CONTROL OF ARTERIAL BLOOD PRESSURE DURING HYPOCAPNIC HYPERVENTILATION

Anna Jaskot\textsuperscript{1}, Miroslaw Latka\textsuperscript{1}, Tadeusz Sebzda\textsuperscript{2}

\textsuperscript{1} Department of Biomedical Engineering, Wroclaw University of Science and Technology, Wybrzeże Wysiańskiego 27, 50-370 Wroclaw, Poland. anna.jaskot@pwr.edu.pl

\textsuperscript{2} Department of Pathophysiology, Wroclaw Medical University, ul. K. Marcinkowskiego 1, 50-368 Wroclaw, Poland.

The effects of hyperventilation on baroreflex sensitivity (BRS) have direct relevance to understanding of cardiovascular control during exercise, hypoxia and hypercapnia. There is a controversy concerning the role of hyperventilation in the pathological event chain leading to vasovagal syncope. The previous studies demonstrated impairment of transfer function arterial baroreceptor gain during prolonged (10 min) isocapnic hyperventilation. Herein we examine cardiovascular control during short maximal voluntary exercise-like hypocapnic hyperventilation in 9 healthy, young volunteers. To determine BRS we measured shortening of RR intervals which followed transient arterial blood pressure falls induced by postural change (sit-stand). The experiments were performed for spontaneous breathing and 120s hyperventilation. In the baseline conditions, the postural change resulted in the drop of systolic blood pressure (SBP) 34 ± 14 mmHg and the compensating shortening of RR interval by 222 ± 71 ms. Hyperventilation increased SBP drop to 47 ± 13 mmHg ($p=0.008$) and decreased change in RR intervals 148 ± 58 ms ($p=0.01$). Consequently, group averaged BRS decreased 55% from 7.3 ± 2.8 ms/mmHg to 3.3 ± 1.2 ms/mmHg ($p=0.0009$). We argue that BRS is dependent on heart rate and discuss the implication of this hypothesis for understanding of pathophysiology of vasovagal syncope.