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Effects of elevated core temperature and normoxic 30% nitrous oxide on human ventilation during short duration, high intensity exercise

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ABSTRACT

It was hypothesized that normoxic 30% nitrous oxide (N_2O) would suppress and hyperthermia would increase exercise ventilation during short duration, high intensity exercise. Thirteen males (24.2 ± 0.8 y; mean ± SE), of normal physique (BMI, 23.8 ± 1.0 kg m^{-2}), performed 4 separate 30 s Wingate tests on a cycle ergometer. Exercise ventilation and its components, as well as mean skin and esophageal temperature (T_{ES}), were assessed in 2 way experimental design with factors of Thermal State (Normothermia or Hyperthermia) and Gas Type (Air or 30% Normomoxic N_2O). In the 2 hyperthermic tests T_{ES} was elevated to ~38.5 °C in a 40 °C bath. The main results indicated a significant interaction ($F = 7.14$, $P = 0.02$) between Gas Type and Thermal state for the exercise-induced increase in ventilation ($\Delta\dot{V}_E$). During both the normothermia and hyperthermia conditions with AIR breathing, the exercise $\Delta\dot{V}_E$ was ~80 L min^{-1} and it was significantly decreased to 73.1 ± 24.1 L min^{-1} in the normothermia condition with N_2O breathing relative to that of 92.0 ± 25.0 L min^{-1} in the hyperthermia condition with N_2O breathing. In conclusion, normoxic N_2O breathing suppressed high intensity exercise ventilation during normothermia relative to that during hyperthermia on account of decreases in the tidal volume and this led CO_2 retention.

1. Introduction

Undersea divers breathing compressed air between 4 and 6 Atmospheres Absolute (ATA) of pressure experience nitrogen narcosis, the effects of which are simulated by breathing normoxic 30% nitrous oxide (N_2O) at sea level pressure (Biersner, 1987). At submaximal exercise intensities, small but significant increases in pulmonary ventilation are evident during normoxic 30% N_2O breathing (Fothergill and Carlson, 1996), but at higher work intensities (Ciammaichella and Mekjavic, 2000) or in hypoxic conditions (Yacoub et al., 1976), N_2O might inhibit exercise ventilation, possibly due to a selective suppression of peripheral chemoreceptors (Yacoub et al., 1976). Suppression of exercise ventilation during work by undersea divers is of concern since there are unexplained deep-water black-outs in this population (Lanphier and Camporesi, 1993). SCUBA diver fatalities (Vann et al., 2005) are most often during the bottom phase of the dive, with ~90% of such divers breathing compressed air. The largest fraction of these fatalities occurred throughout the summer months of June to August during diving in warmer water temperatures in the southeast/southwest USA or the Caribbean. Forty-eight percent of these fatalities due to loss of consciousness were at the bottom of the dive and 27 of these were for no apparent reason (Vann et al., 2006).

Divers conducting physical work at depth can be rendered hyperthermic with hot water from a surface supply warming system (Mekjavic et al., 2001) or by exerting themselves in warm seas or oceans. Potential influences on breathing in these divers include nitrogen gas under pressure (Behnke et al., 1935), compressed air with a higher gas density (Comporesi and Bosco, 2003), and elevated body temperatures (Cunningham and O'Riordan, 1957; White and Cabanac, 1996). The narcotic effects of compressed air at depth are thought to inhibit ventilation (Comporesi and Bosco, 2003), whereas an elevated core temperature is known to stimulate human ventilation at rest (Cabanac and White, 1995; Haldane, 1905) or during exercise (Cunningham and O'Riordan, 1957; Nybo and Nielsen, 2001; White and Cabanac, 1996).

The present study was conducted to assess the separate and combined influences of a pre-exercise, passively-induced hyperthermia and of a narcotic gas on human ventilation during intense exercise. Passively-induced hyperthermia was induced with a pre-exercise, hot tub immersion and a large increase in exercise ventilation with a 30 s supra-maximal Wingate test. This exercise protocol included a transition from light to intense exercise and narcosis was induced with inhalation of normoxic 30% N_2O at

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~1 ATA. As such, the combined and separate effects of hyperthermia and narcosis were assessed for their influences on a sudden increase in exercise ventilation. It was hypothesized that normoxic 30% N_2O would inhibit and that increased core temperature would stimulate exercise ventilation in these exercise conditions.

2. Methodology

2.1. Volunteers

Thirteen male participants volunteered for the study. They were 24.2 ± 0.8 y of age (mean ± SD), 1.8 ± 0.0 m tall, 77.1 ± 4.0 kg in weight and had a body mass index of 23.8 ± 1.0 kg m^{-2}. The volunteers were physically fit as evident from their Wingate power output values when classified with a ranking method developed from Wingate tests on 485 male, Division 1 NCAA athletes (Zupan et al., 2009). Using this ranking method our volunteers peak power of 1107 ± 205 W put them within the range of 1092–1160 W for an 'excellent' ranking and their mean power values of 685 ± 104 W put them within the range of 640–731 W for an 'average' ranking (Zupan et al., 2009). Each volunteer was made aware of any risks associated with the protocol in this experiment. After reading a detailed outline of the study each participant signed a consent form. The sample size was determined using a power calculation. The difference worth detecting was set at 10%, with an alpha level of 0.05, a power of 0.8 and a standard deviation of 7% of the estimated mean scores for exercise ventilation ($\dot{V}_E$), frequency of breathing (F_B), tidal volume (V_T) and end-tidal partial pressure of carbon dioxide ($P_{ET}CO_2$). The Office of Research Ethics at Simon Fraser University approved the research.

2.2. Instrumentation

A calibrated breath-by-breath Vmax 229 series metabolic cart (Sensormedics, CA, USA) was employed to assess expired gases as well as ventilation and its components as described previously (Sancheti and White, 2006).

Esophageal temperatures (T_{ES}) were estimated using an esophageal temperature probe (size 9 Fr, Mallinkroft Medical Inc., St Louis, MO, USA) inserted to the level of the left ventricle (Mekjavic and Rempel, 1990). Mean un-weighted skin temperatures (T_{SK}) were estimated from sites at the chest, thigh, forehead and lower back with surface copper constantan thermocouples. All thermocouples were connected to a data acquisition system (National Instruments, USA) that was controlled by LabVIEW software (National Instruments, USA) on a personal computer.

The gas inhalant included either normoxic 30% nitrous oxide (30% N_2O, 21% O_2, balance N_2, Air Liquide Canada, Inc.) or air supplied to the volunteer in corrugated Collins respiratory tubing from a Tissot spirometer (Model 1464, Boston, Massachusetts, USA) or meteorological balloons.

Exercise trials were completed on an electrically braked, seated cycle ergometer (LODE Excalibur Sport, Sweden).

2.3. Protocol

Following instrumentation the volunteer participated in one of four 30 s Wingate exercise trials (Bar-Or, 1987) on the cycle ergometer. Trials were performed ~1 week apart and all participants were asked to refrain from ingesting caffeine for 12 h prior to exercise tests. Each participant inhaled either room air or normoxic 30% N_2O in a given Wingate test. The participant was normothermic in two Wingate tests and hyperthermic in two other Wingate tests. Prior to the hyperthermic tests the participant was warmed in a 40 °C water bath until T_{ES} increased by ~1.5 °C above the pre-immersion, resting value of ~37.1 °C (Fig. 1). This immersion took an average of 15 ± 4 min. After this participant was quickly seated on the cycle ergometer after exiting the bath and dressed in impermeable rain gear plus wrapped in blankets to maintain an elevated T_{ES} until the start of the Wingate test.

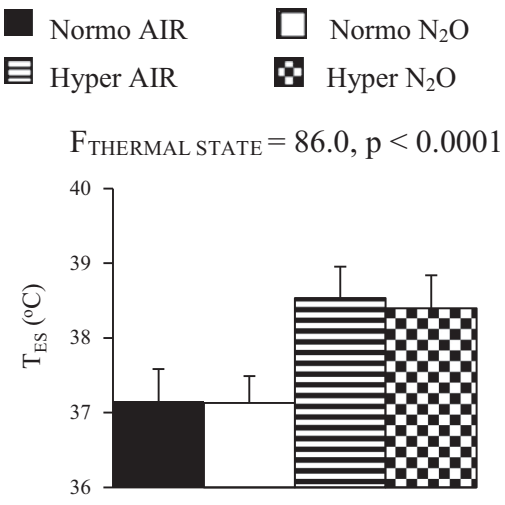

Fig. 1. Mean esophageal temperature (T_{ES}) during supramaximal Wingate exercise in each of the four conditions. Each vertical bar represents the mean response for 13 volunteers and the error bars give the standard deviation of the mean.

At the start of each exercise trial the volunteer, while seated on the cycle ergometer, began breathing air or normoxic 30% N_2O, through the mouthpiece for 5 min of rest, followed by a 30 s warm-up at 40 W with a pedaling cadence between 80 and 90 rpm. The volunteer was given a 5 s countdown at the end of warm-up cycling and then proceeded to perform a 30 s Wingate test, with instructions to pedal as hard and as fast as possible while the resistance was set at 0.09 kp (0.88 N kg^{-1}).

2.4. Statistical analysis

The dependent outcome variables of interest were the 30 s means of T_{ES}, $\dot{V}_E$, F_B, V_T and $P_{ET}CO_2$ as well as the difference (Δ) between mean pre-exercise rest and 30 s mean exercise values for $\Delta\dot{V}_E$, ΔF_B, ΔV_T and $\Delta P_{ET}CO_2$. An ANOVA model was employed with repeated factors of Thermal State (Normothermia and Hyperthermia) and inhaled Gas Type (Air, normoxic 30% N_2O). *A priori* paired *t*-tests were employed to compare means. Results were considered significant at an alpha level of 0.05.

3. Results

For T_{ES} there was a main effect of Thermal State ($F = 86.0$, $P < 0.0001$) but no main effect of Gas Type (Fig. 1). The mean T_{ES} in the normothermic conditions with air breathing was 37.15 ± 0.43 °C and 37.13 ± 0.36 °C during normoxic 30% N_2O breathing. In the hyperthermic conditions T_{ES} was elevated to 38.53 ± 0.42 °C during air breathing and to 38.40 ± 0.44 °C during normoxic 30% N_2O breathing. There were no main effects of Thermal State or Gas Type on mean T_{SK}. The mean T_{SK} in normothermia with air breathing was 33.42 ± 1.05 °C and it was similar at 33.71 ± 0.42 °C in normothermia with normoxic 30% N_2O breathing. For the hyperthermia conditions with air breathing T_{SK} was 32.92 ± 3.63 °C and 34.26 ± 2.83 °C with normoxic 30% N_2O breathing.

Over the 30 s of each Wingate test in each of the 4 conditions, no main effects of Thermal State or Gas Type were evident for $\dot{V}_E$, F_B, V_T (Fig. 2A–C). The mean $\dot{V}_E$ was at similar rates of 99.8 ± 32.9 L min^{-1} in normothermia with air breathing and 100.3 ± 27.7 L min^{-1} in