Key words: respiratory muscle efficiency, dead space, training

Introduction

In response to exercise, ventilation is augmented through an acceleration of respiratory frequency and an increase of tidal volume, which results from the changes in the end inspiratory lung volume, end expiratory lung volume, respiratory cycle time and airflow velocity in the pulmonary airway (Sheel 2002). During the progressive load exercise performed by endurance sports athletes, in some cases, the increase of load above 60–70% of maximal oxygen uptake is accompanied by a plateau phase or a tidal volume (TV) decrease. Further increase of pulmonary ventilation is obtained through an acceleration of respiratory frequency (Carey et al. 2008; Sheel 2002). An opposite pattern of increased TV and respiratory frequency (RF) can be observed in some top level athletes (Lucia et al. 1999). This can alleviate or prevent hypoxemia in areas of fast flowing blood in athletes with high cardiac output (Carey et al. 2008). On the other hand, the growing elastic resistance of lungs and thoracic wall must be

overcome and the increase of TV above the individual critical value entails an intensive internal respiratory work (Carey et al. 2008). Therefore it is difficult to point out unequivocally which modifications of the breathing pattern are beneficial during intensive exercise.

An extension of respiratory dead space may cause an increase of friction inelastic resistance in the airways. As the inspiratory resistance increases, the excitability of the receptors sensitive to pulmonary inflation decreases along with their inhibitory impact on the respiratory center, which leads to an extension of the inspiratory phase (Aleksandrova and Breslav 2009). It has also been observed that a greater respiratory resistance goes along with functional changes in the smooth muscles of the upper pulmonary airway. The airway patency is preserved by the Hering-Bruer reflex (Aleksandrova and Breslav 2009). Moreover, greater respiratory resistance stimulates diaphragmatic nerve activity and excitation of the diaphragmatic muscle. A prolonged respiratory restriction may reduce the activity of the muscle, which, in turn, may lead to hypoxia and hypercapnia (Aleksandrova and Breslav 2009). Similarly, in the initial stage of the obstructive lung disease, inspiratory capacity is reduced and functional residual capacity is augmented (Baarends et al. 1998).

Previous research carried out in our laboratory demonstrated that an extension of respiratory dead space (600–1,400 ml) during exercise is accompanied by an increase of TV, RF, pulmonary ventilation (VE) developing hypercapnia (Zatoń and Smołka 2011). It remains unclear whether increased respiratory activity in the training process actually impacts the breathing pattern during unrestrained exercise and spirometry trial results. The findings of research on resistance respiratory muscle training point to an increase, during maximal aerobic training, of such parameters as TV (Amonette and Dupler 2002; Romer et al. 2002a), maximal inspiratory pressure (Gething et al. 2004; Riganas et al. 2008; Sonetti et al. 2001; Volianitis et al. 2001), maximal inspiratory flow rate (Romer et al. 2002a), the amount of work performed in exercise tests (Gething et al. 2004; Bailey et al. 2010). They also show that the sensation of shortness of breath during exercise is less acute (Romer et al. 2002a), the accumulation of respiratory muscle fatigue resulting from exertion is slowed (Romer et al. 2002b), while lactate concentration is lower and post sprint-exercise restitution is faster (Romer et al. 2002c). Such changes may result from training with additional respiratory dead space.

The objective of our research was to assess the impact of training with increased respiratory dead space on exercise breathing patterns and spirometry results.

We assumed that such training would provoke an increase of tidal volume during intensive aerobic exercise and an improvement of inspiratory and expiratory spirometry parameters.

Methods

The study was approved by the Ethics Committee of University School of Physical Education in Wroclaw and was carried out in accordance with the Declaration of Helsinki. Subjects gave written informed consent before participating.

A group of 20 road cyclists (men, members of the Polish National Team) following a homogeneous training program participated in our research. The participants were divided up in two groups of 10: the experimental group (E) and the control group (C). Table 1 presents basic variables characterizing both groups before the experiment was launched, and shows that there were no statistically significant differences between them.

Table 1. Arithmetical mean and standard deviation of basic parameters in groups E and C before the start of the experiment

Group	Value	Age [years]	Height [cm]	Body mass [kg]	$\dot{V}O_2$max [ml/min/kg]	Work [kJ]
E	mean ± sd	16.5 ±0.5	180.6 ±3.8	70.4 ±5.4	64.4 ±4.6	285.4 ±41.8
C	mean ± sd	17.1 ±0.7	180.2 ±4.4	69.4 ±7.2	64.2 ±4.7	274.9 ±30.9

$\dot{V}O_2$max – maximal oxygen uptake, Work – total work performed in progressive test, mean – arithmetic mean, sd – standard deviation, E – experimental group, C– control group.

The experiment was conducted over a period of 10 weeks. Athletes continued with their initial training program based on aerobic exercise; each training session lasted from 180 to 210 min. The program included high volume training, continuous with moderate intensity, and interval training, with intensity above the anaerobic threshold. Heart frequency (measured by Polar S810 sport testers) was used to monitor intensity. The only innovation was the use, in group E, of devices increasing respiratory dead space. A face mask with a tube of an overall volume of 1,000 cm^3 (Φ = 29) was used to force subjects to inhale atmospheric air mixed with their own expired air remaining in the mask and tube. The device was used twice every week in continuous high volume training. Group C performed the same work but with no breathing restriction.

Each subject underwent two physiological exercise tests conducted immediately before and after the experiment at the Exercise Testing Laboratory of Wroclaw's University School of Physical Education (PN – EN ISO 9001:2001 Certificate). Additional respiratory dead space was not resorted to during those tests.

Participants underwent: a progressive test on a cycle-ergometer designed to assess their aerobic capacity, and a spirometry test. The continuous test, with a constant load of 85% of the maximal aerobic power reached in the progressive test, was carried out one week later.

The progressive test was performed on an Excalibur Sport cycle-ergometer (Lode BV, Groningen, the Netherlands) calibrated, in accordance with manufacturer recommendations, before the start of the tests. The exercise began with a workload of 50 W, increased every 3 minutes by an additional 50 W, until the subject was unable to continue. The cycle-ergometer was controlled by a computer which recorded instant power, work time, rotation frequency and calculated total work. The measurement of respiratory parameters began 2 minutes prior to the exercise session and ended 5 minutes after it. The subject breathed through the mask and tidal air was analyzed by a Quark analyzer (Cosmed, Milan, Italy). The device was calibrated with atmospheric air and the following gas mixture: carbon dioxide (CO_2) – 5%, oxygen (O_2) – 16% and nitrogen (N_2) – 79%. Oxygen uptake was measured to assess the subjects' aerobic capacity. Moreover on the basis of oxygen uptake (VO_2) and carbon dioxide excretion (VCO_2), the anaerobic threshold of metabolism (VT2), was determined. A V-slope method was used in accordance with the guidelines of its creator (Beaver et al. 1986). This assumes an increase VCO_2 in relation to VO_2 at the moment when the production of lactic acid, via glycolysis, increases.

The spirometry test consisted of a maximal inspiration preceded by 2–3 casual breaths and followed by a forced expiration of a maximum air flow, resulting in a minimum volume of residual air. The test was conducted with a Quark spirometer. The following parameters were measured: peak inspiratory flow (PIF), peak expiratory flow (PEF), forced vital capacity (FVC), forced expiratory volume in one second (FEV1). Each subject performed three trials; the one with the best FEV1 value was selected for further analysis. The first trial was considered preliminary.

The continuous exercise test (continuous test) was carried out on an Excalibur Sport cycle-ergometer. The exercise session was preceded by a 10-minute warm-up. The warm-up load was calculated to maintain the heart frequency under the anaerobic threshold. The proper exercise was performed with a constant load of 85% of

the maximal aerobic power reached in the progressive test. Both pre and post experimental trials were conducted with loads calculated on the basis of the initial progressive test. No specific rotation frequency was imposed. The program in control of the cycle-ergometer imposed constant power by modulating resistance in reaction to pedaling cadence changes. The measurement of respiratory parameters began 2 minutes prior to the exercise and ended 5 minutes after it. The subject breathed through the mask and tidal air was analyzed by a Quark analyzer. Respiratory frequency, tidal volume, inspiratory time and expiratory time were measured.

Data calculation and processing

The measurement of the respiratory parameters (respiratory frequency, tidal volume, inspiratory time and expiratory time) was conducted breath by breath. In data analysis results were averaged every 60 seconds.

The load in the continuous test was 85% of the maximal aerobic power (individual for every athlete) reached in the progressive test carried out prior to the experiment. Maximal aerobic power was identified as the maximal load in the progressive test at which the subject continued exercising for at least 90 seconds.

A STATISTICA 9 programme was used to develop statistical data. The arithmetic mean and standard deviation were calculated. The ANOVA variant analysis was used for repeated measurements and the post hoc Duncan test to identify significant differences between groups and before vs. after the experiment. A level of statistically significant $p < 0.05$ was adopted.

Results

In the continuous test tidal volume increased in both groups following the experiment. The changes were greater in group E, where in the 2nd and 10th minute of the continuous test TV increase exceeded 0.5 l compared to the results obtained in trials prior to the test (Figure 1). Following the experiment, the TV value was significantly higher in group E than in group C (except in the 9th minute of the exercise). RF decreased in both groups and the significance level was reached from the 3rd minute in group C and from the 4th minute in group E (Figure 2). Inspiratory time increased in both groups; the significance level was reached from the 4th minute in group E and from the 3rd minute in group C (Figure 3). Expiratory time in both groups increased below the significance level starting from the 3rd minute (Figure 4).

The spirometry test showed a significant PIF increase, in group E only. No significant changes were noticed in expiratory spirometry parameters after the experiment. However, an upward trend in FEV1 and PEF appeared in group E (Table 2).

Table 2. Results from the spirometry test (forced expiratory volume in one second, peak expiratory flow, forced vital capacity, peak inspiratory flow), before and after experiment, in E and C group

Measured parameters	Group E, before	Group E, after	Group C, before	Group C, after
	mean ± sd	mean ± sd	mean ± sd	mean ± sd
FEV1 [l]	6.43 ±1.65	7.00 ±1.86	6.98 ±1.29	6.65 ±1.90
PEF [l/s]	10.12 ±1.46	12.17 ±2.13	11.09 ±1.86	11.3 ±2.41
FVC [l]	8.27 ±1.9	9.25 ±2.65	8.91 ±1.31	8.93 ±2.71
PIF [l/s]	2.24 ±0.64	2.81 ±0.58*	2.57 ±0.67	1.61 ±0.96**^

Means – arithmetic mean, sd – standard deviation, FEV1 – forced expiratory volume in one second, PEF – peak expiratory flow, FVC – forced vital capacity, PIF – peak inspiratory flow, * – $p < 0.05$ after vs. before, ** – $p < 0.005$ after vs. before, ^ – $p < 0.05$ E vs. C.

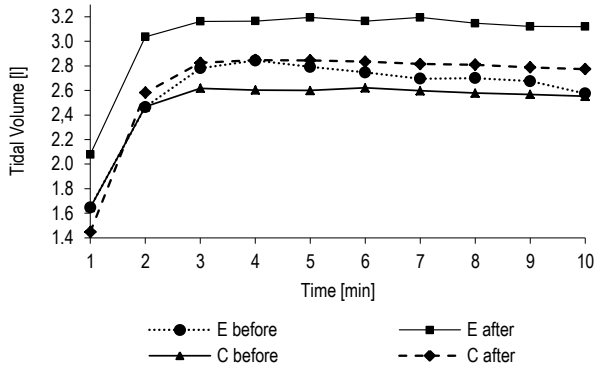

Figure 1. Tidal volume [l] during continuous test, before and after experiment, in E and C group

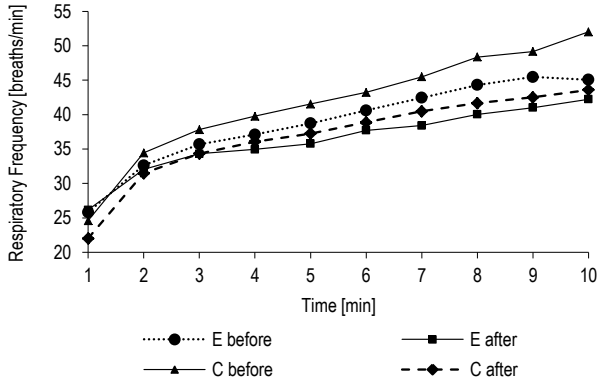

Figure 2. Respiratory frequency [breaths/min] during continuous test, before and after experiment, in E and C group

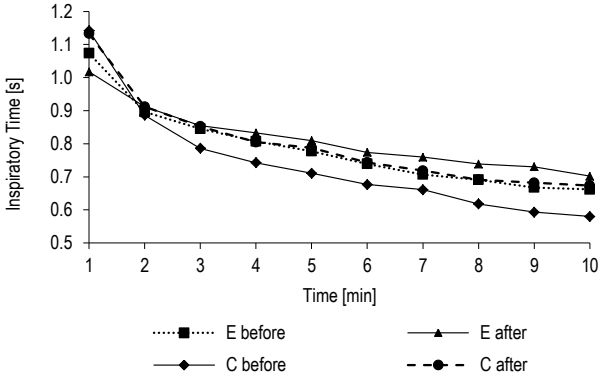

Figure 3. Inspiratory time [s] during continuous test, before and after experiment, in E and C group

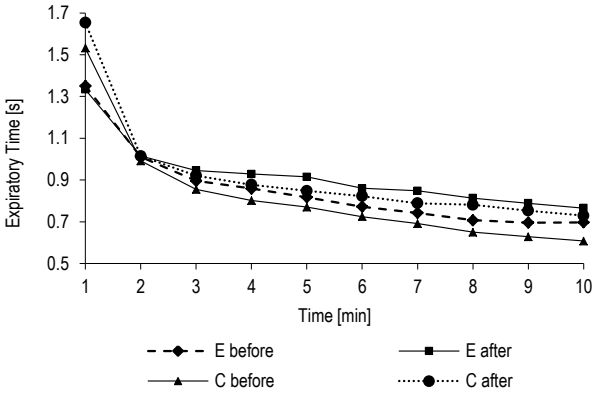

Figure 4. Expiratory time [s] during continuous test, before and after experiment, in E and C group

Discussion

An increased pulmonary ventilation during exercise requires an extra contribution of the respiratory muscles. Consequently, a prolonged work may result in the fatigue of the corresponding muscle groups and lead to reduced efficiency (Eastwood et al. 2001; Nadiv et al. 2012; Passfield et al. 2005). Nadiv et al. (2012) demonstrated that at low exercise intensity levels (walking at 8 km/h) the respiratory muscles of well-trained young men show no signs of fatigue, however, an additional load (a 15 kg backpack) provokes a significant rise of such electromyographic parameters as root mean square and mean power of the intercostal external muscles. Also Taylor and Romer (2008) established that intensive respiratory work impedes the exercise capacity of the limb muscles. Respiratory muscle training is thus implemented to improve respiratory efficiency. There are two main types of respiratory muscle training: one resorting to inspiratory and expiratory airflow resistance and the other, consisting in unrestrained hyperventilation (Gething et al. 2004; Markov et al. 2001; Passfield et al. 2005; Tong et al. 2008). In our research, we used resistance. It amounted to an extension of respiratory dead space by 1,000 ml (Φ = 29), which resulted in an increase of inelastic respiratory resistance.

The results point to the increase of TV during intensive exercise (85% of the maximal aerobic power) as a result of training with additional respiratory dead space. When the values of this parameter are high, the growing elastic resistance of lungs and thoracic wall must be overcome and the increase of TV above the individual critical value entails an intensive internal respiratory work (Carey et al. 2008). It is then difficult to determine whether the change put forth by the present research is beneficial in cycling. It must be underlined however that the diaphragm muscle is resistant to fatigue (Sheel 2002). In addition, our previous research demonstrated a higher work time value in progressive test and, in exercises of submaximal intensity, lower oxygen uptake values were observed, pointing to a lower energetic expenditure of the work performed (Zatoń et al. 2008a; Zatoń et al. 2008b). Therefore, the TV increase probably does not reduce tolerance to intensive aerobic exercise. Other authors have observed similar changes resulting from respiratory muscle training. An increase in the work performed in continuous exercises with constant load was demonstrated by Gething et al. (2004), and by Markov et al. (2001). The increase in the time of aerobic work was accompanied by a decline in metabolite (urea, ammonia and lactate) concentration (Tong et al. 2008) as well as lower heart frequency and pulmonary ventilation (Gething et al. 2004). These changes

are accounted for by improvements in respiratory efficiency and blood redistribution (Amonette and Dupler 2002; Dempsey 2006; Sheel 2002).

Improved TV and the lack of significant changes in inspiratory and expiratory times, point to an increase of the airflow volume in a given time unit. Similar changes were observed at rest for PIF and, below statistical significance level, PEF and FEV1. It may result from a greater pressure difference between the air in pulmonary alveolus and atmospheric air, in both the inspiratory and the expiratory phase (Aleksandrova and Breslav 2009), which could point to an increase in the respiratory muscle tension. Greater values of airflow volume may well be generated by a modification of smooth muscle tension around the airways. And breathing obstacles trigger the Hering-Breuer reflex (Aleksandrova and Breslav 2009). But the pattern of training-induced durable changes remains unclear. Perhaps, regular training sessions with additional respiratory dead space modify the activity of the slow adapting receptors. Further research must comprise inspiratory and expiratory pressure measurement to determine whether the observed changes result from an increase of the force of the respiratory muscles. Hypercapnia, which is caused by exercise with additional respiratory dead space (Zatoń and Smołka 2011), may constitute yet another pattern. Greater carbon dioxide partial pressure (pCO_2) in artery blood irritates the chemoreceptors of the circulatory system and causes higher VE, mainly through an increase of TV (Toklu et al. 2003; Ursino et al. 2001; Zhao et al. 2004). Such an intensification of the respiratory work may improve respiratory muscle efficiency. Regular hypercapnia may also modify the responsiveness of the chemoreceptive areas and, in turn, change the respiratory pattern.

Research conducted on non-athletes indicates that resistance training of the respiratory muscles causes improved PEF and FEV1 (Sutbeyaz et al. 2010; Yamashita and Kakizaki 2011). But the results obtained by Amonette and Dupler (2002) showed no spirometry changes in triathlon and marathon athletes, in spite of TV increase and RF decrease during maximal aerobic training. Likewise, no spirometry changes were identified in basketball players by Goosey-Tolfrey et al. (2010), despite an increase in maximal inspiratory and expiratory pressures. Our research indicates a training-induced increase of PIF as well as, statistically insignificant, PEF and FEV1 in cyclists, resulting from additional respiratory dead space. The divergences between our work and other research results may lie in stimulus intensity. Our research project comprised two weekly 3.5-hour respiratory resistance training sessions, while in Amonette and Dupler (2002), only two daily series of 30 inspirations/expirations executed with additional resistance are described.

Conclusion

Training with additional respiratory dead space brought about changes in the exercise breathing pattern, predominantly through an increase of TV. The spirometry trial demonstrated an improvement of inspiration and forced expiration airflows. The mechanism governing those changes requires further research. We presume that the modifications we observed may result from greater respiratory muscle force, changes in smooth muscle tension around the airways, changes in chemoreceptor responsiveness.

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