

Effect of hyperbaric oxygen on MRI including T1, T2, T2*, and Bo

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Target Audience: neuroscientists, researchers in oxygen therapy, relaxometry

PURPOSE: Hyperbaric oxygen (HBO) therapy has been used in the treatment of neurological disease such as stroke and traumatic brain injury, among others (1). A better understanding of brain physiology and function during HBO could lead to a better understanding of its therapeutic effects on disease. MRI is widely used to study brain function and physiology, but oxygen has several effects on MRI signals (as has been demonstrated at normobaric pressure (2-4)) which could impede the use of MRI. Dissolved oxygen is paramagnetic which should shorten T1 (2). Decreased deoxyhemoglobin under HBO should increase T2* and T2 due to the BOLD effect (3). In addition, Bo/frequency are shifted by O2, causing spatial drift of the image, unfavorable geometric distortions, and intensity fluctuations (4). The goal of this study is to investigate T1, T2, T2*, and Bo of the rat brain under HBO and compare to normobaric and hyperbaric air conditions.

METHODS: A custom-made hyperbaric 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 sealant as needed. Male Sprague-Dawley rats (n=13, 254-600g) were anesthetized with 1.5g/kg urethane i.p. and imaged under spontaneous breathing conditions. Respiration and heart rate were monitored and rectal temperature maintained at 37°C. The chamber was pressurized with air to 4 atm absolute. A separate gas line with a nose cone was used to deliver 100% oxygen to the animal. A vent in the chamber allowed fresh air flow and prevented buildup of high concentrations of high-pressure oxygen. Animals were imaged under normobaric air (NBAir), hyperbaric air (HBAir), and hyperbaric oxygen (HBO) conditions.

MRI was performed at 7T with a 2cm surface coil. Frequency shift was measured with non-localized water spectroscopy. Bo maps were acquired with 3D multi-gradient echo with FOV=25.6x25.6x30mm, matrix=64x64x75, TR=20ms, TE=2.35, 8.06 ms. T1, T2, and T2* were measured from seven 1.5mm thick 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 ranging from 23-3623ms, n=3-13; T2: multi-echo RARE, TR=3s, effective TE=25, 40, 75, 120ms, n=13; T2*: multi-gradient echo, TR=1.5s, 10 TEs ranging from 3.1-22.9ms, n=4-13. Statistical analysis used ANOVA and t-tests with Bonferroni-Holm correction.

RESULTS: Under NBAir, HBAir, and HBO pO2 is about 160, 638, and 3040 mmHg, respectively (assume 1atm~760mmHg and 21% O2 in air). Water proton frequency differences between HBAir-NBAir and HBO-HBAir were -6.8±2.1 and -21.7±10.5 Hz (whole brain), respectively (P<0.05 for both). Between NBAir and HBAir the largest differences were around the ear canals (Fig 1). Going from HBAir to HBO, large frequency changes occurred around the olfactory bulb, due to the delivery of oxygen locally to the nose through nose cone. Whole brain T1, T2, and T2* at NBAir were 1726, 52.4, and 35.4 ms, respectively. Differences in T1, T2, and T2* between NBAir, HBAir, and HBO in four ROIs are shown in Fig 2. The largest differences occurred going from NBAir to HBAir, rather than HBAir to HBO. Slight changes in position, shape, and dropout of the brain occurred between conditions, with the larger changes between NBAir and HBAir. The brain was shifted in the phase encode (dorsal-ventral) direction by ~1-1.8 pixels going from NBAir to HBAir and only by <0.1 up to 0.29 pixels from HBAir to HBO.

DISCUSSION & CONCLUSION: Increase in O2 partial pressure during HB condition caused frequency/Bo shift and increased inhomogeneity across the brain causing some change in position, shape, and dropout of images, similar to normobaric oxygen (4). HBAir and HBO shortened T1 due to dissolved paramagnetic O2 acting as a T1 shortening agent (2). T2 and T2* were lengthened due to the BOLD effects (3). Future studies will investigate the effects of HBO on cerebral blood flow and fMRI responses in stroke and traumatic brain injury.

Reference: 1) Bennett et al, Stroke 2010, 41:e185. 2) Shen et al, Brain Res 2011, 1425:132. 3) Uematsu et al, J Comp Assist Tom 2007, 31:662. 4) Pilkinton et al, MRM 2011, 66:794.

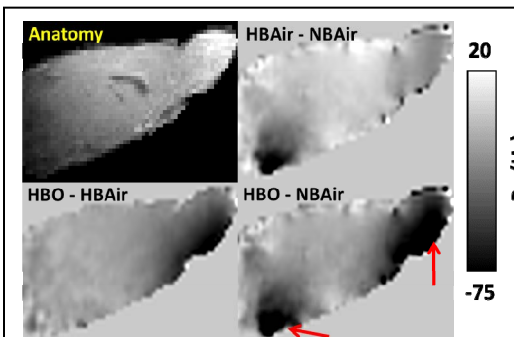


Figure 1. Bo difference maps between NBAir, HBAir, and HBO from a mid-sagittal slice with an anatomical image for reference. The largest changes were near the ear canals and olfactory bulb (red arrows).

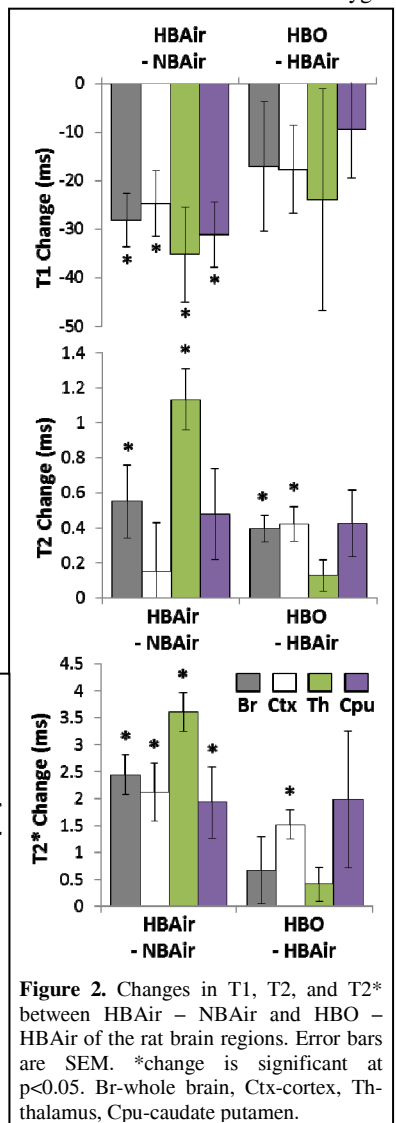


Figure 2. Changes in T1, T2, and T2* between HBAir - NBAir and HBO - HBAir of the rat brain regions. Error bars are SEM. *change is significant at p<0.05. Br-whole brain, Ctx-cortex, Th-thalamus, Cpu-caudate putamen.