

The Effect of Hypoxia on Cerebral Arterial Calibre and Flow Velocity - An MRI Study

Ravjit Singh Sagoo¹, David Bailey¹, Sarah Wayte², Eddie Ng'andwe¹, Charles Handford³, Sanjoy Nagaraja¹, Mahmud Saedon⁴, Helen Parsons⁵, Alex Wright^{6,7}, Arthur Bradwell^{6,7}, Christopher Imray^{4,7}, and Charles Hutchinson^{1,8}

¹Department of Imaging, University Hospitals Coventry and Warwickshire NHS Trust, Coventry, West Midlands, United Kingdom, ²Department of Medical Physics, University Hospitals Coventry and Warwickshire NHS Trust, Coventry, West Midlands, United Kingdom, ³University Hospitals Birmingham NHS Foundation Trust, Birmingham, United Kingdom, ⁴Department of Surgery, University Hospitals Coventry and Warwickshire NHS Trust, Coventry, West Midlands, United Kingdom, ⁵Division of Health Sciences, Warwick Medical School, University of Warwick, Coventry, West Midlands, United Kingdom, ⁶University of Birmingham, Birmingham, United Kingdom, ⁷Birmingham Medical Research Expeditionary Society, Birmingham, United Kingdom, ⁸University of Warwick, Coventry, West Midlands, United Kingdom

Target Audience

Radiologists, neurosurgeons, neurologists, and physicians with an interest in basic physiology.

Purpose

An adequate supply of oxygen is essential for normal cerebral function. It is believed that one of the processes responsible for maintaining oxygen delivery in high altitude climbers is an increase in cerebral arterial flow velocity. Cerebral arterial dilatation has long thought to play no part in maintaining cerebral oxygenation. Recent studies have disputed this theory and suggest that cerebral vasodilatation also contributes to the maintenance of oxygen delivery in hypoxic conditions¹. In addition, studying these cerebrovascular effects might also provide new insights into the pathophysiology of acute mountain sickness (AMS) and high altitude cerebral oedema (HACE). The aims of this study were to document changes in middle cerebral artery (MCA) calibre, flow and calculated oxygen delivery in response to normobaric hypoxia over a 22-hour period.

Methods

Subjects (n=12) underwent 3-T MRI scanning (GE HDxt, Milwaukee USA) at baseline (FiO₂=21%) and at 2, 4, 6, 11 and 22 hours of continuous normobaric hypoxia (FiO₂=12%, approximately equivalent to an altitude of 4,400m). A 3D time of flight MRA was performed to enable measurement of MCA cross-sectional area and diameter (figure 1). A sagittal 2D phase contrast MRA was performed to enable estimation of the MCA flow velocity (figure 2). Arterial oxygen saturation (SaO₂) was measured after each scanning session using a near infrared finger pulse oximetry probe. Blood samples were obtained at baseline, 11 and 22 hours to provide measurements of haemoglobin (Hb). Blood oxygen content was calculated as the product of the SaO₂ and Hb. MCA flow was calculated as the product of the MCA area and flow velocity. Oxygen delivery was calculated as the product of the blood oxygen content and MCA flow velocity. Paired t-test was used to assess the significance of changes in the variables at each time point against the baseline. Significance was set at <0.05.

Results

10 subjects completed the 22-hour study and 2 subjects dropped out due to symptoms after 11 hours. MCA flow and diameter trends are displayed in figures 3 and 4, respectively. Mean MCA flow velocity significantly increased by 21.1% between baseline and 2 hours (47.5cm/s to 57.5 cm/s, p<0.05) before gradually decreasing towards baseline levels. A similar trend was observed for mean MCA flow (4.5 ml/s at baseline and 5.5 ml/s at 2 hours, p<0.05). Mean MCA diameter appeared to increase between baseline and 6 hours by 3.9% (3.10mm to 3.22mm) before decreasing, although this rise was not statistically significant (p=0.09). Blood oxygen content decreased from 20.2 ml O₂/dl blood at baseline to 18.3 ml O₂/dl blood at 22 hours (p<0.001), whereas cerebral oxygen delivery did not alter over the 22 hours.

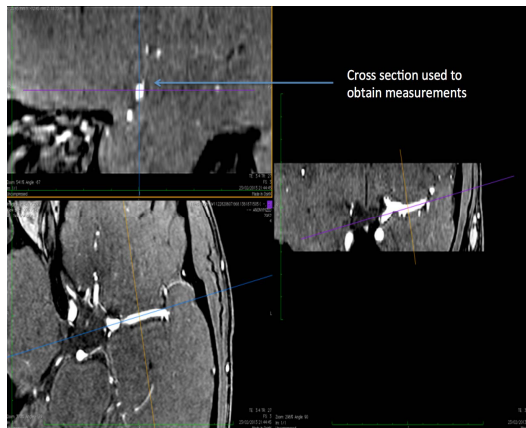


Fig 1: Orthogonal MPR of a 3D time of flight MRA used to measure MCA diameter and cross-sectional area

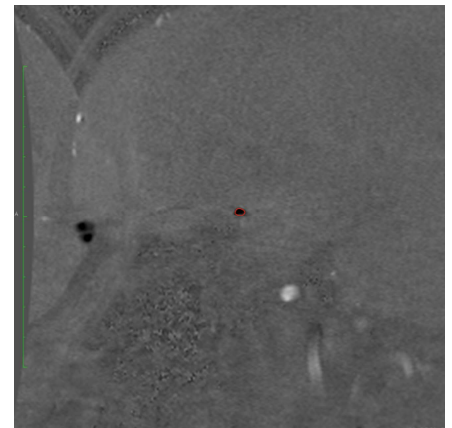


Fig 2: Sagittal 2D MRA with region of interest encircling the MCA (red) to estimate MCA flow velocity

Mean MCA Flow

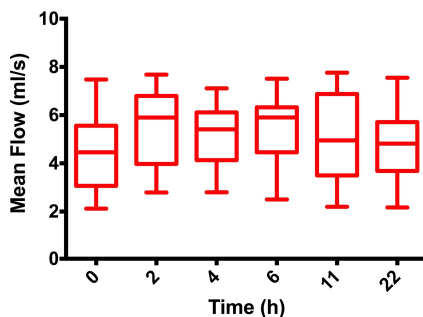


Fig 3: Box plot (2.5-97.5 percentile) displaying the changes in MCA flow with time

MCA Diameter

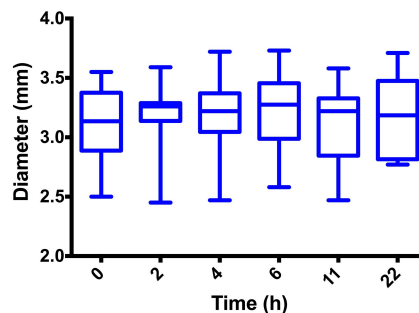


Fig 4: Box plot (2.5-97.5 percentile) displaying the changes in MCA diameter with time

Conclusion

This study confirms findings from other studies that hypoxia causes an increase in MCA flow velocity. This in turn contributes to an increase in MCA flow, which maintains oxygen delivery despite a drop in blood oxygen content. It was also observed that the rise in MCA diameter approached significance. A repeat study with a larger number of subjects may be needed to detect significance in the early rise in MCA vasodilatation. This would lead to a reappraisal of the effects of MCA diameter on maintenance of cerebral oxygenation.

References

1. Wilson, M, Edsell M, Davagnanam I et al. Cerebral artery dilatation maintains cerebral oxygenation at extreme altitude and in acute hypoxia - an ultrasound and MRI study. J Cereb Blood Flow Metab. 2011;31(10):2019-29.