

Oxygen Enhancement of CSF Demonstrated by FLAIR: Temporal and Spatial Characteristics

J. A. Butman¹, A. J. Rebmam¹

¹Diagnostic Radiology Department, National Institutes of Health, Bethesda, MD, United States

Introduction: Fluid attenuated inversion recovery imaging (FLAIR) suppresses signal from cerebrospinal fluid (CSF) based on T1 recovery. Failure to suppress CSF signal on FLAIR imaging has been noted in patients imaged under anesthesia^{1,2}. This can be attributed to the use of supplemental 100% oxygen. To characterize this phenomenon we performed FLAIR imaging using high temporal (EPI) and high spatial resolution (FSE) techniques during supplemental O₂ breathing in healthy volunteers.

Materials and Methods:

Subjects: Eighteen healthy volunteers (age range 26-67y mean 41y) gave written informed consent was obtained under an IRB approved protocol.

Breathing apparatus: A non rebreathing airway was established with a Y-connector attached to a mouthpiece. Wall oxygen at ~10 L/min O₂ (100% O₂) was delivered through one limb of the Y, and exhaled air exited the other limb. One way valves minimized dead space or recirculation. Nasal breathing was prevented with a nose clamp.

MR imaging: High temporal resolution FLAIR imaging was performed on a 1.5 T GE Signa scanner with 150 mT/m/s slew rate gradients using TR 10000 TI 2200 and TE 140 ms and a 2D SE-EPI readout with a 128 x 128 matrix and 20 cm FOV. Twenty-four contiguous 5mm axial slices were obtained every 30 sec for 20 minutes. At 5 minutes, ventilation was switched from room air (RA) to 100% O₂. At 12 min, ventilation was returned to RA.

High spatial resolution FLAIR imaging was performed using TR 10000 TI 2200 and TE 140 ms and a 21 ETL RARE readout with a 256 x 256 matrix and 20 cm FOV. Twenty-four contiguous 5mm axial slices were obtained every 4 min (240 sec) for 24 minutes. A total of 6 acquisitions were obtained sequentially, one on RA, 3 on 100% O₂ and the final two on RA.

Data Analysis: Spatial distribution of signal changes were analyzed with difference and ratio images. The time course of signal changes in various CSF and brain parenchymal compartments was analyzed with ROIs directed by anatomical location.

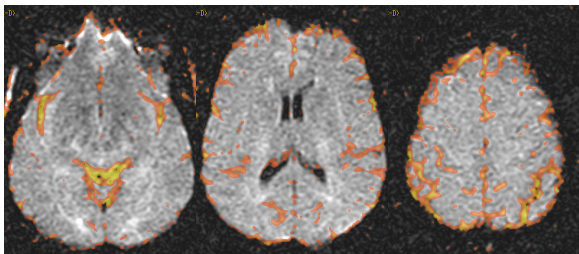


Fig. 1 Spatial distribution of O₂ induced signal changes in the CSF on FLAIR EPI. Note the absence of signal change in the ventricles.

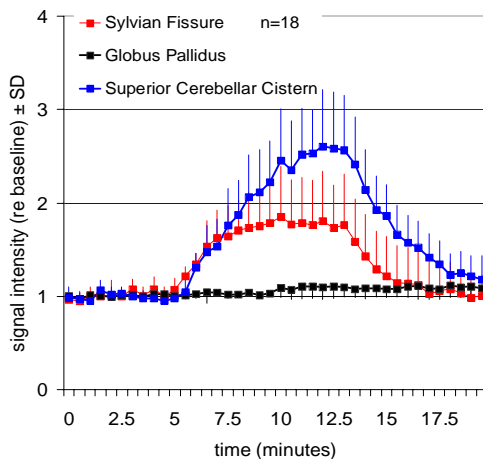


Fig. 2 O₂ induced signal intensity ratio change in different compartments (relative to baseline signal) measured by EPI-FLAIR. Note the absence of signal change in gray matter and the 2-2.5 fold signal change in CSF signal.

Results: Signal changes were confined to the CSF of the subarachnoid spaces (Figure 1). Signal changes of up to 260% were identified (Figure 2). No change was present in the intraventricular CSF. Within the subarachnoid compartments, the signal change in sulcal subarachnoid spaces clearly preceded that in the cisternal CSF spaces, but the cisternal spaces had a higher maximal signal intensity. (Fig.1)

Although a "global" ROI encompassing the whole brain demonstrated a 4% change in signal intensity, this could be attributed to volume averaging of included subarachnoid spaces. ROIs carefully restricted to gray matter or white matter regions did not demonstrate a consistent signal change in the presence of 100% O₂.

No signal changes were evident in arteries or veins. These retained a uniformly dark appearance. For example, the middle cerebral arteries appeared as flow voids within the hyperintense CSF of the sylvian fissure.

The global brain ROI revealed a two-component time course of signal change, a fast component which plateauing at ~ 2 minutes and a slow component that never saturated over the 8 minutes of oxygen breathing. Comparison to the spatially restricted ROI indicates that the rapid component reflects the sulcal subarachnoid CSF compartments immediately adjacent to the pia. The slow component reflects diffusion into cisternal spaces or into widened sulcal compartments. Following return to room air, CSF signal remained elevated in some compartments for over 10 minutes

Discussion: As hemoglobin saturation is essentially 100% at room air, breathing 100% O₂ results in a marked increase in dissolved O₂ with little change in the ratio of oxyhemoglobin and deoxyhemoglobin. This dissolved O₂ may then diffuse into the CSF from the densely vascularized pial surface of the brain. As O₂ is weakly paramagnetic, dissolved O₂ shortens the T1 relaxation time of CSF.¹ This alters the inversion time required to null CSF, resulting in failure of CSF suppression on the FLAIR sequence, and therefore high signal as the T2 of CSF is little affected.

Approximately 2 minutes are required to equilibrate alveolar pO₂ with inspired pO₂.³ This matches the initial time course of signal change in sulcal CSF indicating that there is a rapid equilibration of dissolved plasma O₂ with CSF adjacent to the pial surface. The slow increase in signal in larger CSF spaces (e.g. cisterns) reflects the slower processes such as diffusion of O₂ from the sulci as well as and bulk flow of CSF.

Enhancement of signal in the CSF in patients being ventilated with 100% O₂ has recently been recognized on FLAIR imaging as a potential artifact in the routine clinical setting.^{1,2} However, because ventilation of the lung, alveolar capillary oxygen exchange, perfusion of the pia, permeability of pial capillaries and diffusion of O₂ into the subarachnoid space all contribute to the time course and magnitude of signal changes measured by FLAIR, we propose that FLAIR signal of CSF may be a valuable probe of O₂ delivery to the underlying brain.

References:

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