Attenuated Cerebral Vasodilatory Capacity in Response to Hypercapnia in Young Obese Individuals

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Category: Masters

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ABSTRACT

Obese individuals are at a greater risk for the development of a variety of cardio and cerebral vascular diseases including hypertension, atherosclerosis, coronary artery disease and stroke. Furthermore, obesity is associated with cognitive impairment and is a risk factor for dementia and Alzheimer's disease. The exact mechanisms of this elevated risk are not fully characterized; however, impaired microvascular function is believed to be a contributor. This study tested the hypothesis that the cerebral vasodilatory capacity in response to hypercapnia is reduced in obese individuals relative to age and sex matched lean counterparts. Cerebral blood velocity (CBFV) was measured using transcranial Doppler before and during rebreathing-induced hypercapnia in obese (Obese, n=14) and lean (Lean, n=14) subjects. Cerebral vascular conductance (CVCI) was calculated as CBFV / mean arterial pressure (MAP), and a four parameter logistic regression was applied for sigmoidal curve fitting of the relationship between % change in CVCI and end-tidal CO_2 tension ($PETCO_2$). The magnitude of hypercapnia (Δ $PETCO_2$) during rebreathing was similar between groups (Obese 14 ± 3 mmHg vs. Lean: 15 ± 2 mmHg; $P = 0.13$). The maximum increase in CVCI (Obese: 155 ± 17% vs. Lean: 176 ± 23%; $P < 0.05$) and the total range of change in CVCI (Obese: 50 ± 15% vs. Lean: 75 ± 22%; $P < 0.01$) during rebreathing were reduced in the obese relative to the lean individuals. These data indicate that cerebral vasodilatory capacity in response to changes in $PETCO_2$ during hypercapnia is attenuated in obese individuals compared with lean individuals.

This study was supported by institutional start-up funds awarded to R. Matthew Brothers.