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Voluntary hypocapnic hyperventilation lasting 5 min and 20 min similarly reduce aerobic metabolism without affecting power outputs during Wingate anaerobic test

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K.D. and T.N. conceived and designed experiments; K.D. and T.F. performed experiments; K.D. analyzed data; K.D., N.F., M.I., T.F. and T.N. interpreted results of experiments; K.D. prepared figures; K.D. drafted manuscript; K.D., N.F., M.I., T.F. and T.N. edited and revised manuscript; All authors approved the final version of manuscript.

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ABSTRACT

Twenty minutes of voluntary hypocapnic hyperventilation prior to exercise reduces the aerobic metabolic rate with a compensatory increase in the anaerobic metabolic rate without affecting exercise performance during the Wingate anaerobic test (WAnT). Thus, pre-exercise hypocapnic hyperventilation may be a useful means of stressing the anaerobic energy system during training, ultimately improving anaerobic exercise performance. However, it remains unclear whether a shorter (e.g., 5 min) pre-exercise hypocapnic hyperventilation is sufficient to reduce the aerobic metabolic rate during high-intensity exercise. We therefore compared the effects of 5-min and 20-min pre-exercise hypocapnic hyperventilation on aerobic metabolism during the 30-s WAnT. Ten healthy young males and one female performed the WAnT following 20 min of spontaneous breathing (control trial) or 5 or 20 min of voluntary hypocapnic hyperventilation. Both the 5-min and 20-min hyperventilation reduced end-tidal CO_2 partial pressure (an index of arterial CO_2 partial pressure) to ~23 mmHg, whereas it remained unchanged during the spontaneous breathing. The peak, mean and minimum power outputs during the WAnT did not differ among the three trials. Oxygen uptake during the WAnT was lower in both the 5-min (1493±257 mL min^{-1}) and 20-min (1397±447 mL min^{-1}) hyperventilation trials than during the control trial (1847±286 mL min^{-1}), and was similar in the two hyperventilation trials. These results suggest that 5 min of pre-exercise hypocapnic hyperventilation reduces aerobic metabolism during the 30-s WAnT to a

level similar to that seen with the 20-min hyperventilation. Moreover, exercise performance was unaffected, which implies anaerobic metabolism was enhanced.

(249/250 words)

INTRODUCTION

Sporting events such as the 200-m run or 50-m swim take trained athletes ~30 s to complete (Rodríguez and Mader 2011; Spencer and Gastin 2001). The relative energy contribution by the anaerobic energy system during short-term, high-intensity exercise lasting ~30 s is approximately 70% (Rodríguez and Mader 2011; Spencer and Gastin 2001). And because this energy system is highly involved in generating high-power output (Gastin 2001; Rodríguez and Mader 2011), training that improves performance of the anaerobic energy system is likely critical to improve short-term high-intensity exercise performance. Indeed, the overload principle (ACSM 2009) predicts that stressing the anaerobic energy system during training will enhance its capacity. In that regard, short-term, high-intensity intermittent training is known to effectively stress the anaerobic energy system during training (Tabata 2019). Thus, interventions that facilitate utilization of the anaerobic energy source during short-term, high-intensity exercise training should improve performance of the anaerobic energy system, ultimately leading to better high-intensity exercise performance.

Previous studies have consistently shown that voluntary hypocapnic hyperventilation

increases the anaerobic metabolic rate, as evidenced by a reduction in the aerobic energy supply (Chin et al. 2010, 2013; Chin et al. 2007; Keir et al. 2018; LeBlanc et al. 2002) and an increase in phosphocreatine break down (Forbes et al. 2007) with unchanged total workload during moderate intensity exercise. This suggests voluntary hyperventilation accompanied by reduced arterial CO_2 partial pressure (hypocapnia) may be an effective approach to stressing the anaerobic energy system. Recently, similar metabolic responses induced by voluntary hypocapnic hyperventilation were observed during a 30-s high-intensity intermittent cycling exercise (Dobashi et al. 2017) and the 30-s Wingate anaerobic test (WAnT) (Fujii et al. 2015; Leithauser et al. 2016). Those studies employed prolonged (e.g., 15-20 min) pre-exercise voluntary hyperventilation to develop hypocapnia, since ~15 min of voluntary hyperventilation is required to remove 90% of the body's CO_2 stores (Brandi and Clode 1969). This protocol also reduces the aerobic metabolic rate during exercise by inhibiting activation of mitochondrial pyruvate dehydrogenase (LeBlanc et al. 2002) and delaying the increase in active muscle blood flow (Chin et al. 2010, 2013). However, 15-20 min of voluntary hyperventilation lengthens the training duration, which may offset the beneficial effect (i.e., an increase in the anaerobic energy supply) of this intervention. If a shorter period of hypocapnic hyperventilation would sufficiently augment the anaerobic energy supply during high-intensity exercise, one could minimize the length of the training session.

Chin et al. (2007, 2010) demonstrated that voluntary hypocapnic hyperventilation increased oxy-hemoglobin for up to ~5 min following the onset of hyperventilation. This indicates that a leftward shift in the oxy-hemoglobin dissociation curve (Guyton 2011) mediated by short-term hypocapnic hyperventilation attenuates oxygen off-loading from hemoglobin. In addition, vasoconstriction within active muscles (e.g., femoral arteries) can be induced by ≥5 min of voluntary hypocapnic hyperventilation (Chin et al. 2010, 2013). This suggests short-term (e.g., 5 min) hypocapnic hyperventilation prior to exercise may be sufficient to reduce the aerobic metabolic rate during high-intensity exercise to the same level achieved by 20 min of hyperventilation. Hence, the purpose of the present study was to compare the effects of 5 min and 20 min of pre-exercise voluntary hypocapnic hyperventilation on metabolic responses during the 30-s WAnT. We hypothesized that 5 min of voluntary hypocapnic hyperventilation prior to exercise would decrease the aerobic metabolic rate to an extent similar to that achieved through 20-min of voluntary hyperventilation, without affecting the power output during the 30-s WAnT. If this is true, it would suggest the anaerobic energy supply increases to compensate for the reduced aerobic energy supply under these conditions.

METHODS

Participants

This study was approved by the local ethical committee and was carried out in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to their participation in this study. Ten healthy males and one female participated in the present study (n = 11). The menstrual cycle phase of the one female was not consistent across tests. The participants' age, height and body mass, presented in the mean ± standard deviation, were 24 ± 2 years, 1.73 ± 0.05 m, and 68.7 ± 7.0 kg. None of the participants were smokers or were taking any prescription medication. These participants included both trained athletes (seven college sprinters (one female) and one college badminton player; 120-240 min of structured training per day, 5-6 days per week) and three physically active young adults. All refrained from caffeine and alcohol for >24 h and food for 2 h prior to the experiment, and all were instructed to avoid intense exercise the night before the experiment.

Experimental session

Participants performed a 30-s WAnT using an electromagnetic brake bicycle ergometer (Powermax-V3; COMBI, Japan) with toe clips and sprint racing handlebar in an environmental chamber regulated to 25°C. The exercise was performed under three breathing conditions on separate days (> 48 h between trials in randomized order): 1) 20-min spontaneous breathing rest (control), 2) 5 min or 3) 20 min of voluntary hypocapnic hyperventilation (5-min or 20-min

hyperventilation trial, respectively). In all trials, each participant initially adjusted the seat position on the bicycle ergometer to their individual preference. After all equipment had been attached, the participants rested for 3 min while sitting on the seat of the ergometer (Baseline). Participants then performed a 3-min warm-up at 1.5 kp with a pedaling rate of 60 revolutions min^{-1}, followed by a 5-min rest on the bicycle. Thereafter, they spent 20 min resting under three breathing conditions (Breathing intervention), as described above. During the 5-min and 20-min voluntary hyperventilation periods, the participants increased their minute ventilation to 30 L min^{-1} achieved with a tidal volume of 1 L and a respiratory frequency of 30 breaths min^{-1}. This respiratory pattern was accomplished using visual feedback from a computer display showing the tidal volume and auditory cues from a metronome indicating the respiratory frequency. This respiratory pattern was selected based on earlier studies (Dobashi et al. 2017; Fujii et al. 2015). Immediately after each 20-min Breathing intervention period, the participants performed the 30-s WAnT, during which he/she was instructed to reach peak revolutions as quickly as possible and to make a full effort until the end of the exercise. Pedal resistance was set to 0.075 kp per kg of individual body mass. In all trials, the participants breathed spontaneously during the WAnT.

Measurements

Respiratory and metabolic variables

The participants breathed from a low-dead space mask that covered the nose and mouth. A pneumotachograph transducer for evaluating respiratory volume was attached to the mask. A gas-sampling tube was attached to the pneumotachograph transducer. Respiratory and metabolic variables were assessed using a mass spectrometer (ARCO-2000; Arco System, Japan), which analyzed respiratory O_2 and CO_2 pressure. Before making any measurements, the mass spectrometer was calibrated using reference gases of known concentration, and the pneumotachograph transducer was calibrated using a syringe with a known volume of 3 L. Based on the respiratory volume and/or gases measured, the mass spectrometer provided breath-to-breath minute ventilation, tidal volume, respiratory frequency, end-tidal CO_2 partial pressure, end-tidal O_2 partial pressure, oxygen uptake and carbon dioxide elimination. Air expired during the 30-s WAnT was collected into a Douglas bag to determine oxygen uptake and minute ventilation. After completion of the experiment, the internal volume of the Douglas bag was measured using a dry gas meter (DC-5A; Shinagawa, Japan). Mixed expired O_2 and CO_2 fractions in the Douglas bag were analyzed using the aforementioned mass spectrometer. We used a Douglas bag during the 30-s WAnT because in our pilot work we occasionally detected artifacts in the breath-to-breath data collected from the mass spectrometer that were associated with rapid changes in respiration and body movements during the 30-s WAnT.

The power output and pedaling rate were fed into a computer every 0.1 s and were averaged over every 5 s. Heart rate was recorded every 5 s using a heart rate monitor (Vantage NV; PORAL, Finland). Borg's ratings of perceived exertion (Borg 1982) were recorded immediately after the WAnT.

Data analyses

We determined peak power from the highest 5-s power and minimum power from the lowest 5-s power recorded during the 30-s WAnT. Mean power was determined by averaging the power over the entire 30 s of the WAnT period. A fatigue index was calculated as the absolute difference between the peak and minimum power expressed as a percent of the peak power.

Respiratory, metabolic and heart rate values at Baseline were obtained by averaging the values recorded over the 3-min period before the warm-up. Respiratory, metabolic and heart rate values during the Breathing intervention were obtained by averaging values recorded over a period extending from the 18th to the 19th min of the Breathing intervention. Peak heart rate was determined from highest 5-s value observed during the 30-s WAnT.

Due to technical difficulties, the respiratory and metabolic variables measured using the Douglas bag in two male participants and the heart rate in one male participant were not determined. We therefore analyzed respiratory and metabolic variables during the WAnT for the

remaining 9 participants and analyzed heart rates for the remaining 10 participants, respectively.

Statistical analyses

The data are expressed as the mean ± standard deviation. Power output, peak heart rate, respiratory and metabolic variables during the 30-s WAnT, and ratings of perceived exertion were analyzed using one-way repeated measures analysis of variance with a factor of trial (control, 5-min hyperventilation, 20-min hyperventilation). Respiratory and metabolic variables and heart rate during Baseline and Breathing intervention were analyzed using two-way repeated-measures analysis of variance with two factors: trial (levels: control, 5-min hyperventilation and 20-min hyperventilation) and time (levels: Baseline and Breathing intervention). When a main effect or interaction was detected, post hoc analyses were done using a Tukey's HSD procedure. Statistical significance was set at < 0.05. All statistical analyses were performed using SPSS version 22 for Windows (IBM SPSS Statistics, IBM, USA).

RESULTS

Respiratory, metabolic and heart rate responses at Baseline and during Breathing intervention

Time-dependent changes in respiratory and metabolic variables and heart rate measured at Baseline and during the Breathing interventions are presented in Table 1. At Baseline, none of

the variables differed significantly among the three trials. By design, tidal volume, respiratory frequency and minute ventilation were all higher during the Breathing interventions in the two hyperventilation trials than in the control trial ($P < 0.05$), with no significant difference between the two hyperventilation trials. As a result, the end-tidal CO_2 partial pressure was lower in the 5-min and 20-min hyperventilation trials than in the control trial ($P < 0.05$, Table 1). End-tidal O_2 partial pressures during the Breathing interventions were higher in both hyperventilation trials than in the control trial, with no significant difference between the two hyperventilation trials. Heart rates measured at Baseline were similar among the three trials, whereas those measured during the Breathing interventions were higher in the 5-min hyperventilation than in the control or 20-min hyperventilation trial ($P < 0.05$, Table 1).

Power output and respiratory, metabolic and heart rate responses during the 30-s WAnT

Performance of the 30-s WAnT was similar in the control, 5-min hyperventilation and 20-min hyperventilation trials. This was reflected by the comparable peak (794 ± 113 vs. 786 ± 103 vs. 795 ± 81 W, $P > 0.05$, Figure 1A), mean (627 ± 87 vs. 623 ± 69 vs. 633 ± 87 W, $P > 0.05$, Figure 1B), and minimum (478 ± 87 vs. 474 ± 69 vs. 493 ± 27 W, $P > 0.05$) power outputs and fatigue indexes (39 ± 4 vs. 39 ± 6 vs. 38 ± 5 %, $P > 0.05$).

Oxygen uptake during the 30-s WAnT was lower in the 5-min and 20-min hyperventilation trials than the control trial (1493 ± 257 vs. 1397 ± 447 vs. 1847 ± 286 mL min^{-1}, $P < 0.05$, Figure 2A), with no difference between the two hyperventilation trials ($P = 0.564$). Minute ventilation during the 30-s WAnT was lower in the 5-min hyperventilation trial (89 ± 33 L min^{-1}) than the control trial (108 ± 37 L min^{-1}, $P = 0.023$). Minute ventilation tended to be lower in the 20-min hyperventilation trial (93 ± 49 L min^{-1}) than the control trial, but the difference was not statistically significant ($P = 0.077$). Minute ventilation was similar in the two hyperventilation trials ($P > 0.05$). Peak heart rate was similar among the control (169 ± 10 beats min^{-1}), 5-min hyperventilation (168 ± 14 beats min^{-1}) and 20-min hyperventilation (165 ± 10 beats min^{-1}) trials.

Ratings of perceived exertion

Ratings of perceived exertion immediately after the 30-s WAnT were similar among the control, 5-min hyperventilation, and 20-min hyperventilation trials (18 ± 1 vs. 17 ± 2 vs. 18 ± 2, $P > 0.05$).

DISCUSSION

We evaluated the effects of 5-min and 20-min pre-exercise voluntary hypocapnic hyperventilation on metabolic responses during the 30-s WAnT. As per our original hypothesis,

5 min of hyperventilation reduced oxygen uptake during the 30-s WAnT to an extent similar to that observed with the 20-min hypocapnic hyperventilation (Figure 2A). Despite this reduction, power output did not differ among the three trials. These results suggest that voluntary hypocapnic hyperventilation increased the anaerobic metabolic rate to compensate for the reduced aerobic metabolic rate during the exercise, regardless of the hyperventilation duration (i.e., 5 vs. 20 min). In other words, a hyperventilation duration of 5 min was sufficient to reduce aerobic metabolism and thus augment anaerobic metabolism during high-intensity short-term exercise.

The observed reduction in oxygen uptake during the WAnT, associated with pre-exercise voluntary hyperventilation, may have been mediated in part by the lower minute ventilation during the exercise in both hyperventilation trials (Figure 2B). This reduction in exercise ventilation associated with hypocapnic hyperventilation is in good agreement with earlier studies testing responses during submaximal (Ward et al. 1983) and high-intensity constant workload exercise (Dobashi et al. 2017). An increase in end-tidal CO_2 partial pressure, which is an index of arterial CO_2 partial pressure, can stimulate central chemoreceptors by altering the medullary hydrogen ion concentration, thereby increasing ventilation (Ainslie and Duffin 2009; Bruce and Cherniack 1987; Duffin 2005; Duffin 2010; Duffin et al. 2000). Thus, hypocapnia-induced deactivation of the central chemoreflex may contribute to the observed attenuation of exercise ventilation. It is noteworthy, however, that some participants did not exhibit a reduction in minute

ventilation during the 30-s WAnT in the 5- and/or 20-min hyperventilation trials as compared to the control trial (Figure 2B). There appear to be individual differences in the ventilatory response to hypocapnia, and one or more factors other than ventilation likely contribute to the observed reductions in oxygen uptake in those who showed no reduction in ventilation in response to hypocapnia.

There are several possible non-ventilatory mechanisms that could be responsible for the reduced oxygen uptake seen during the WAnT. Chin et al. (2010, 2013) reported that 20 min of voluntary hypocapnic hyperventilation caused vasoconstriction of the femoral arteries, which delayed the initial increase in active muscle blood flow, thereby reducing oxygen uptake. This femoral arterial vasoconstriction was also observed ~5 min after the onset of hyperventilation, though the magnitude of the response was smaller than after 20 min (Chin et al. 2010, 2013). We therefore speculate that hemodynamic impairment within the active skeletal muscle may have contributed to the reduction in oxygen uptake in the 5-min hyperventilation trial, and perhaps to a greater extent in the 20-min hyperventilation trial. Moreover, Chin et al. (2007, 2010) demonstrated that voluntary hypocapnic hyperventilation increased oxy-hemoglobin for up to ~5 min following the onset of hyperventilation. This indicates that short-term (~5 min) hypocapnic hyperventilation induces a leftward shift in the oxy-hemoglobin dissociation curve (Guyton 2011), attenuating oxygen off-loading from hemoglobin. This mechanism may be specifically involved

in the reduced oxygen uptake in the 5-min hyperventilation trial. By contrast, 15 min of voluntary hypocapnic hyperventilation impairs activation of mitochondrial pyruvate dehydrogenase for oxidative phosphorylation during submaximal exercise (LeBlanc et al. 2002) (Spriet and Heigenhauser 2002). A similar response may have selectively occurred in the 20-min hyperventilation trial, contributing to the reduced oxygen uptake during WAnT. Taken together, these findings suggest that some of the mechanisms contributing to the reduction in the aerobic metabolic rate during the 30-s WAnT may be common to both hyperventilation trials, but other mechanisms may differ, depending on the duration of voluntary hyperventilation. For example, some participants exhibited greater reductions in oxygen uptake during the 30-s WAnT in the 20-min hyperventilation trial than in the 5-min hyperventilation trial (Figure 2A). In addition to the individual differences in ventilatory responses discussed above, hypocapnic hyperventilation-mediated decreases in leg blood flow and/or deactivation of pyruvate dehydrogenase may also have varied among the participants, contributing to the interindividual heterogeneity. This possibility will to be assessed in future studies.

Although marked reductions in oxygen uptake were observed with hypocapnia during exercise, this was not detected under resting conditions (i.e., Breathing intervention). Previous studies reported that hypocapnic hyperventilation reduced leg blood flow and inhibited pyruvate dehydrogenase activity during the onset of submaximal exercise, but not during pre-exercise

resting periods (Chin et al. 2010, 2013; LeBlanc et al. 2002). These differential responses between rest and exercise may explain why reduced oxygen uptake was only detected during exercise.

Limitations

There are several limitations to note. First, during the voluntary hyperventilation maneuver (i.e., Breathing intervention), end-tidal CO_2 partial pressure was slightly higher (i.e., 2.9 mmHg) after the 5-min hyperventilation than after the 20-min hyperventilation (Table 1). We do not know if or to what extent this small difference affected our results. Second, we did not control for the menstrual cycle of the one female participant. The menstrual cycle is known to affect ventilation, metabolic rate, and cardiovascular responses (Janse de Jonge 2003; Tanaka et al. 2003). Although the female participant's pattern of responses in the 5-min and 20-min hyperventilation trials was similar to those of the males, we cannot draw a clear conclusion regarding sex-related differences and the impact of the menstrual cycle. Delineating these will require recruitment of more female participants and controlling for the menstrual cycle in the future. Third, because we did not measure oxy-hemoglobin at the active skeletal muscles, activity of mitochondrial pyruvate dehydrogenase, and skeletal muscle blood flow, we cannot say if or to what extent altered these factor(s), if any, contributed to the reduced aerobic metabolic rate during the 30-s WAnT in the 5-min and 20-min hyperventilation trials. Fourth, because we investigated responses only in a

combined group of athletes and physically active individuals, it is unclear whether similar responses would be observed in other populations, such as more sedentary individuals. Finally, because we did not measure indices of anaerobic metabolism, it remains unclear whether hypocapnic hyperventilation truly augmented the anaerobic energy supply during the 30-s WAnT. Previous studies demonstrated that 20 min of hypocapnic hyperventilation, as employed in the present study, increased the blood lactate concentration as compared to spontaneous breathing (Dobashi et al. 2017; Fujii et al. 2015). A similar response may have been induced in the present study. However, blood lactate concentrations may not precisely reflect anaerobic lactate production in exercising muscles (Green and Dawson 1993; Sacks and Sacks 1937). To draw a firm conclusion regarding anaerobic metabolism, it will be necessary to assess muscle glycogen, lactate and phosphocreatine, as was done in previous studies (Forbes et al. 2007; LeBlanc et al. 2002).

Practical applications

Traditionally, high-intensity exercise under hypoxic conditions has been used to increase the anaerobic metabolic rate without influencing exercise performance (Calbet et al. 2003; Ogawa et al. 2007; Ogawa et al. 2005; Weyand et al. 1999). This implies that hypoxia has an increasing effect on the anaerobic energy system during high-intensity exercise training. However, the

practical application of altitude/hypoxic training is often limited, as visiting high-altitude sites or installing a hypobaric chamber is quite expensive. Alternatively, previous studies have now shown that 15-20 min of pre-exercise voluntary hypocapnic hyperventilation increases the anaerobic metabolic energy supply as compared to spontaneous breathing (Dobashi et al. 2017; Fujii et al. 2015; Leithauser et al. 2016). Voluntary hypocapnic hyperventilation can be easily produced by monitoring arterial CO_2 partial pressure. However, 15-20 min of voluntary hyperventilation prolongs training duration, which may offset the beneficial effect (i.e., an increase in the anaerobic energy supply) of this intervention. Our results demonstrate that 5-min of voluntary hypocapnic hyperventilation is sufficient to reduce aerobic energy metabolism during short-term high-intensity exercise (30-s WAnT) to an extent similar to that observed with 20-min voluntary hypocapnic hyperventilation, without affecting exercise performance. We therefore propose that short-term (e.g., 5 min) hypocapnic hyperventilation is a more time-efficient approach for stressing the anaerobic energy system during high-intensity exercise than 20-min hypocapnic hyperventilation. However, because we tested only the acute effect of hypocapnic hyperventilation, it remains to be determined whether greater adaptation of the anaerobic energy system is achieved through repeating high-intensity exercise with hypocapnic hyperventilation or through training with spontaneous breathing. This knowledge gap will be directly addressed in the future.

CONCLUSIONS

Our results demonstrate that 5-min and 20-min pre-exercise voluntary hypocapnic hyperventilation similarly reduce oxygen uptake during 30-s short-term all-out exercise without influencing exercise performance. This indicates that shorter duration, voluntary hypocapnic hyperventilation (~5 min) sufficiently reduces the aerobic metabolic rate with a compensatory increase in the anaerobic metabolic rate during the exercise.

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CONFLICT OF INTEREST

The authors have no competing interests to declare.

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Table 1. Respiratory and metabolic variables and heart rates at Baseline and Breathing intervention

	Control	5-min hyperventilation	20-min hyperventilation
Minute ventilation (L min^{-1})			
Baseline	13.4 ± 3.0	13.3 ± 1.9	13.3 ± 2.2
Breathing intervention	13.8 ± 3.4	29.5 ± 2.5*	27.4 ± 3.2†
Tidal volume (L)			
Baseline	0.69 ± 0.14	0.69 ± 0.08	0.68 ± 0.10
Breathing intervention	0.70 ± 0.15	1.07 ± 0.18*	0.94 ± 0.10†
Respiratory frequency (breaths min^{-1})			
Baseline	20 ± 2	20 ± 2	21 ± 3
Breathing intervention	21 ± 3	29 ± 3*	30 ± 1†
Oxygen uptake (mL min^{-1})			
Baseline	361 ± 83	362 ± 75	355 ± 86
Breathing intervention	376 ± 111	339 ± 82	371 ± 140
Carbon dioxide elimination (mL min^{-1})			
Baseline	335 ± 87	325 ± 66	329 ± 72
Breathing intervention	341 ± 106	505 ± 134*	418 ± 83†‡
End-tidal O_2 partial pressure (mmHg)			
Baseline	118 ± 3	118 ± 4	119 ± 3
Breathing intervention	118 ± 4	136 ± 3*	133 ± 4†
End-tidal CO_2 partial pressure (mmHg)			
Baseline	32.4 ± 2.5	32.0 ± 3.1	31.9 ± 2.7
Breathing intervention	31.8 ± 3.1	22.5 ± 3.7*	19.6 ± 3.0†‡
Heart rate (beats min^{-1})			
Baseline	74 ± 11	76 ± 11	75 ± 9
Breathing intervention	78 ± 13	87 ± 12*	76 ± 8‡

The values are expressed as the mean ± standard deviation. *$P < 0.05$, Control vs. 5-min hyperventilation; †$P < 0.05$, Control vs. 20-min hyperventilation; ‡$P < 0.05$, 5-min vs. 20-min hyperventilation.

Figure 1

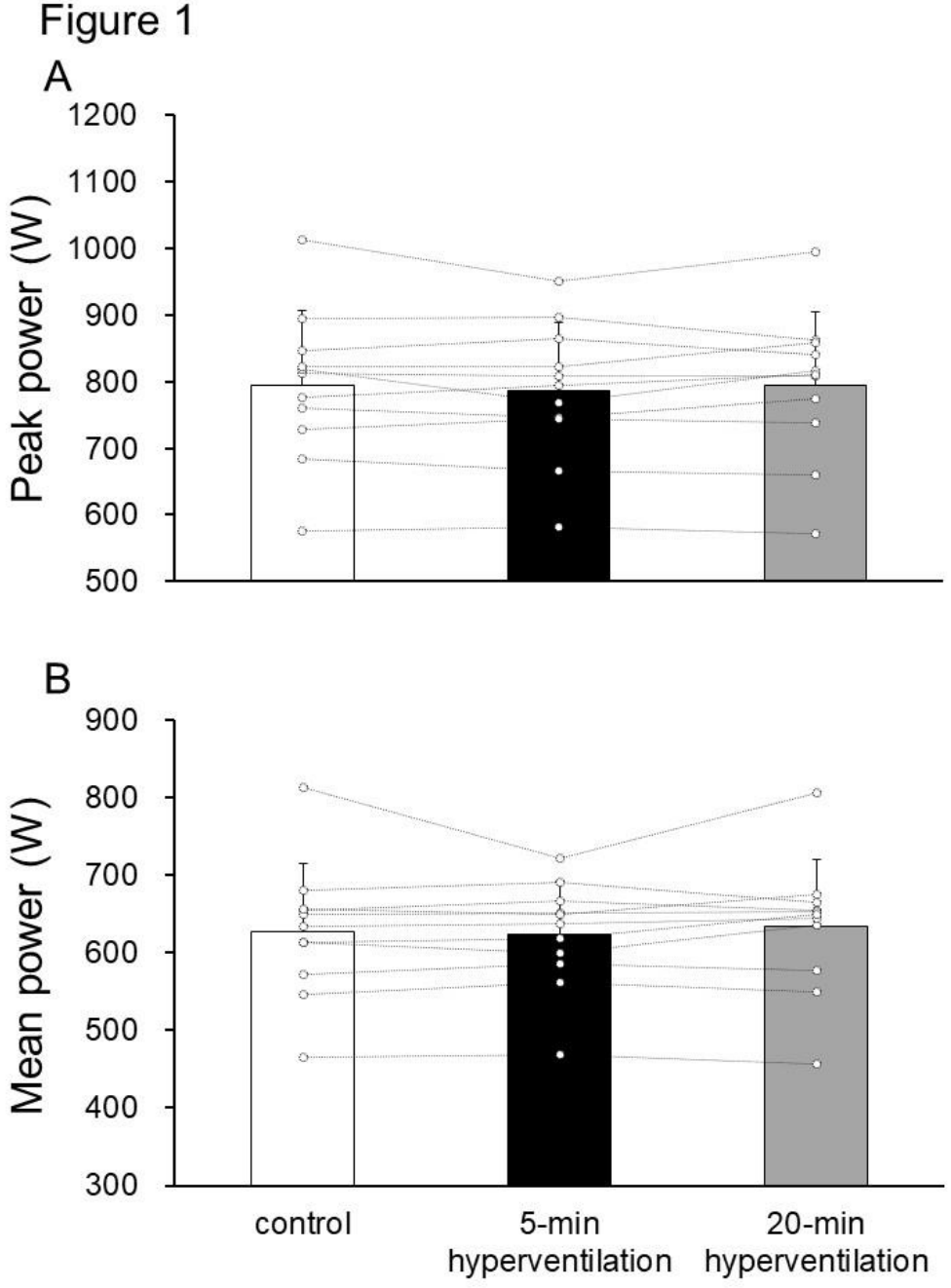

Figure 2