Effect of hypobaric pressure on MRI parameters, including B0, T2, T2*, and T1

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Target Audience: neuroscientists and physiologists, researchers in altitude sickness, relaxometry

PURPOSE: High altitude sickness (experienced by mountain climbers and U2 pilots) could lead to a broad spectrum of disorders in the brain and other organs. For the brain, they include headache, irritability, dizziness, vomiting, confusion, etc. White matter hyperintensity, metabolic changes, and brain swelling among others, associated with acute and chronic high altitude exposure have been reported [1]. These previous studies investigated high altitude sickness post exposure. It would be interesting to study how the brain responds and adapts to acute high altitude, where air pressure and oxygen are low. In this work, we constructed an MRI-compatible hypobaric chamber for animal MRI scanner and performed B0 and relaxation time measurements during acute hypobaric exposure (approximately equivalent to an altitude of 3000m). In addition, we also modulated the oxygen content to mimic oxygen supplementation often used at high altitude.

METHODS: A custom-made hypobaric chamber was constructed for use in the MRI scanner, consisting of a cradle for the animal which slid into a PVC pipe and was then sealed on both ends. Cables of the coils, gas lines, and lines of physiological monitoring equipment were passed through tight fitting holes on the two ends of the chamber and sealed with silicone sealant as needed. The chamber pressure was reduced with a vacuum pump to 0.67±0.02 atm absolute (equivalent to an altitude of ~3000m). A separate gas line with a nose cone was used to deliver air and/or oxygen to the animal. A vent in the chamber allowed fresh air flow.

Male Sprague-Dawley rats (n=4, 286-445g) were anesthetized with 1.5g/kg urethane i.p. and imaged under spontaneous breathing conditions. Arterial oxygen saturation, heart rate, and respiration rate were monitored and rectal temperature maintained at 37^{o}C. Animals were imaged under 1) normobaric air, 2) hypobaric air, and 3) hypobaric oxygen (air+O2 mixture to maintain O2 saturation at comparable levels as normobaric air).

MRI was performed at 7T with a 2cm surface coil. B0 field/frequency shift was measured with non-localized water spectroscopy and B0 maps acquired with 3D multi-gradient echo with FOV=25.6x25.6x30mm, matrix=64x64x75, TR=20ms, TE=2.35 and 5.20 ms. T1, T2, and T2* were measured from axial slices, with FOV=25.6x25.6mm, and matrix=96x96. Other parameters were, T1: inversion-recovery EPI, TR/TE=10,000/9.86ms, 10 TIs from 23-3623ms, and seven 1.5mm slice; T2: multi-echo RARE, TR=3s, effective TE=18, 54, 90, 126ms, and seven 1.5mm slices; T2*. multi-gradient echo, TR=1.5s, 10 TEs from 3.1-22.9ms, and 14 0.75mm slices. Statistical analysis used paired t-tests with Bonferroni-Holm correction.

RESULTS: Water proton frequency under hypobaric conditions were slightly and significantly shifted negative relative to normobaric air (**Fig 1**). The frequency shift under hypobaric air and O2 were not significantly different. Cortical T2, T2*, and T1 under all 3 conditions are in **Fig 2**. T2 and T2* were significantly reduced under hypobaric air compared to normobaric air and hypobaric O2, which were similar to each other.

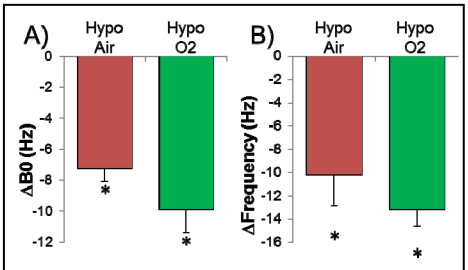

Fig 1. Changes in the main field due to hypobaric pressure. The shift in frequency in the brain relative to normobaric air from A) B0 maps and B) non-localized water spectroscopy. Error bars are SEM. *P<0.05 compared to normobaric air.

DISCUSSION & CONCLUSION: Decreased ambient O2 content during hypobaric conditions caused frequency/B0 shift, analogous to previous findings under normobaric O2 [2] and our previous findings under *hyper*baric pressure [3]. Slight spatial shifts in the phase encode direction of hypobaric conditions relative to normobaric air were noted in all images.

T2 and T2* under hypobaric air, causing hypoxia, were shortened due to the BOLD effects [4]. T2/T2* were unchanged under normoxic hypobaric pressure, indicating pressure itself does not affect them. Dissolved O2 acts as a paramagnetic agent shortening T1 [5]. We expected T1 may increase under hypobaric air due to lower blood and tissue oxygen, but T1 was not significantly altered herein. This is likely because the reduced dissolved O2 content due hypobaric air is relatively small.

In sum, hypobaric studies with and without oxygen supplementation have significant clinical and military relevance. This work sets the foundation for further MRI investigation in rodents under hypobaric conditions. Future studies will investigate the effects of hyperbaric pressure on cerebral blood flow, cerebrovascular reactivity, and evoked fMRI responses.

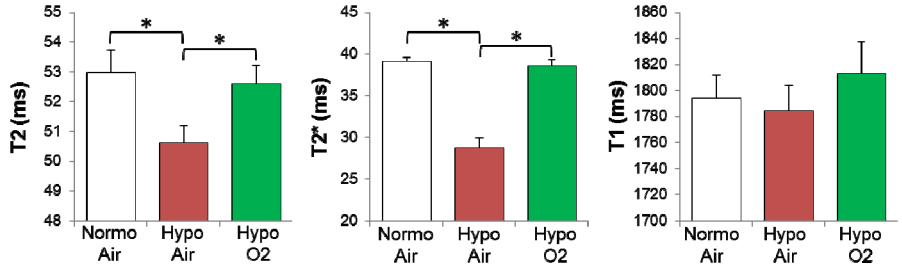

Fig 2. T2, T2*, and T1 in the cortex under normobaric air, hypobaric air, and hypobaric O2. Error bars are SEM. *P<0.05.

Reference: Hornbein. J Exp Bio 2001, 204:3129. 2) Pilkinton et al. MRM 2011, 66:794. 3) Muir et al. MRM 2014, 72:1176. 4) Uematsu et al. J Comp Assist Tom 2007, 31:662. 5) Shen et al. Brain Res 2011, 1425:132.