Sodium MRI of Core and Penumbra in ischemic stroke patients

A. Tsang[1], R. Stobbe[1], K. Butcher[2], S. Hussain[3], Y. Bhagat[1], D. Emery[4], and C. Beaulieu[1]

[1]Biomedical Engineering, University of Alberta, Edmonton, Alberta, Canada, [2]Neurology, University of Alberta, Edmonton, Alberta, Canada, [3]University of Alberta, [4]Radiology, University of Alberta, Edmonton, Alberta, Canada

Background and Purpose: Diffusion- and perfusion-weighted imaging (DWI/PWI) of acute ischemic stroke can be used to identify the ischemic core (DWI) and to estimate the penumbral tissue (PWI-DWI mismatch)[1] which is at risk of infarction but is potentially salvageable. MRI of sodium, a marker of ionic homeostasis that is disrupted after ischemia, within the lesion may be complementary by indicating regions of irreversible injury[2] or by providing an estimate of stroke onset time[3], the latter of which could be useful for unknown onset stroke patients. However, while sodium changes in the DWI-defined core have been evaluated previously, the relationship of relative sodium signal intensity to penumbral tissue has not yet been investigated. The purpose of this study is to evaluate tissue sodium in the penumbra and core infarct regions from acute to sub-acute times after stroke.

Methods: Four male stroke patients (age range 46-54 years) were examined both at acute (4-7h) and subacute (23-32h) timepoints after stroke onset using standard ^{1}H imaging including DWI/PWI on a 1.5T Siemens Sonata, followed by ^{23}Na imaging on a high field 4.7T Varian Inova. DWI was acquired with single-shot spin-echo diffusion EPI, nineteen 5-mm axial slices, 1.5mm gap, b = 1000 s/mm^2, TR/TE = 2.6s/86ms, GRAPPA R=2, and PWI was acquired using single-shot GE-EPI with i.v. injection of Gd-DTPA (5ml/s), thirteen 5-mm axial slices, 1.5mm gap, TR/TE = 1320ms/50ms. Sodium imaging used a steady-state projection acquisition sequence (Na-PASS)[4] with TR = 25ms, TE = 0.6ms, 55^{o} flip angle, 2.4x2.4x4.8mm^3 voxel size, 10 min acquisition time. A customized MATLAB tool called Penguin[5] implementing singular value decomposition was used to generate PWI maps of Tmax, cerebral blood flow and volume. PWI, DWI, and sodium images were co-registered using SPM5. Penumbral tissue was defined objectively as those regions with Tmax+4s delay[6] and no DWI abnormality. Relative sodium intensity was calculated as the ratio of signal intensities in core or penumbra to contralateral homologous regions.

Results: Mean acute DWI lesion (core) and PWI-DWI mismatch (penumbral) volumes were 28 ± 20 ml and 46 ± 31 ml, respectively. Sub-acutely, DWI lesion volumes increased to 45 ± 25 ml, while mismatch volumes decreased to 16 ± 15 ml. Sodium intensity appeared to be only elevated in the DWI core (Figure 1). As shown in Figure 2, the mean relative sodium intensity in the infarct core was slightly elevated (1.09 ± 0.11) acutely and then increased further (1.39 ± 0.27) subacutely. However, in mismatch regions relative sodium intensity was not significantly increased either acutely (1.01 ± 0.04) or sub-acutely (1.03 ± 0.07).

Conclusion: Relative tissue sodium intensity in ischemic stroke patients increased in a time-dependent fashion in infarct core regions, but was not elevated in the PWI-DWI mismatch penumbral region. Sodium MRI could potentially estimate the onset time based on the temporal change in the core of patients with unknown stroke onset. On the other hand, unchanged relative sodium intensity in the penumbra suggests that ionic homeostasis has not yet been compromised.

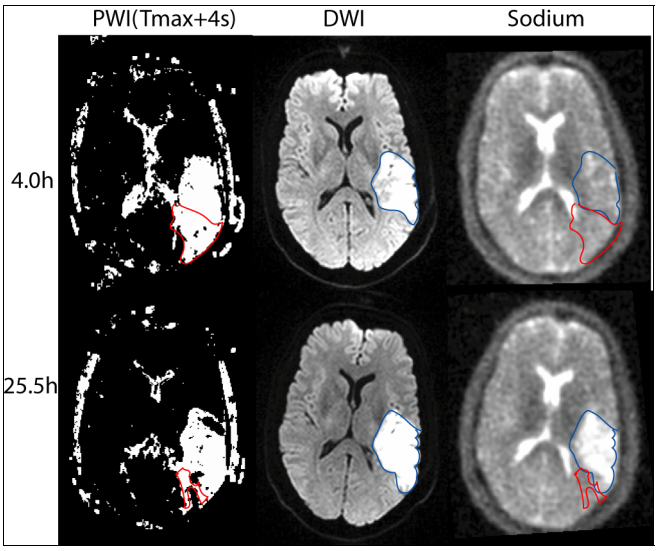

Figure 1 - Images of a 46 year old male stroke patient (P1) demonstrating elevation in relative sodium intensity in the DWI lesion core (blue outline) in the subacute time but no change in the penumbral region (red outline).

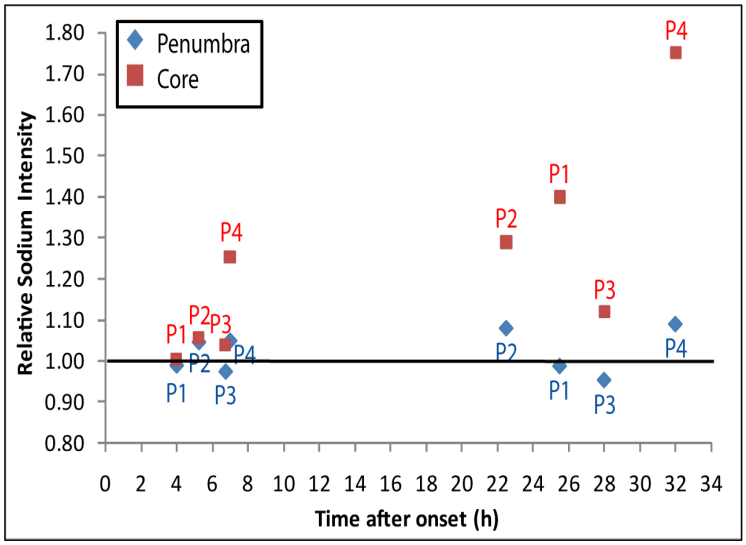

Figure 2 – Relative sodium intensity of the four stroke patients is slightly elevated acutely and more elevated subacutely in the DWI core whereas there is no increase in the PWI/DWI mismatch region.

References: [1]. N. Hjort, et al., *Stroke* 36:388(2005). [2]. K. Thulborn, et al., *Radiology*, 213:156(1999). [3]. MS Hussain, et al., *Stroke*, 39(2):580 (2008). [4]. R. Stobbe, et al., *Magn Res Med*, 59:345(2008). [5]. PErfusioN Graphical User INterface, CFIN, http://www.cfin.au.dk/. [6]. K. Butcher, et al., *Stroke* 36:1153 (2005).