**TACSM Abstract**

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

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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 + rebreathe) 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 ± 0.05; rebreathe: 5.03 ± 1.11 % / Torr, R = 0.83 ± 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 ± 0.09; rebreathe: 4.34 ± 1.07 % / Torr, R = 0.89 ± 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.