

A COMPARISON OF THERMAL HYPERPNEA RESPONSES BETWEEN YOUNGER AND OLDER MALES DURING HYPERTHERMIA

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INTRODUCTION

Hyperthermic-induced hyperventilation is evident with a passively-induced increase in core temperature of approximately 1.0°C (1, 12). As indicative of a hyperventilation or a thermal hyperpnea, this response is associated with a decrease in end-tidal CO₂ (P_{ET}CO₂) and an increase in end-tidal PO₂ (P_{ET}O₂). The mechanisms underlying this response during hyperthermia are still in the process of being resolved (3, 4, 12) and it is not known if and how this thermal hyperpnea response changes with aging in humans.

Older individuals have reduced thermoregulatory responses relative to those of younger individuals (5). Some of these thermoregulatory changes with aging include reduced sweat rate (9) and attenuated cutaneous blood flow (11). The increased breathing-related illness during heat waves suggests that in addition to their impaired thermoregulation, that there are changes in the control of breathing in older populations. It is unknown how hyperthermic-induced hyperventilation in older individuals compares to younger individuals. Similar to that seen for thermoregulatory responses, hyperthermic-induced hyperventilation was hypothesized to be reduced in older compared to younger individuals. To address this question, a younger control group was compared to an older test group assessing their ventilatory responses to a hyperthermic exposure in a climatic chamber.

METHODS

Volunteers: A sample size of 5 volunteers/group with an effect size to detect a 10% change in pulmonary ventilation was determined from preliminary results in our laboratory; with an $\alpha=0.05$ this gave a power of 0.9 for the study. Volunteers included 5 older males (mean±SD): age 57.8 (±5.3) years, weight 88.1±13.3 kg, height 1.80±0.09 m) and 5 younger controls (4 male 1 female; age 30.0±9.1 years, weight 71.1±11.4 kg, height 1.77±0.08 m). All were all non-smokers, non-asthmatics and refrained from caffeine, alcohol, and heavy exercise for 24 h prior to testing. The Office of Research Ethics at SFU granted approval for the study that was in compliance with the Helsinki Declaration. Each volunteer gave a written, informed consent after being thoroughly familiarized with the experimental protocol, instrumentation and risks of participation.

Instrumentation: Each participant wore a nose clip and was fitted with a mouthpiece mounted on a 2-way non-rebreathing valve (NRB 2700, Hans Rudolph Inc, Kansas City, Mo.) through which

gases were passed across the flow sensor. Breath-by-breath gas samples for measurement of gas partial pressures, pulmonary ventilation (V_E) as well as its components of Tidal Volume (V_T) and Frequency of Breathing (F_B), were drawn from the inspired and expired gases by a metabolic cart (Model: Vmax 229, Sensormedics, Yorba Linda, Calif.) at a rate of $\sim 600 \text{ mL} \cdot \text{min}^{-1}$. The details of the calibration of the metabolic cart have already been reported (2).

Arterial hemoglobin oxygen saturation (SaO_2) was measured using a pulse oximeter (Masimo Radical, Irvine, Calif.) attached to the participant's left ear lobe. In each volunteer a calibrated nasopharyngeal esophageal temperature (T_{ES}) thermistor probe (Mon-a-therm, Mallinckrodt Med. Inc., St Louis, Mo.) was positioned at the T8/T9 level (7).

The data acquisition system (Nat. Instr., Austin TX, USA) included a program written in LabVIEW (v. 7.0, Nat. Instr., Austin) operating on a Windows-based personal computer. An analog signal from the metabolic cart's flow sensor from each breath was used to trigger breath-by-breath data collection for pulmonary ventilation as well as HR, SaO_2 and T_{ES} .

A walk-in climatic chamber (L - 5.08 m, W - 3.75 m and H - 2.49 m; Tenney Engineering Inc., Union, NJ, USA) was employed for the passive heating portion of the study.

Protocol: With normothermic core temperatures (NT) resting data were collected from each volunteer for a 30-min period when resting T_{ES} was not significantly different ($p=0.80$) between the younger group at $36.76 \pm 0.17^\circ\text{C}$ and older group at $36.83 \pm 0.39^\circ\text{C}$. Next each volunteer was passively heated in a climatic chamber at 50°C , $\sim 20\%$ RH until the T_{ES} had risen by $\sim 1.4^\circ\text{C}$. Each volunteer wore shorts, T-shirt and running shoes and a tightly sealed vapour impermeable rain suit. When the T_{ES} was increased to the target hyperthermic core temperature (HT) the seals on the rain suit were opened and T_{ES} was maintained at that level. The increase in T_{ES} in the climatic chamber for younger group of $1.24 \pm 0.36^\circ\text{C}$ was not significantly different ($P=0.14$) than that of $1.47 \pm 0.23^\circ\text{C}$ for older group.

Statistical Analysis: A univariate ANOVA was employed with non-repeated factor Age (control and older groups) and repeated within group factor of Temperature (NT and HT). Between-age group post-hoc tests were conducted with an unpaired t-test. The level of significance was set to $\alpha < 0.05$. Analyses were performed with SPSS[®] (ver 17.0) statistical software package for Macintosh (SPSS Inc., Chicago, Ill.).

RESULTS

Pulmonary ventilation was not significantly different between the control and older group in either the NT ($p=0.75$) or HT ($p=0.45$) conditions. For pooled values across the two groups, body warming in the climatic chamber gave a significant increase ($p=0.03$) of V_E from approximately 14 L/min to 22 L/min (Fig. 1a). There was a corresponding trend for an increase ($p=0.09$) in V_T from ~ 0.95 L in the NT condition to ~ 1.34 L in the HT condition (Fig. 1b). Neither the younger nor the older group's F_B was influenced by the elevation of T_{ES} (Fig 1c) and each group's mean F_B response was not significantly different between conditions ($P=0.28$). For the younger and older group the $P_{ET}\text{CO}_2$ significantly decreased ($p=0.006$) from ~ 38 mm Hg to ~ 29 mm Hg (Fig. 2a) and $P_{ET}\text{O}_2$ significantly increased ($p=0.006$) from ~ 105 mm Hg to ~ 115

mm Hg (Fig. 2b). Heart rate significantly increased from 64.8 ± 9.8 b/min in NT to 115.7 ± 21.1 b/min in HT.

DISCUSSION AND CONCLUSIONS

The main result from the study was that similar pulmonary ventilation responses to hyperthermia were evident in the younger and older groups. Irrespective of age, the significant increase in V_E during hyperthermia was explained by a trend for a significant increase in tidal volume. The V_E response was confirmed to be a hyperventilation as was evident from the significant decreases in $P_{ET}CO_2$ and significant increases $P_{ET}O_2$ (Fig. 2).

Some evidence suggests chemosensitivity to the normal modulators of ventilation is reduced (6, 8) in older individuals during normothermia. Other evidence, however, supports during normothermia that septuagenarians have similar ventilatory responses to acute hypoxia as younger controls (10). These conflicting results demonstrate it remains to be resolved if during normothermia the impairments in breathing with aging are associated with changes in chemosensitivity.

The current novel results provide preliminary evidence to suggest that hyperthermic-induced hyperventilation is not reduced across an age range from ~30 to 60 years of age. Further research needs to be directed at resolving the independent and combined effects of hypoxic and hypercapnic as well as hyperthermic challenges on ventilatory responses in the older population.

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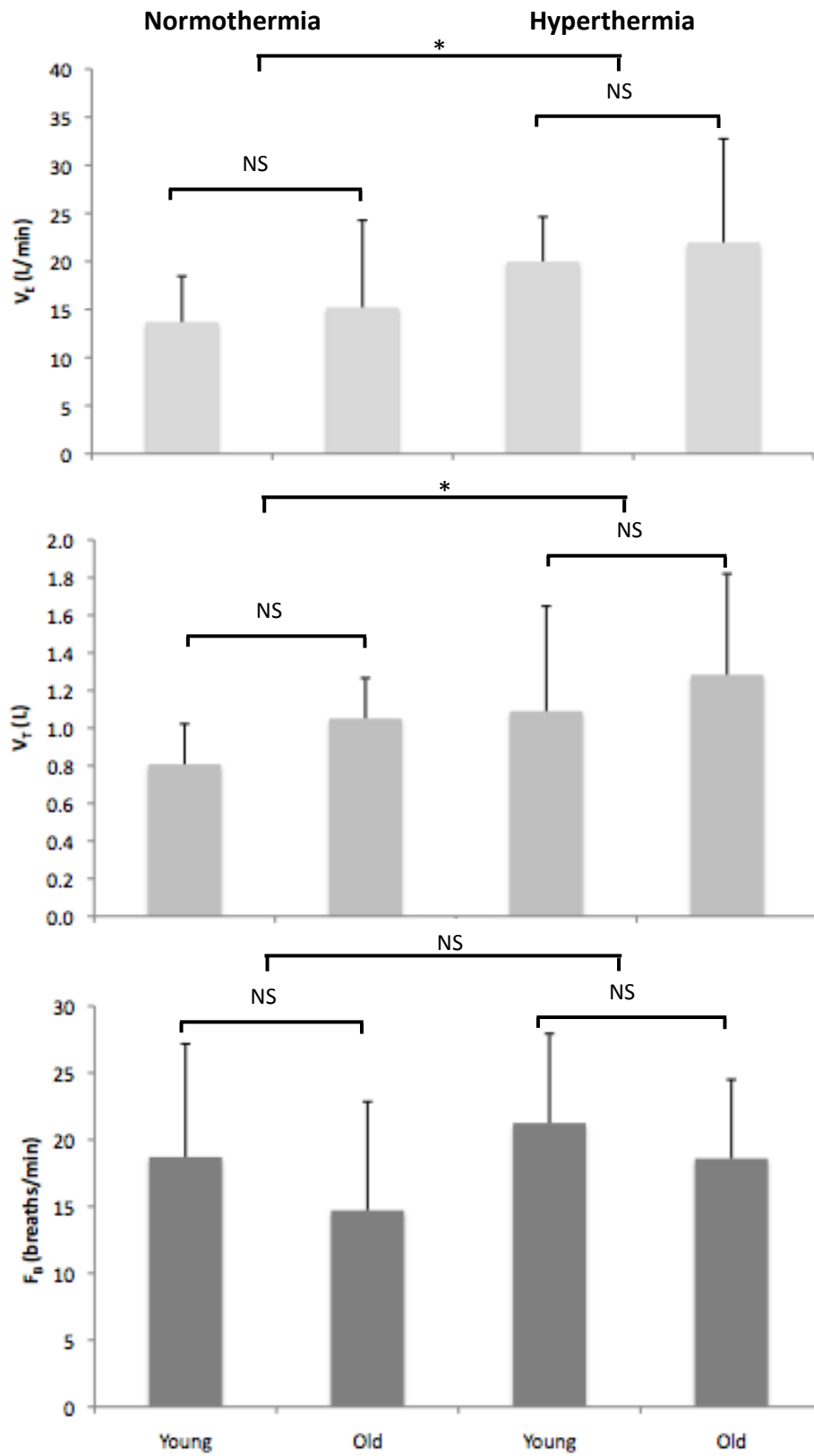


Figure 1. Pulmonary ventilation (V_E), tidal volume (V_T) and frequency of breathing (F_B) responses to hyperthermia in younger and older males (* $P < 0.05$, NS= non-significant).

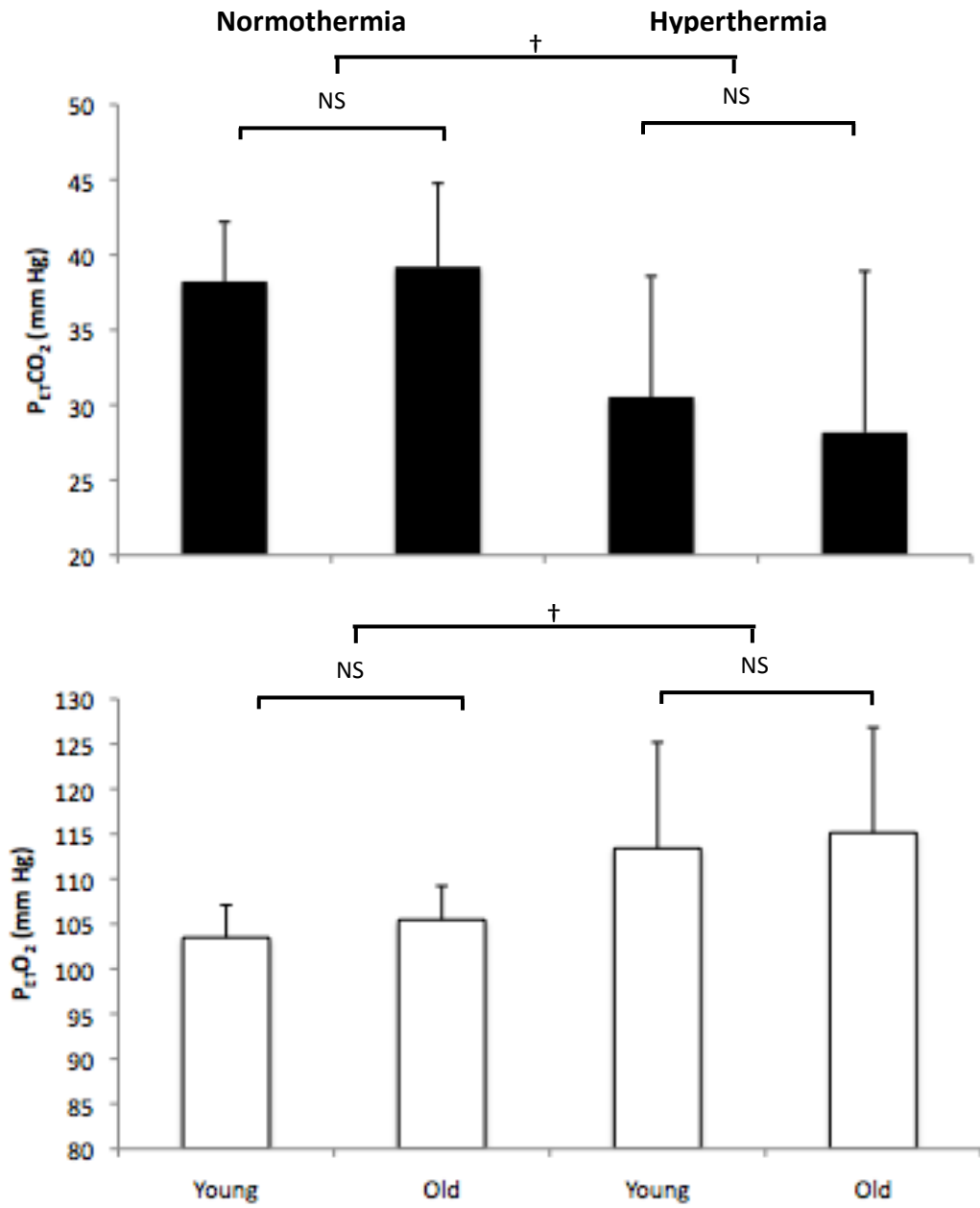


Figure 2. End-tidal partial pressure of CO₂ (P_{ET}CO₂) and end-tidal partial pressure of O₂ (P_{ET}O₂) responses to hyperthermia in younger and older males (†P < 0.01, NS= non-significant).