MRI Measurements of ICP in Simulated Acute Mountain Sickness (AMS)

Justin Stevan Lawley[1], Noam Alperin[2], Sang H Lee[2], Paul Gerald Mullins[3], Samuel James Oliver[1], and Jamie Hugo Macdonald[1]
[1]*Health and Exercise Sciences, Bangor University, Bangor, Gwynedd, United Kingdom,* [2]*University of Miami, Miami, FL, United States,* [3]*Bangor Imaging Center, Bangor University, Bangor, Gwynedd, United Kingdom*

Introduction

Prolonged exposure to high altitude is often followed by the development of AMS. Symptoms include headache, nausea and vomiting, fatigue and sleep disturbances [1]. The prevalence of AMS increases with rate of ascent, total altitude gain and irrespective of altitude (2500 – 4500 m), a striking individual susceptibility, thought to be the result of a predisposition to intracranial hypertension at altitude [2]. Despite the elegance and longevity of this hypothesis, evidence for elevated intracranial pressure (ICP) in concert with AMS is rare. Using a novel magnetic imaging technique (MR-ICP) [3], we hypothesised that the changes in ICP after 10 hours in a hypoxic environment (simulated 4500 m) would be correlated with the severity of AMS.

Methods

A total of 13 male (mean age = 26) volunteers took part after informed consent was attained.. This study was approved by the Bangor University Ethics Committee. Participants where scanned after 10 hours of exposure to normoxia (21% O_2) and hypoxia (12% O_2) on two separate days (at least 5 days between each condition). Acute mountain sickness was measured using the Lake Louse Questionnaire and ICP was estimated by MRI using a 3 tesla MRI scanner (Achieva, Philips Healtcare). MRI data suitable for analysis was available from 10 subjects. MRICP is estimated from a measurement of the change in the intracranial volume and pressure that occurs during the cardiac cycle. These were derived from volumetric flow rates of arterial inflow, venous outflow, (i.e., internal carotid and vertebral arteries), (internal jugular and secondary veins), and cerebral spinal fluid (CSF) into and from the cranium, using a high VENC (70 cm/sec) and low (7cm/sec) VENC, respectfully. Constant imaging parameters for all VENC images include FOV 14 cm, slice thickness 5 cm, and minimum TR and TE with a flip angle of 20 degrees. All MR-ICP sequences were obtained in duplicate. Values are means (SD) whereby statistical comparisons between normoxia (10h) and hypoxia (10h) were conduced by paired t-test and the relationship between AMS and ICP was analysed by pearsons r.

Results

As expected, hypoxia reduced oxygen saturation by 18% ($P < 0.01$, n = 10), increased heart rate by 18 beats/min ($P < 0.01$, n = 10), increased mean arterial pressure by 8 mmHg ($P < 0.01$, n = 10) and increased AMS by 3 points ($P < 0.01$, n = 10). Overall hypoxia had no effect on cerebral blood flow (normoxia, 1046.75(178.69) *versus* 989.55(192.16) ml/min, $P = 0.45$, n = 10) ICP (Normoxia, 10.29(3.11) *versus* hypoxia, 12.39(4.82) mmHg, $P = 0.29$) or cerebral perfusion pressure (normoxia, 70.08(8.31) *versus* hypoxia 76.22(9.81), $P = 0.14$, n = 10). However, the change in ICP correlated with the severity of AMS (r = 0.85, $P < 0.01$, n = 10, see figure 1). An example blood and CSF flow rates waveforms from a subject who demonstrated significant increase in ICP from normoxia to hypoxia, from 9.4 to 19.2 mmHg, is shown in Fig. 2

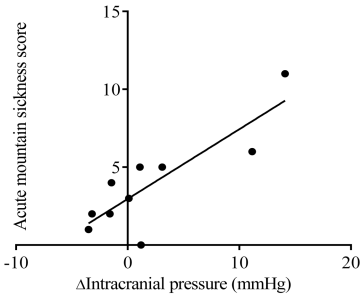

Fig. 1. Relationship between the change in ICP and acute mountain sickness score after 10 hours in hypoxia (12% O_2).

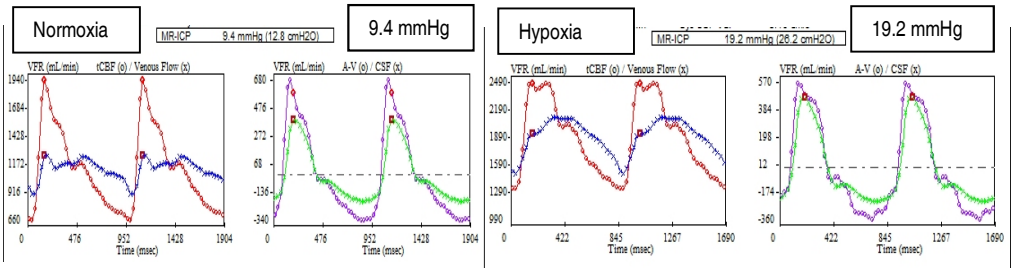

Fig 2. Blood and CSF flow rates waveforms demonstrating a lower intracranial compliance (higher ICP) at the hypoxic state (right graph). Higher CSF pulsatility relative to the arterial-venous flow (A-V) (right), i.e., the driving force for CSF flow, indicates lower intracranial compliance.

Conclusions

This data suggests that changes in ICP during 10 hours of exposure to a hypoxic environment are related to the severity of AMS. Future research examining the mechanism responsible for the susceptibility to intracranial hypertension and AMS will likely improve the treatment of altitude illness and advance our knowledge surrounding the effect of hypoxia on the brain.

References

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