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Blunted blood pressure response during hyperpnoea in endurance runners

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ABSTRACT

The purpose of this study was to elucidate the cardiovascular response during hyperpnoea in endurance-trained runners compared to sedentary controls. Twelve runners and ten sedentary individuals participated in this study. A maximal respiratory endurance test (MRET) was performed as follows: target minute ventilation was initially set at 30% of maximal voluntary ventilation (MVV_{12}) and was increased by 10% MVV_{12} every 3 min. The test was terminated when the subject could no longer maintain the target ventilation. Heart rate and mean arterial blood pressure (MBP) were continuously measured. Respiratory endurance time during the MRET was longer in the runners than the controls. The change in MBP during the MRET was lower in the runners compared to the sedentary controls (runners: 100.2 ± 2.4 mmHg vs. controls: 109.1 ± 3.0 mmHg at 6 min of hyperpnoea). Therefore, the blood pressure response during hyperpnoea is blunted in endurance runners, suggesting that whole-body endurance exercise training attenuates the respiratory muscle-induced metaboreflex.

1. Introduction

High-intensity whole-body exercise induces respiratory muscle fatigue (Johnson et al., 1993; Romer and Polkey, 2008). This respiratory muscle fatigue affects cardiovascular regulation and blood flow distribution during exercise, through the respiratory muscle-induced metaboreflex (Dempsey et al., 2008; Dempsey et al., 2006; Harms et al., 1997; Harms et al., 1998; Romer and Polkey, 2008). This respiratory muscle-induced metaboreflex generates sympathetic vasoconstriction and reduces blood flow (oxygen transport) to active limbs, thereby exacerbating limb fatigue and compromising endurance performance (Dempsey et al., 2008; Harms et al., 1997; McConnell and Lomax, 2006; Sheel et al., 2001). Conversely, attenuating respiratory muscle-induced fatigue may improve endurance performance (HajGhanbari et al., 2013; Illi et al., 2012). Specific training of the inspiratory muscles (respiratory muscle training) can blunt the cardiovascular response to resistive inspiratory muscle activity (Witt et al., 2007), indicating that the respiratory muscle-induced metaboreflex is attenuated by this training. Given this, whole-body endurance training may attenuate the cardiovascular response to increased respiratory muscle work (i.e., blunted respiratory muscle-induced metaboreflex). In a valuable study by Callegaro et al. (Callegaro et al., 2011), vascular resistance to enhanced respiratory muscle work increased in sedentary subjects, but not in runners who experienced considerable hyperpnoea during regular endurance training. These results indicate that whole-body endurance training blunts the inspiratory muscle-induced metaboreflex. However, Callegaro et al. (Callegaro et al., 2011) used breathing against an inspiratory load to increase inspiratory muscle work, which meant that breathing frequency was lower (i.e., high-resistance, low-speed inspiratory muscle contractions) than spontaneous breathing during whole-body exercise. Another way to model the respiratory muscle metaboreflex during whole-body exercise is to measure the cardiovascular response to exercise-mimicking hyperpnoea (low-resistance, high-speed inspiratory and expiratory muscle contractions).

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Respiratory muscle endurance is higher in endurance athletes (Eastwood et al., 2001; Martin and Stager, 1981), and endurance training, including running, improves respiratory muscle endurance (O'Kroy and Coast, 1993; Robinson and Kjeldgaard, 1982). These findings are supported by animal studies, which indicate that endurance exercise training increases oxidative capacity of both inspiratory and expiratory muscles (Gosselin et al., 1992; Powers et al., 1992, 1990; Uribe et al., 1992). On this basis, we hypothesized that endurance athletes have a lower cardiovascular response to low-resistance, high-speed inspiratory and expiratory muscle contractions. To test this hypothesis, the cardiovascular response during voluntary normocapnic incremental hyperpnoea was evaluated in endurance-trained runners and compared to the response in sedentary controls.

2. Methods

2.1. Subjects

Twelve endurance runners and ten sedentary controls, all males, volunteered for this study. The runners belonged to a collegiate track team that had competed in the 2012–2014 Japanese intercollegiate road relay. The sedentary controls had not engaged in exercise training. All subjects were informed about the experimental procedures and potential risks involved, and written consent was obtained. This study was approved by the Human Research Committee of the Research Center of Health, Physical Fitness and Sports, Nagoya University, the Japan Institute Sciences Ethics Committee, and the Ethics Committee for Human Experiments at Ritsumeikan University.

2.2. Experimental procedure

During the preliminary visit, subjects were familiarized with the equipment and practiced the pulmonary function and maximal exercise tests. They reported to the laboratory on three additional occasions. On day 1, an incremental exercise test was performed to determine peak oxygen uptake (VO_{2peak}) using a treadmill and bicycle ergometer for the runners and sedentary controls, respectively. On day 2, pulmonary function tests and diaphragm thickness (T_{di}) measurement were performed. On day 3, a maximal respiratory endurance test (MRET) with cardiovascular measurement was performed.

2.3. Maximal exercise test

The runners first performed at a submaximal level on the treadmill at three different speeds (14, 16, and 18 km h^{-1}; 0% grade) for 4 min separated by 1-min rest periods. The speed was then set at 18 km h^{-1} for 1 min and increased by 1 km h^{-1} every minute up to 20 km h^{-1}. Once this velocity was attained, the gradient was increased 1% every minute until volitional exhaustion (Saunders et al., 2004). Respiratory parameters were determined breath-by-breath using an online system (AE300S, Minato Ikagaku, Osaka, Japan). Heart rate (HR) was continuously measured using a wireless HR monitor (Accurex Plus; Polar Electro Oy, Kempele, Finland). For sedentary controls, the bicycle ergometer (Aerobike 75XL; Combi, Tokyo, Japan) was initially set at 90 W, and the workload was increased 15 W per minute until exhaustion (Katayama et al., 2014; Katayama et al., 2015). The pedaling rate was maintained at 60 rpm with the aid of a metronome. Oxygen uptake (VO_2), carbon dioxide output (VCO_2), expired minute ventilation (VE), and respiratory exchange ratio (RER) were determined using an online system that enabled breath-by-breath measurements (ARCO-1000, Arco System, Chiba, Japan). HR was continuously monitored by telemetry (OEC-6401, Nihon Koden, Tokyo, Japan). Cardiorespiratory parameters were recorded during the tests and were averaged every 30 s afterward. The highest VO_2 value obtained during the exercise protocol was used as the VO_{2peak} value.

2.4. Pulmonary function and respiratory muscle strength

Pulmonary function [forced vital capacity (FVC), the forced expiratory volume in 1 s ($FEV_{1.0}$, $FEV_{1.0\%}$), and maximal voluntary ventilation for 12 s (MVV_{12})] was determined using a computerized spirometry system (AS-507, Minato Ikagaku, Osaka, Japan). These measurements were repeated five times, and the highest three values were averaged for all variables except for MVV_{12}. MVV_{12} was measured twice, and the higher value was accepted. Adopted values of each measurement were agreed within 3% at FVC, $FEV_{1.0}$ and $FEV_{1.0\%}$ (Miller et al., 2005; Wanger, 2012). The maximal inspiratory and expiratory pressures (PI_{max} and PE_{max}, respectively) were measured using a hand-held mouth pressure meter (AAM377, Minato Ikagaku, Osaka, Japan) connected to a computerized spirometry system, while the subjects sat in a reclined chair (the chair back was reclined to 75°). The PI_{max} was taken from the functional residual capacity (FRC) (Agostoni and Rahn, 1960; Uldry and Fitting, 1995). The PE_{max} was taken from the end-inspiration at rest; during the measurement, the subjects brought their hand to their cheeks and pressed forcefully during the expiratory effort to prevent air leaks around the mouthpiece (American Thoracic Society/European Respiratory Society, 2002; Katayama et al., 2015). For the PI_{max} and PE_{max} measurements, additional maneuvers were performed when the final maneuver had the highest value (Wanger, 2012). The adopted values of each measurement were agreed within 10% at PI_{max} and PE_{max} (Miller et al., 2005; Wanger, 2012).

2.5. Diaphragm thickness

T_{di} was monitored by B-mode ultrasonography (Vivid i; GE Healthcare Japan, Tokyo, Japan) and recorded while the subject was sitting in a reclined chair, similar to the pulmonary function test. The subjects placed their right arm on an arm stand, and held the spirometry system transducer in their left hand to perform the vital capacity (VC) maneuver. A researcher simultaneously monitored the ultrasound and the spirogram. T_{di} images were stored in a computer at a frequency of 20–30 Hz using a frame grabber (VGA2USB LR; Epiphan Video, Ottawa, Canada) for offline analysis and measured using image-analysis software (ImageJ, National Institutes of Health, Bethesda, MD, USA), as previously reported (Katayama et al., 2016). The measurement of T_{di} was done as described in previous studies (Cohn et al., 1997; De Bruin et al., 1997; Ueki et al., 1995). The 7th, 8th or 9th intercostal spaces on the right side, between the anteroaxial and midaxial lines, were identified in each subject and marked with a semi-permanent marker. The ultrasound probe was held between the ribs perpendicular to the chest wall. Part of the diaphragm in the zone of apposition, 0.5–2.0 cm below the costophrenic sinus, was observed in this area. The diaphragm was identified by two clear parallel bright lines, the pleural and peritoneal membranes, and the distance between the outside walls of the pleura and peritoneal membrane was measured. T_{di} at FRC was measured during resting breathing. T_{di} at total lung capacity (TLC) was measured while subjects held their breath with an open airway. The FRC steadiness and plateau at TLC were confirmed with the spirogram. For each image, T_{di} was measured at five separate points in one image within 0.3–0.5 cm, and the values, except for one outlier (median ± 2SD), were averaged.