Effects of CO_2 on ventilatory and cerebrovascular responses during passive heating in humans

Tsuji, B.[1], Filingeri, D.[2], Fujii, N.[3], Hayashi, K.[4], Eguchi, T.[1], Kondo, N.[5], Honda, Y.[1], Nishiyasu, T.[1]

[1] Institute of Health and Sports Science, University of Tsukuba, Japan

[2] Loughborough University, UK

[3] University of Ottawa, Canada

[4] University of Shizuoka, Japan

[5] Kobe University, Japan

Introduction

During passive heating of resting humans, minute ventilation (V_E) changes little as esophageal temperature (T_{es}, an index of core temperature) rises until the temperature reaches about 38°C. Above this threshold, V_E increases by ~30 L/min with respect to 1°C rise in T_{es} and results in a decrease in arterial CO_2 pressure ($PaCO_2$) (Tsuji et al. 2012). In nornothermic condition, generally, change in $PaCO_2$ can affect both ventilatory and cerebrovascular responses such that the decrease in $PaCO_2$ suppresses V_E via central chemoreceptors, and also reduces cerebral blood flow by cerebral vasoconstriction. However, the effect of reduced $PaCO_2$ on ventilatory and cerebrovascular responses during hyperthermia at rest is not well understood. We previously found that, during prolonged moderate exercise in the heat, restoration of $PaCO_2$ to the eucapnic level augmented the ventilatory sensitivity to rising T_{es} (slope of the T_{es}-V_E relation) by threefold (Hayashi et al. 2011). The present study thus examined the effect of $PaCO_2$ on the ventilatory sensitivity and cerebrovascular response to rinsing T_{es} at rest.

Methods

Fourteen healthy male were passively heated using hot-water immersion (41°C) and a water-perfused suit until T_{es} reached 39°C or the subjects could no longer tolerate the heating. During the heating on two separate occasions, subjects breathed room air (Control trial) or CO_2-enriched air (a mixture of room air and 100% CO_2) to prevent a reduction in P_{ETCO2} (an index of $PaCO_2$) (CO_2 trial).

Results and Discussion

T_{es} threshold for increase in V_E was seen at both trials (Control vs. CO_2: 38.4±0.4 vs. 38.1± 0.6°C, P = 0.13), and above the thresholds V_E increased linearly with rising T_{es}. In Control trial P_{ETCO2} declined gradually with rising T_{es} above the threshold, whereas it in CO_2 trial remained at eucapnic level. Ventilatory sensitivity to increasing T_{es} did not differ between Control and CO_2 trials (38.1±43.1 vs. 16.5±11.1 L/min/°C, P = 0.15). These suggest that ventilatory response with rising core temperature at rest is unaffected by a decrease in $PaCO_2$. Middle cerebral artery mean blood velocity ($MCAV_{mean}$) decreased gradually with rising T_{es} in Control trial (59.9 ± 8.4 and 49.9 ±9.7 sm/s at T_{es} 36.8 and 38.8°C, respectively), and the reduced $MCAV_{mean}$ was restored by about 40% in CO_2 trial (53.8 ± 5.6 cm/s at T_{es} 38.8°C), but inhaling CO_2 had no significant effect on $MCAV_{mean}$. Furthermore, the restoration of $MCAV_{mean}$ by CO_2 inhalation was similar among each T_{es} revel of 37.2, 37.6, 38.0, 38.4 and 38.8°C. These suggest that the decrease in $PaCO_2$ accounts for the reduction in $MCAV_{mean}$ by 40% at T_{es} range of 37–39°C during passive heating at rest.

References

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