Relationship Between Airway Resistance indices and Maximal Oxygen Uptake in Young Adults

by

Roholah Fatemi¹, Mohsen Ghanbarzadeh²

The present study aims at assessing the relationship between airway resistance indexes (FEV₁, FVC and FEV₁/FVC) and maximal oxygen uptake (VO₂max) in young adults. Subjects of the study included 50 healthy males (age, 22.1 ± 2.47 years; FEV₁, 3.41 ± .66 liter; FVC, 3.96 ± .56 liter; VO₂max, 38.83 ± 9.83 ml.kg⁻¹.min⁻¹) studying at Shahid Chamran University of Ahvaz. After determining subjects’ volumes of FEV₁, FVC and FEV₁/FVC by digital spirometer, maximal oxygen uptake was measured. The study protocol measured VO₂max levels using the sub-maximal Astrand–Ryming test on the ergometer cycle. The data were analyzed through descriptive and inferential statistics. Results revealed a significant correlation among the three independent variables of FEV₁, FVC, FEV₁/FVC and projected VO₂max values. Based on the results, it can be concluded that these parameters have a close interaction with higher VO₂max levels, and therefore, having a lower airway resistance seems beneficial.

Key words: Maximal Oxygen Uptake, Airway Resistance, FEV₁, FVC

Introduction

Physical fitness is required not only by athletes for better performance but also by non-athletes for maintenance of physical and mental health. A Buffalo health study concluded that pulmonary function is the long-term predictor for overall survival rates in both genders, and could be used as a tool in general health assessment. (Prajapati et al. 2008).

The condition of asthma is characterized by some degree of airway inflammation, and high rates of ventilation result in drying mucosal airway cells, which can lead to increases in bronchoconstrictor mediator release (Beck 1999). Measurements of maximal O₂ uptake (VO₂max), which is defined as reaching a plateau of VO₂ despite further increases in work load, reflects the upper limit of body O₂ uptake (VO₂) and utilization during muscular exercise, which is traditionally considered the criterion for measuring cardiovascular fitness (Oguz 2002).

Exercise-induced bronchospasm (EIB) is defined as a decrease in lung function, usually characterized by a 10% decrement in forced expiratory volume in 1 s (FEV₁), or a decrease of 20% in mid-forced expiratory flow (FEF25%–75%), which occurs after vigorous exercise. EIB can be triggered by intense exercise, cold dry environments, chronic asthma, or by a variety of air pollutants (Wagner 1998). Histamine is one inflammatory mediator that has been shown to increase during heavy exercise, which could cause bronchoconstriction at the level of the small airways.
and/or increase microvascular permeability (Anselme et al 1994).

Bronchospasm caused by exercise results in increased airway resistance, which could increase the ventilation-perfusion ratio (VA/Q) in the lung and may lead to a drop in partial pressure of arterial oxygen content (PaO2) (Alex 2006). Airway inflammation and narrowing, leading to abnormal ventilation distribution, could contribute to the widened alveolar-arterial oxygen difference (A-aDO2) seen during incremental exercise. Furthermore, Exercise-Induced Arterial hypoxemia (EIAH) begins to occur even in sub-maximal exercise in some subjects, and often worsens as work rate is further increased (Wetter et al. 2001).

In addition, increases in peripheral airway resistance could constrain ventilation and contribute to the inadequate alveolar hyperventilation seen in subjects with EIAH (Thomas et al. 2001). Exercise-induced bronchospasm due to maximal physical activities, as well as inhalation of cold and dry air, can not be differentiated from asthma. It is known that one of the most common predisposing factors for an asthmatic attack is exercise. Sometimes the only cause of asthmatic symptoms in mild asthma is exercise. Other studies revealed the prevalence of exercise-induced bronchospasm was 90% in asthmatics and 40% in allergic rhinitis patients (Ehteshami- Afshar et al. 2002).

Dempsey (2006) states “a carefully selected combination of increased frequency and tidal volume must be achieved, taking into account the need to minimize dead space ventilation (i.e., the increase in breathing frequency should not be excessive)” (Faintuch et al. 2004).

Due to the fact that scholars hold divergent ideas about the relationship among pulmonary function, aerobic capacity and exercise, further research seems to be indispensable. It is, thereby, our intention to achieve more information on pulmonary function. The purpose of this study is to determine the relationship between airway resistance and VO2max in young adults.

Methods

Subjects

All 120 subjects, whose participation was voluntary, were young (age 19-24) healthy, nonsmokers studying in the faculty of physical education at Shahid Chamran University of Ahvaz in Iran.

Before the administration of the tests, subjects completed health and physical activity level forms. Afterwards, we selected 50 subjects whose scores were in the same range (n = 50); had a mean (± SD) age of 22.1±2.4 years, a mean body height of 174.4±6.3 cm, mean body mass of 72.4±9.7 kg and a mean VO2max of 38.8±9.8 ml.kg⁻¹.min⁻¹. All subjects completed an informed consent form before participating and completing in this study.

Preliminary procedures

Tests were performed in the laboratory, maintained at 24 - 26 °C, between 10 a.m. and 12 noon. All the participants had breakfast at 7 a.m. Subjects had no history of any major diseases and were not participating in any physical training program or taking any medications. All were informed about the purpose, requirements and the experimental protocol of the investigation. Experimental procedures were demonstrated to allay their apprehension. Body height and weight of the subjects were measured with the help of a height measuring stand and weighing machine (Krups company manufactured by Dr. Beli Ram and sons-New Delhi). Body mass was measured to an accuracy of ± 0.25 kg and body height to an accuracy of ± 0.5 cm. Airway resistance indexes (FEV1, FVC and FEV1/FVC) were measured by digital spirometer (HI-601made in Japan). To measure FVC, each subject blew into the instrument with maximum force after full inspiration; three readings were taken and the best was recorded. To determine VO2max, we selected the sub-maximal Astrand-Ryming protocol on a Monark cycle. Duration of this test was approximately 6 minutes based on individual heart rate responses. The initial load was 98.1 watts. In the first 2 minutes, where the heart rate was less than 60% of predicted HRmax, the load increased 49 watts per 2 minute; whereas if the heart rate was 60-70% HRmax, the load increased 24.5 watts per 2 minutes. If heart rate rose above 70% HRmax, the test continued without load change until the heart rate reached steady state, at which point the test was terminated.

Results

All enrolled subjects agreed to participate and successfully performed in this series. There was no exclusion or complication in this series. In this study, in
order to highlight the relation between pulmonary function and maximum oxygen uptake, we decided to select subjects with wide ranges of VO2max (min, 27.52 ml.kg⁻¹.min⁻¹; max, 55 ml.kg⁻¹.min⁻¹). Results are shown as mean ± SD. Table 1 summarizes the characteristics of the study group. Correlations between individual pulmonary function indexes (FEV1, FVC and FEV1/FVC) and maximal oxygen uptake (VO2max) are shown in Table 2.

The results show that there is a significant correlation between FEV1 and VO2max (r = 692*, (p < 0.001). There is significant relationship between FVC and VO2max (r = 512*, (p < 0.001). Our study also confirmed a strong correlation between FEV1/FVC and VO2max (r = 621*), (p < 0.001). The increase in VO2max correlated best with the increase in FEV1 and to a lesser degree with the increase in FVC.

**Discussion**

The dynamic factors of pulmonary function depend on different variables such as age, physical activity level, environments pollutants, body composition and prolong disease of the lungs (Prajapati 2008, Game 2006, Faintuch 2004, Gökoğlu 2007). The most salient finding of our study is the direct correlation between airway resistance parameters and VO2max, as this relation with FEV1 is markedly noted, however, although not as pronounced as with FEV1, the correlation between FVC and FEV1/FVC ratio is high. Researchers should caution in making immediate conclusions, for such variables as smaller relative lung sizes would inevitably alter statistical analyses, for such subjects would have smaller airways and blood vessels which could accentuate any regional homogeneities differences in the distribution of air and blood flow (Wagner 1998). Evidence clearly supports the importance of exchanging high volumes of air (VE) to maintain PaO2 and hemoglobin saturation (Martin et al. 1979).

It has been reported that endurance athletes may have a lower ventilatory equivalent for CO2 (VE/VCO2) than non-athletes. It is hypothesized that this desensitization to CO2 and its ability to increase VE in athletes may be an adaptation to training and confers an advantage to the athlete in performance (Martin et al. 1979).

It is contended that, like metabolic requirements, mechanical limitations of ventilatory muscles are reached only in highly fit athletes [9], and during intensive training, athletes encounter a decline in PaO2 due to decreased transit time of red blood cells through the pulmonary arterials (Gökoğlu 2007, Martin et al. 1979). In contrast, some studies have shown that the decrement in endurance due to added airway resistance will be small at high and low work-rates, and will be maximal at some intermediate work-rates. The small added airway resistance commonly found in equipment for making various respiratory measurements during exercise can cause a decrement in endurance in proportion to the size of the resistance (Craig 1974). An increase in aerobic enzymes during exercise in humans would equate to more efficient energy utilization of the respiratory muscles and lower fatigueability. Like all other skeletal muscles, the pulmonary muscles also require oxygen when for aerobic metabolism. The fatigue resistance of this process is related to the training status of the muscle. If the muscle is more endurance trained, then it will be less likely to constrain ventilation and exercise performance (Amonette 2002).

This condition seems primarily due to an excessive alveolar-to-arterial O2 difference (A-aDO2) and inadequate compensatory hyperventilation, but also to increasing temperature and metabolic acidosis, which cause a rightward shift in the Hb-O2 dissociation curve (Dempsey 1999).

### Table 1

<table>
<thead>
<tr>
<th>Subject characteristics and pulmonary function</th>
<th>mean ± SD</th>
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<tbody>
<tr>
<td>Age, yr</td>
<td>22.1± 2.47</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>72.48± 9.72</td>
</tr>
<tr>
<td>Height, cm</td>
<td>174.44± 6.38</td>
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<tr>
<td>FEV1, liters</td>
<td>3.41± 0.66</td>
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<tr>
<td>FEV1, % predicted</td>
<td>87.66± 8.78</td>
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<tr>
<td>FVC, liters</td>
<td>3.96 ± 0.56</td>
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<tr>
<td>FVC, % predicted</td>
<td>75.28± 8.33</td>
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<tr>
<td>FEV1/FVC, liters</td>
<td>0.84± 5.91</td>
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<tr>
<td>VO2max, ml.kg⁻¹.min⁻¹</td>
<td>38.83± 9.80</td>
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### Table 2

<table>
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<tr>
<th>Correlation between FEV1, FVC, FEV1/FVC and VO2max.</th>
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<tbody>
<tr>
<td>Variables</td>
<td>FEV1</td>
</tr>
<tr>
<td>VO2max</td>
<td>692*</td>
</tr>
</tbody>
</table>

*<p <0.001

FVC=Forced vital capacity; FEV1=Forced expiratory volume in one second; VO2max =Maximal oxygen consumption
The fall in PaO$_2$ that occurred in the rest-to-exercise transition accounted for the majority of the decreased SaO$_2$ early in the exercise bout (Wetter et al. 2001). However, this condition may result in diffusion transposition of oxygen from alveoli to arterials, and subsequently changing the VO$_2$max. Another finding in this study is the significant correlation between FEV1/FVC ratio and VO$_2$max in young subjects. The higher FEV1 (rather than FVC) volume in subjects with correspondingly higher rates of VO$_2$max provides support for this proposal.

Although increased airway resistance caused a decrement in VO$_2$ and peak aerobic capacity of exercise, this condition had no affect on anaerobic capacity (Oguz 2002). Tone states that any increase in VO$_2$max, due to increases in FEV1 volume, showed that pulmonary function variables predict 30 percent of the variance of peak exercise capacity (Long 1999).

Jackson (1996) found an expiration limitation during exercise with moderate and high intensity in elite male and female athletes and concluded three factors potentially limiting aerobic capacity: diffusion capacity, ventilatory muscle power and limitation in expiration flow.

In spite of divergent scholarly views, it had been documented that higher FEV1/FVC ratios equates to lower airway resistance and better pulmonary function. Furthermore, such a condition may result in differences in aerobic capacity for endurance athletes, which gives rise to inherent advantages for these athletes (Rundell 2002, Unal and et al.2004, Voy 1984).

Moorcroft and colleagues (1997) reported a decline in absolute and predicted values of FEV1 in adult patients with cystic fibrosis (CF), not associated with aerobic capacity.

Other results suggest that deterioration in lung function in children with CF might also point to a significant decrease in peak exercise capacity (Klijn et al. 2003). A direct relation between the decrease in maximum aerobic capacity and FVC has also been reported. It has also been observed that physical conditioning improves the FCV and inspiratory capacity (IC) of patients, and positively influences the maximum aerobic capacity with improvement (Ghanbarzadeh 2009).

In our study, the increase in absolute VO$_2$max was mainly related to an increase in FEV1 and FVC.

In contrast, the correlations of VO$_2$max with these three variables were of borderline significance.

Significance. The studies in which this relationship was examined have indicated that only 30 to 60% of the variability in exercise tolerance among COPD patients can be accounted for by the reduction in FEV1. FEV1 was not predictive of either maximal (VO$_2$ peak) or sub-maximal (MRT) aerobic capacity (Palange 1998).

Besides, it seems that pulmonary response in inactive subjects occurs at a larger scale than in trained athletes (Uçok et al. 2004). Pulmonary functional adaptations and prolonged training conditions may be possible mechanisms that attributed to these conditions.

It is clear that exercise training inducement influences pulmonary function, as well as VO2max (Ehteshami- Afshar 2002, Wetter 2001, Klijn 2003, Giacomoni 1999). Therefore, it can be concluded that these parameters have close interactions, and subjects with higher VO$_2$max have a lower airway resistance.

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