Respiratory alkalosis

It is unknown whether respiratory alkalosis impacts the global cerebral metabolic response as well as the cerebral pro-oxidation and inflammatory response in passive hyperthermia.

A study demonstrated that the cerebral metabolic rate was increased by ~20% with passive hyperthermia of up to +2°C oesophageal temperature, and this response was unaffected by respiratory alkalosis. Additionally, the increase in cerebral metabolism did not significantly impact the net cerebral release of oxidative and inflammatory markers. These data indicate that passive heating up to +2°C core temperature in healthy young men is not enough to confer a major oxidative and inflammatory burden on the brain, but it does markedly increase the cerebral metabolic rate, independently from the PaCO2.

There is limited information concerning the impact of arterial PCO2 /pH on heat-induced alteration in cerebral metabolism, as well as on the cerebral oxidative/inflammatory burden of hyperthermia. Accordingly, Bain et al. sought to address two hypotheses, that; 1) passive hyperthermia will increase the cerebral metabolic rate of oxygen (CMRO2) consistent with combined influence of Q10 and respiratory alkalosis; and 2) the net cerebral release of pro-oxidative and pro-inflammatory markers will be elevated in hyperthermia, particularly in poikilocapnic hyperthermia. Healthy young men (n = 6) underwent passive heating until an oesophageal temperature of 2°C above resting. At 0.5°C increments in core temperature, the CMRO2 was calculated from the product of cerebral blood flow (ultrasound) and the radial artery-jugular venous oxygen content difference (cannulation). Net-cerebral glucose/lactate exchange, and biomarkers of oxidative and inflammatory stress were also measured. At +2.0°C oesophageal temperature, arterial PCO2 was restored to normothermic values using end-tidal forcing. The primary findings were; 1) while the CMRO2 was increased ($P < 0.05$) by ~20% with hyperthermia of +1.5°C to +2.0°C, this was not influenced by respiratory alkalosis, and 2) although biomarkers of pro-oxidation and pro-inflammation were systemically elevated in hyperthermia ($P < 0.05$), there were no differences in the trans-cerebral exchange kinetics. These novel data indicate that passive heating up to +2°C core temperature in healthy young men is not enough to confer a major oxidative and inflammatory burden on the brain, despite it markedly increasing CMRO2, which is irrespective of arterial pH [1)].

1)
Bain AR, Hoiland RL, Donnelly J, Nowak-Flück D, Sekhon M, Tymko MM, Greiner JJ, DeSouza CA, Ainslie PN. Cerebral metabolism, oxidation, and inflammation in severe passive hyperthermia with and without respiratory alkalosis. J Physiol. 2020 Jan 3. doi: 10.1113/JP278889. [Epub ahead of print] PubMed PMID: 31900940.