

Cerebral Vasomotor Reactivity: Impact of Hyperventilation Induced Hypocapnia on Hypercapnia Induced Vasodilation.

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Category: Doctoral

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Cerebral vasomotor reactivity (CVMR; the relationship of cerebral blood flow responses to changes in arterial carbon dioxide (PCO₂)) is used to assess cerebral vascular function. CVMR is generally assessed during either steady-state or transient breathe-by-breathe changes in PCO₂. In the transient method, a brief period of hyperventilation hypocapnia is often followed by hypercapnic rebreathing to induce a wide range of change in PCO₂ to assess the capacity of CVMR. Assessment of CVMR during transient changes in PCO₂ has been reported to be about 30% lower than that under steady-state conditions. One possibility for this discrepancy is that cerebral vasoconstriction during hyperventilation immediately before rebreathing may attenuate cerebral vasodilation and thus, CBF responses during the subsequent hypercapnic rebreathing.

PURPOSE: To test the hypothesis that CVMR during rebreathing hypercapnia would be blunted when the rebreathing is preceded by a period of hyperventilation.

METHODS: End-tidal carbon dioxide (PETCO₂), middle cerebral artery blood velocity (CBFV), cerebral vascular conductance (CVCI; CBFV/mean arterial pressure) and CVMR (slope of the linear regression between changes in CBFV and CVCI vs. PETCO₂) were assessed in 8 healthy individuals. CVMR was assessed twice, once immediately following a brief period of hyperventilation (hyperventilation for 1 min + rebreathe 4 min) and once during rebreathing only (rebreathe). The trials were counterbalanced and randomized with a minimum of 15 min elapsing between the protocols.

RESULTS: The slope of the linear regression for CBFV vs. PETCO₂ was reduced during hyperventilation + rebreathe relative to rebreathe only (hyperventilation + rebreathe: 3.16 ± 0.97 % / Torr, $R = 0.93 \pm 0.05$; rebreathe: 5.03 ± 1.11 % / Torr, $R = 0.83 \pm 0.25$; $P < 0.01$). Likewise, the slope of the linear regression for CVCI vs. PETCO₂ was reduced during hyperventilation + rebreathe (hyperventilation + rebreathe: 2.58 ± 0.89 % / Torr, $R = 0.88 \pm 0.09$; rebreathe: 4.34 ± 1.07 % / Torr, $R = 0.89 \pm 0.08$; $P < 0.01$).

CONCLUSION: These results indicate that hyperventilation induced hypocapnia attenuates the cerebral vasodilatory responses during a subsequent period of rebreathing induced hypercapnia.