CSF Suppressed Sodium Imaging of the Brain at 4.7 Tesla

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Introduction : Sodium MRI is complicated by the fact that the electric quadrupole moment of the spin 3/2 sodium nucleus gives rise to biexponential relaxation in the presence of local electric field gradients, such as those exhibited by cellular macromolecules, with a fast T2 decay component of a few ms *in-vivo* (1). To limit signal loss, 3D sequences, such as 3D-GRE and 3DPI, with short TE times have recently been used to acquire good quality single quantum sodium images at high magnetic fields of 3.0 and 4.0 Tesla (2, 3). Sodium imaging may be useful in the setting of acute stroke since the concentration of sodium ($[Na^+]$) has been shown to increase linearly in the ischemic region during acute stroke and a $[Na^+]$ of > 70mM may be an indicator of infarcted tissue (4, 5). This small increase from the normal $[Na^+]$ of ~ 45 mM in healthy gray matter may not be particularly conspicuous in peripheral brain regions since MR images of sodium in the brain are dominated by bright signal from cerebro-spinal fluid (CSF) because of its relatively high $[Na^+]$ of 145mM. The purpose of this study was to determine the viability of suppressing the signal contribution from sodium in CSF of healthy human brain using inversion-recovery (IR) at 4.7 Tesla.

Methods : Images were acquired using a 4.7 Tesla Varian Inova MRI scanner equipped with 35mT/m gradient strength, and an in-house designed and built 27cm diameter quadrature birdcage RF coil. A modified 3D gradient-echo sequence was used with a hard RF pulse for excitation, 50% asymmetric acquisition, and a TE of 1.65ms for each scan. To provide a basis for SNR comparison between inverted and non-inverted scans, all images, both phantom and *in-vivo*, were acquired with the same spatial parameters: FOV = 320mm x 320mm x 320mm, acquisition matrix = 42 x 42 x 20, resolution = 7.6mm x 7.5mm x 16mm. Signal nulling of CSF with inversion-recovery was achieved by preceding the 3D-GRE sequence with a 180° hard RF pulse and an inversion delay time of 33 ms {i.e. $\sim T_{1CSF}*\ln 2$}. The longitudinal decay curves of sodium in both saline and 5% agar were measured to be mono-exponential with T_1 values of 53ms and 25ms, respectively.

Preliminary experiments were performed on a 4L jug with two compartments: an outer annulus of 50mM $[Na^+]$ in 5% agar and a central 250mL solution with 150mM saline. The macromolecular matrix of 5% agar was used to generate the biexponential relaxation Na^+ would experience within brain tissue (6) whereas the saline solution was used to represent CSF. Phantom image comparison was carried out using the same repetition time (TR = 188ms), the same excitation flip angle of 90°, and the same number of averages (NEX = 10) for both the normal 3D-GRE sequence and the IR sequence, to determine signal loss directly related to inversion-recovery. The scan times for both were 25 mins. *In-vivo* images were acquired from a healthy volunteer. The normal 3D-GRE image, for the *in-vivo* case, was acquired using a shorter TR of 30 ms, a flip angle of 70° to maximize SNR in brain tissue for a given scan duration, and 24 averages, which yielded a scan length of 10 min. The *in-vivo* IR scan used the same parameters as the phantom IR scan.

Results and Discussion : **Figure 1** displays sodium images from the phantom. The SNR of the saline in the center of the IR image was reduced to 10% of the saline SNR in the normal 3D-GRE sequence demonstrating the efficacy of the IR suppression pulse. A small signal contribution from saline Na^+ remained in the IR image at edge of the phantom due to B_1 inhomogeneity and an imperfect inversion pulse. The 5% agar experienced an SNR drop of 36% (SNR normal 3D-GRE = 56; SNR IR = 36) whereas the expected SNR drop using measured T_1 values is ~50%. A ringing artifact is apparent in all images, parallel to edges of abrupt signal change, and is due to Gibb's ringing and a small acquisition matrix size. **Figure 2** displays sodium images from a healthy brain *in-vivo*. In the normal 3D-GRE scan, SNR in the brain tissue was 45 while the SNR in the CSF was 105. CSF SNR is not 3x the SNR of brain tissue as the relative $[Na^+]$ might predict because of T_1 weighting in the short TR normal 3D-GRE sequence. In the *in-vivo* IR images, CSF SNR was reduced to < 4% of the CSF SNR in the normal 3D-GRE images. Brain tissue SNR was reduced to 14 in the *in-vivo* IR case. Although the SNR is much lower than that in the normal 3D-GRE brain tissue (SNR 45), much of the difference is due to the lower number of averages in the IR case (NEX 10) than the normal GRE case (NEX 24). SAR regulations limit the practical number of possible averages since the IR sequence TR must be longer than in standard GRE. Also, as mentioned in the phantom study, SNR loss is inherent in IR acquisitions. Finally, although the resolution is low and the images exhibit some ringing, CSF has been successfully suppressed making brain tissue $[Na^+]$ the dominant source of image contrast.

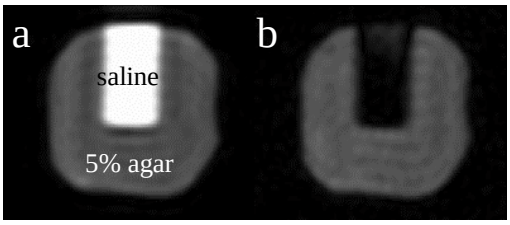

FIGURE 1 : Sodium images of two-compartment phantom {50mM Na^+ in 5% agar on outside / 150 mM Na+ in saline on inside}. (a) Without and (b) with inversion recovery to suppress the long T1 sodium species in the central saline.

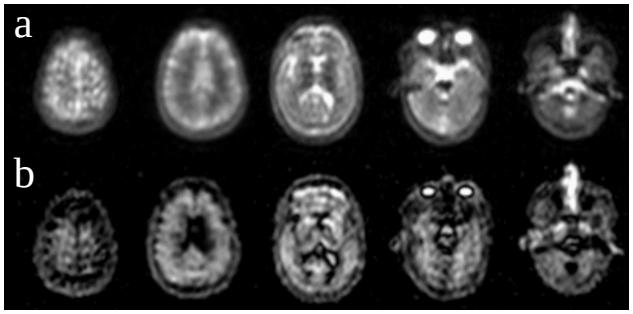

FIGURE 2 : Sodium images of five slices in healthy human brain at 4.7T. (a) Without (NEX=24) and (b) with (NEX=10) inversion recovery to suppress the sodium in the CSF.

References: [1] Van Emous et al, *MRM* 40: 679 (1998); [2] Boada et al, *MRM* 37: 706 (1997); [3] Clayton et al, *Acad Radiol* 10: 358 (2003); [4] Thulborn et al, *Radiology* 213: 156 (1999); [5] Wang et al, *Stroke* 31: 1386 (2000); [6] Jung et al, *JMR* 124: 393 (1997).

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