56

# <u>1-B-02</u> Arterial blood pressure response to severe passive heating at rest relates to hyperthermia-induced hyperventilation

Naoto FUJII<sup>1</sup>, Masashi ICHINOSE<sup>2</sup>, Bun TSUJI<sup>3</sup>, <sup>,</sup> Kazuhito WATANABE<sup>3</sup>, Narihiko KONDO<sup>4</sup>, Takeshi NISHIYASU<sup>3</sup>

<sup>1</sup> JSPS, <sup>2</sup> Meiji University,

<sup>3</sup>University of Tsukuba, <sup>4</sup>Kobe University

### **Purpose** :

The purpose of this study was to investigate that whether the mean arterial blood pressure (MAP) change during passive heating at rest is associated with hyperthermia-induced hyperventilation in humans.

### Methods:

Eighteen healthy males were subjected to passive heating using legs-only hot water immersion and a water-perfused suit until the subject could no longer endure the heat. We then divided the subjects into two groups: in MAP<sub>NOTINC</sub> (n = 8) MAP did not increase by >3 mmHg (-11.5 to 2.3 mmHg), and in MAP<sub>INC</sub> (n = 10) MAP increased by >3 mmHg (9.7 to 32 mmHg).

#### **Results and Discussion:**

Heating-induced increases in esophageal temperature were similar in MAP<sub>NOTINC</sub> and MAP<sub>INC</sub> (+2.3±0.3 vs. +2.4±0.4 °C). However, subjects in MAP<sub>NOTINC</sub> showed significantly greater increases in minute ventilation ( $V_E$ ) (+19.1±7.8 vs. +7.1±4.7 l min<sup>-1</sup>, P < 0.05) and greater decreases in end-tidal CO<sub>2</sub> pressure (-15.6±4.3 vs. -5.1± 4.3 mmHg, P < 0.05) than those in MAP<sub>INC</sub>. Among all subjects, heating-induced changes in  $V_E$  significantly and negatively correlated with heating-induced changes in MAP (r = -0.74, P < 0.05). Our results suggest the extent of the MAP response to passive heating at rest is associated with the degree of hyperthermia-induced hyperventilation.

**Key words:** hyperthermic hyperpnea; respiratory alkalosis, systemic blood pressure

## **1-B-03** Effect of pre-exercise core temperature on circadian variation in hyperthermic hyperventilation during exercise

Bun TSUJI<sup>1</sup>, Naoto FUJII<sup>2</sup>, Narihiko KONDO<sup>3</sup>, Takeshi NISHIYASU<sup>1</sup>

<sup>1</sup> Graduate School of Comprehensive Human Sciences, University of Tsukuba, <sup>2</sup> JSPS Fellow, <sup>3</sup> Faculty of Human Development, Kobe University

**Purpose:** Hyperthermia during exercise leads to hyperventilation. We recently reported that core temperature  $(T_c)$  threshold for hyperventilation as well as pre-exercise resting  $T_c$  was higher by 0.6°C in evening (PM) than morning (AM) However, the change in  $T_c$  from resting to the threshold was same between AM and PM. We therefore tested the hypothesis that even though higher resting  $T_c$  at PM is adjusted to the level at AM before exercise,  $T_c$  threshold for hyperventilation is still higher at PM than AM.

**Methods:** Nine male subjects performed cycle exercise at 50% of peak oxygen uptake in the heat (37°C) at AM (6:00) and PM (18:00). Before each exercise, subjects were immersed in water (18°C) for 25-min at AM and 50-min at PM to detect esophageal temperature ( $T_{es}$ ) threshold for hyperventilation (Tsuji *et al.* 2009) and to adjust  $T_{es}$  to same level.

**Results and Discussion:** Despite same resting  $T_{\rm es}$  before exercise after the immersion at AM and PM (35.6 ± 0.7 vs. 35.7 ± 0.7°C),  $T_{\rm es}$  threshold for hyperventilation was significantly higher at PM than AM (37.2 ± 0.7 vs. 36.5 ± 0.7°C). The finding suggests that even though elevated  $T_{\rm c}$  in evening is adjusted to the level in morning,  $T_{\rm c}$  threshold for hyperventilation is higher in evening than morning.

**Key Words:** Hyperpnea; Hyperthermia; Thermoregulation; Circadian rhythm; Precooling