Computation of ADC maps from FLAIR b=0 and DWI b=1000 reduces contamination by cerebral spinal fluid with minimal increase in scan time.

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Background: Large voxel sizes in DWI-EPI result in signal averaging of brain parenchyma with cerebral spinal fluid (CSF) and the contamination of ADC values of healthy and ischemic tissue. Such contamination can be mitigated by suppressing the unwanted signal from CSF through the use of FLAIR-DWI. However, FLAIR-DWI sequences require greater than four times longer to aquired as compared to conventional DWI. From the ratio of FLAIR T2 weighted to conventional T2 weighted images, it is possible to obtain an approximation of parenchymal volume fraction. We hypothesized that by estimating volume fraction using DWI T2 weighted and a FLAIR T2 weighted, we could reduce CSF contamination while at the same time significantly decrease overall scan time.

Methods: Eight patients were selected from our natural history stroke image database having both conventional and FLAIR DWI prior to therapy, lesions larger than 5 cc in the MCA territory, and an onset to scan time of less than 6 hours. Both sets of EPI images were aligned A/P and R/L and co-registered. An investigator (SW) blinded to clinical information manually defined lesion ROIs on conventional b=1000, and the ROIs were mirrored across midline to obtain homologous regions of contralateral tissue. ADC maps were generated by standard methods for the conventional and FLAIR DWI. A third ADC map was computed from the conventional DWI b=1000 image and the FLAIR b=0 image according the following equation:

$$ADC = -\tfrac{1}{b}\ln\left[k_0\,\tfrac{DWI}{FLAIR} - k_1\,\tfrac{k_0\lambda_{app}}{\lambda_{app}}\right]$$

where k_0 =0.85 is the average ratio of FLAIR to T2 signal intensity in the parenchyma (correcting for incomplete recover of parenchymal signal in inversion recovery), k_1 =0.05 is the factor that CSF is attenuated by diffusion weighting at b=1000, both kept constant for all voxels and patients, and λ_{app} is the ratio of FLAIR to DWI b=0 calculated on a per voxel basis. Maps of volume fraction, λ, were also estimated according to previously published methods. Only voxels having volume fractions of greater than 20% were considered in the analysis. The time savings was estimated from an optimized protocol currently being used to collect prospective data.

Results: Sequence times for DWI and FLAIR DWI were 24 and 108 seconds respectively. The addition of an EPI-FLAIR matched to DWI added 14 seconds to the prospective protocol. The potential time savings was estimated to be approximately a factor of three. Shown in Figure 1 are the DWI b=0, 1000, matching FLAIR-T2 images, and ADC maps from an acute stroke patient having a large lesion in the left MCA territory. Lesions were not evident on b=0, FLAIR, or volume fraction map (not shown). Demonstrated on the conventional ADC map are heterogeneously decreased ADC values in the lesion. CSF suppressed ADC maps and ADC maps calculated according to above equation appeared similar and less heterogeneous, allowing better contrast-to-noise and discrimination of lesion boundaries. In Figure 2 are scatter plots of ADC value *vs* volume fraction for all voxels in the lesion and matched contralateral ROIs for the three different ADC maps. ADC values from conventional DWI asymptotically increase toward the ADC of CSF with decreasing λ. ADC values calculated as above showed less of a dependence on λ, however lesion ADC values tended to be underestimated when compared to ADC values from FLAIR DWI.

Discussion: DWI b=1000 images are often preferred over ADC maps for the clinical diagnosis because of the higher contrast to noise and lesion conspicuity. CSF suppressed ADC maps from FLAIR-DWI have similar conspicuity at the cost of significantly longer acquisition times and decreased SRN. We have found that "pseudo" CSF suppressed ADC maps can be generated from conventional DWI though the addition of a matched FLAIR T2 weighted image. Lesion conspicuity was high, however further work is required to understand and reduce the remaining bias, including a robust method for estimating k_0 on a per patient basis. It is hoped that the increased discrimination between healthy and ischemic ADC values will help reduce the misclassification of tissue by automated methods.
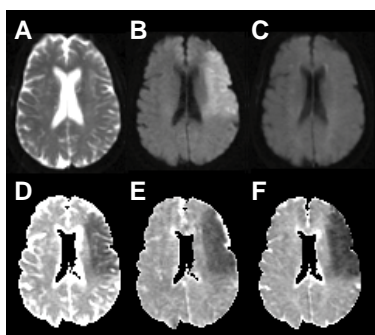

Figure 1. Axial slice from acute stroke patient. A) DWI b=0, B) DWI b=1000, C) FLAIR b=0, D) Conventional ADC, E) CSF suppressed ADC, amd F) CSF suppressed ADC calculating by substituting the DWI b=0 image with the FLAIR image.
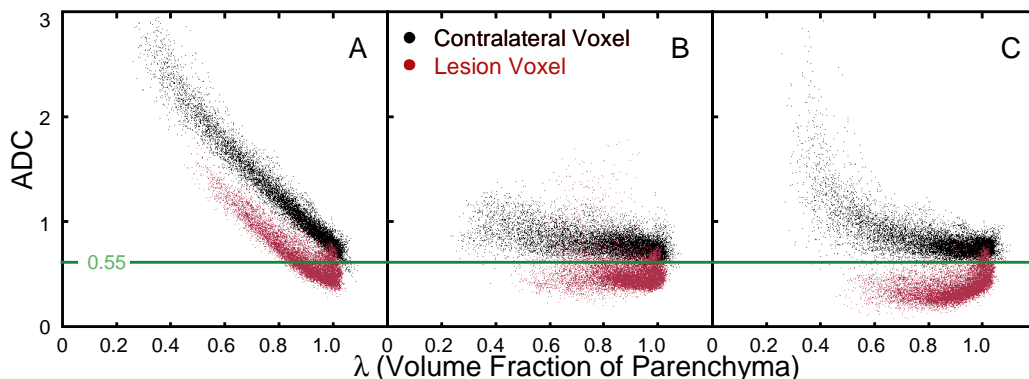

Figure 1. Scatter plots of ADC value for a voxel as a function of the volume fraction of parenchyma in that voxel obtained by A) conventional DWI, B) CSF suppressed DWI, and C) substituting convention DWI b=0 image with the FLAIR image. Data are from one patient, both lesion and contralateral homologous tissue. Line at ADC=0.55 indicates threshold found to be predictive of tissue outcome.