Respiratory Physiology & Neurobiology 175 (2011) 181–184

Contents lists available at ScienceDirect

Respiratory Physiology & Neurobiology

journal homepage: www.elsevier.com/locate/resphysiol

Short communication

Ventilation-limited exercise capacity in a 59-year-old athlete

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ARTICLE INFO

Article history:
Accepted 11 October 2010

Keywords:
Ventilation
Exercise
Limitation
Airway obstruction
Training

ABSTRACT

Aerobic exercise performance may be limited by the respiratory system in fit elderly adults whose lungs undergo the normal ageing process or who develop lung diseases but can maintain high pulmonary blood flows and ventilatory requirements. Here we describe a 59-year-old athlete demonstrating high aerobic exercise performance limited by his relatively low ventilatory capacity.

The male hobby cyclist (59 years, 176 cm, 83 kg), undergoing cardiopulmonary exercise testing, reported no complaints except a history of dyspnoea and exercise intolerance at high altitude (>2000 m). Lung function testing indicated mild airway obstruction (FEVC: 4.22 l, FEV1: 2.91 l, FEV1/FEVC: 69%) which was not reversed post-bronchodilator. However, FEV1/FEVC was slightly above the 5th percentile and thus not confirming the diagnosis of COPD.

The athlete completed 300 W (3.6 W/kg) and his maximal oxygen uptake was 45 ml/min/kg (156% predicted!). Above 250 W he was unable further increasing minute ventilation, and oxygen pulse and oxygen uptake even decreased. The related changes of the respiratory pattern (increase of breathing frequency, decreases of inspiratory capacity and tidal volume) indicated dynamic lung hyperinflation resulting in cardiac output constraint, arterial oxygen desaturation, severe dyspnoea and exercise limitation.

This case report delineates the pathophysiological situation of ventilation-limited exercise capacity in a well-trained middle-aged subject. However, beneficial adaptations to regular exercise may have helped maintain high aerobic performance without any adverse symptoms during submaximal exercise.

1. Introduction

Aerobic exercise performance in untrained but otherwise healthy individuals is primarily limited by cardiac and skeletal muscle capacities (Hsia, 2001). In contrast, in endurance athletes and patients with pulmonary diseases the respiratory system may dominate exercise limitation (Dempsey et al., 1990; Wasserman et al., 2004). This also applies to older athletes whose lungs undergo the normal ageing process or who develop lung diseases but can maintain high levels of fitness associated with high pulmonary blood flows and ventilatory requirements (Dempsey et al., 1990). Here we describe a 59-year-old athlete demonstrating high aerobic exercise performance limited by his relatively low ventilatory capacity.

2. Methods

A 59-year-old male hobby cyclist (176 cm, 83 kg) underwent cardiopulmonary exercise testing as base for planning his future training. He was life-long non-smoker, had no allergy-history, did not take any medication and reported regular physical activity, mostly cycling of about 10 h/week. He did not report any complaints except dyspnoea and exercise intolerance at high altitude (>2000 m) where ventilatory limitation emerges earlier than at sea level (Burtscher et al., 2009a). Routine pre-test examination including lung function testing (ZAN 300, Germany) indicated mild airway obstruction (FEVC: 4.22 L, 91% predicted; FEV1: 2.91 L, 80% predicted; FEV1/FEVC: 69%) which was not reversed post-bronchodilator (GOLD stage 1: post-bronchodilator FEV1 $\geq$ 80% predicted and FEV1/FVC < 0.7) (Rabe et al., 2007). However, FEV1/FEVC (69%) was slightly above the 5th percentile (67%) using the prediction equation of Falaschetti et al. (2004) and thus not confirming the diagnosis of COPD (Hansen et al., 2007). Resting lung diffusion capacity for carbon monoxide was normal (106% predicted).

2.1. Exercise testing

After a 10 min warm-up period the patient was subjected to a symptom-limited incremental cycle spiro-ergometry (Lode Excalibur Sport, The Netherlands) starting with 50 W and an increment of 50 W per 3 min up to exhaustion. Ventilation and gas exchange were measured by an open spirometric system (Oxycon Alpha,

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doi:10.1016/j.resp.2010.10.003

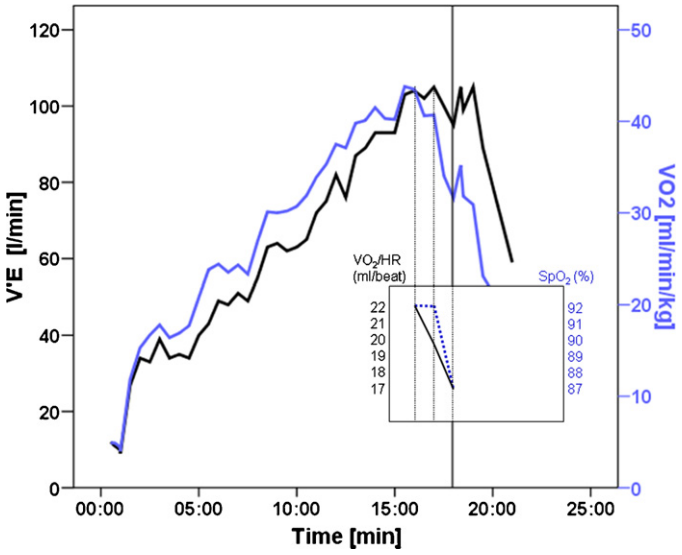

Fig. 1. Minute ventilation (VE) and relative oxygen uptake (VO_2) during the incremental exercise test. The vertical line indicates unloading pedal resistance at the workload of 300 W. Changes of oxygen pulse (VO_2/HR) and oxygen saturation (SpO_2) at maximal work load are shown in the insertion.

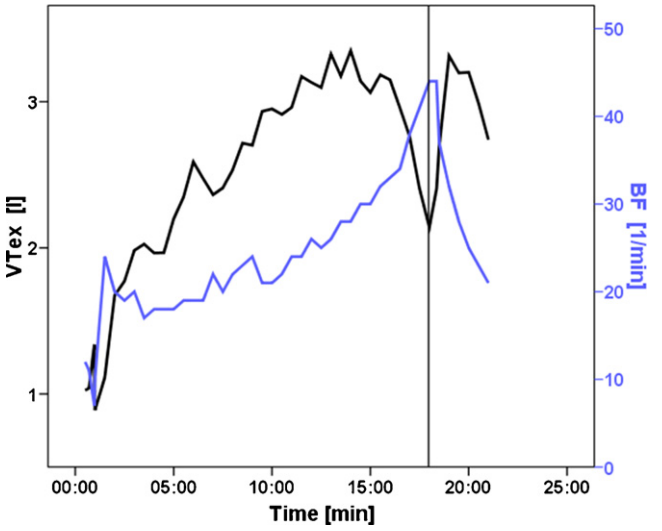

Fig. 2. Expiratory tidal volume (VT_{ex}) and breathing frequency (BF) during the incremental exercise test. The vertical line indicates unloading pedal resistance at the workload of 300 W.

Jaeger, Germany). Inspiratory capacity maneuvers were performed at 3-min intervals, after the first minute at each workload. A 6-lead electrocardiogram (Schiller AT10, Switzerland) and arterial oxygen saturation by finger pulse-oximetry (SpO_2) (Pulsox-7, Minolta, Japan) were recorded continuously. Blood samples for measurements of blood gases (ABL80 FLEX CO-OX, Radiometer, Denmark) and lactate concentrations (Biosen 5040, EKF diagnostic Sales GmbH, Germany) were drawn from a hyperaemised ear lobe at the end of each workload.

An additional CT scan of the lung was performed on a 64-row multi-slice CT scanner (Lightspeed VCT, General Electrics, Milwaukee, USA). Images were acquired using 120 kV and 60 mA, with a pitch of 3 and a gantry rotation time of 0.5 s. Transversal images were reconstructed with a slice thickness of 2.5 mm and 0.625 mm. The 0.625 mm slices were used for coronal reconstruction.

3. Results

The athlete completed 300 W (3.6 W/kg) at a maximal heart rate of 175 bpm and his maximal oxygen uptake ($VO_{2\,max}$) was 45 ml/min/kg (156% predicted; Wasserman et al., 2004). Beside a relatively low ratio of inspiratory time to the duty cycle (T_i/T_{tot}: 39%) the pattern of the cardiopulmonary responses remained normal up to 250 W. At this power output the minute ventilation was 103 l/min with a corresponding tidal volume of 3.2 l (76% FEVC) and a breathing frequency of 32 breaths/min. After increasing the workload to 300 W he was unable further increase minute ventilation and oxygen pulse and VO_2 decreased markedly (Fig. 1) whereas VCO_2 continued to rise. No decrease in heart rate and no abnormal ECG changes were observed. The ratio between maximal minute ventilation and the maximal voluntary ventilation, calculated as 40FEV1, amounted to 89%. The changes of the respiratory pattern at 300 W were characterized by an increasing breathing frequency and decreasing tidal volume (Fig. 2). The inspiratory capacity compared to rest remained unchanged up to 250 W and then decreased by 0.55 l indicating dynamic hyperinflation. The dead space to tidal volume ratio remained below 0.3 throughout the test but might have been slightly underestimated due to the use of arterialized capillary blood for calculation. SpO_2, as measured by pulse oximetry, dropped from 92% to 87% during the workload of 300 W and fell to 85% when derived from blood gas analysis (pH 7.34, PaO_2 53 mmHg, $PaCO_2$ 43 mmHg) at the end of maximal exercise. The blood lactate values increased from 3.45 mmol/l at 250 W to 8.59 mmol/l 2 min after finishing the exercise test. The patient reported extremely high respiratory distress before stopping the exercise test with rapid release after unloading. The ventilatory anaerobic threshold (V-slope method) has been determined at 76% of $VO_{2\,max}$ with a corresponding heart rate of 136 bpm.

The CT scans of the lung showed a slight bronchial wall thickening and a beginning mosaic attenuation pattern. We did not observe any vascular attenuation or emphysema (Fig. 3).

4. Discussion

This case report demonstrates high aerobic capacity in a 59-year-old hobby athlete which is limited by his insufficient maximal minute ventilation and the related breathing distress. This is supported not only by the observed ventilatory plateau but also by the low respiratory reserve and the high sensation of dyspnoea which were even more pronounced when compared to mild COPD patients with low or normal exercise capacity (Burtscher et al., 2009b). When the highest possible minute ventilation was reached, the breathing frequency increased at considerable costs of the tidal volume. These unfavourable changes in breathing pattern (Neder et al., 2003) were associated with decreases in SpO_2, oxygen pulse and oxygen uptake despite unchanging workload. The reduced inspiratory capacity at the highest workload indicates dynamic pulmonary hyperinflation which is the established major limitation to exercise performance in COPD (O'Donnell and Laveneziana, 2006) but may also occur in healthy fit elderly adults (McClaran et al., 1995). Lung hyperinflation causes increased elastic loading and functional weakness of the respiratory muscles and hinders tidal volume expansion accompanied by an increasing central neural drive (O'Donnell and Laveneziana, 2006). Apparently, the related breathing mechanics also adversely affected cardiac function likely by right ventricular dysfunction (Aliverti and Macklem, 2001) and/or blunted venous return (Aliverti et al., 2004; Miller et al., 2005) According to the Fick equation (oxygen uptake = heart rate × stroke volume × arteriovenous oxygen difference) the initial decrease of oxygen pulse and VO_2 at the workload of 300 W can be explained by a reduced stroke volume because SpO_2 and thus probably also arteriovenous oxygen difference initially remained unchanged.