The b factor dependence of signal characteristics in dynamic ADC functional imaging

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Introduction: It has recently been proposed that dynamic changes in the apparent diffusion coefficient can be used as a contrast mechanism for functional brain imaging (1,2). Based on these previous reports, the ADC contrast is thought to originate within the arterial networks with decaying sensitivity downstream, based on the timing differences in BOLD and ADC time courses. In this report, we further investigate the signal differences in areas found to be active based on the ADC contrast with multiple degrees of diffusion weighting.

Methods: Five subjects participated in this study after giving informed consent. A typical visual activation fMRI paradigm was used with 30 s blocks of rest and activation over a 3.5 min acquisition. A temporally ramped isotropic diffusion weighted acquisition was used with three b factors of 2, 116, and 229 s/mm², incorporated into a gradient-recalled spiral imaging sequence. This cycle of three b factors was repeated 140 times. The initial b factor, when considered alone, produces a typical BOLD time course with TR=1.5 s. The initial b factors of 2 and 116 can be used to determine a low b factor ADC measure, while a high b factor condition can be determined from the b factors of 116 and 229. The two conditions will likely have different sensitivity to different vasculature. For each ADC time course, as well as for the BOLD time course, a multiple regression algorithm was used to determine active voxels. The three control-activation epochs were averaged and normalized in order to better determine timing differences. The ADC time courses were shifted appropriately to account for the differences in the acquisition timing.

Results and Discussion: All subjects showed dynamic ADC activity based on both the high b and low b conditions. The spatial extent of the low b activation was larger than for the high b condition. The low b ADC changes were measured to occur 1.02 s before the high b changes. The magnitude of the measured low b ADC was also significantly higher than the high b ADC (Fig 1). These results are consistent with the hypothesis that the low b signal stems from blood flow changes more upstream and in larger vessels than the high b ADC signal. This can be further confirmed by looking at the BOLD signals in these separate b factor compartments. The low initial b factor used in this study will reduce the signal received from larger and faster moving vessels. It was found that the BOLD signal in voxels undergoing high b ADC activation of any sort was significantly higher than in those with low b ADC activation alone. This further suggests that the high b ADC activation is from smaller capillaries, and that the low b ADC activation is from larger vessels. Combined with the timing information, the low b ADC activation likely results from vessels upstream from the high b ADC changes.



Figure 1: Measured ADC time courses for low b (broken line) and high b. Figure 2: Measured BOLD signal in voxels with high b ADC activation, low b ADC activation, and BOLD only activation (curves from top to bottom).

Conclusion: Our results indicate that significant differences are present in the signal characteristics derived from the dynamic ADC contrast mechanism with a range of b factors. These differences indicate that the ADC contrast can be sensitized to particular vascular pools. Manipulation of the b factor range used for the determination of the dynamic ADC signal should allow for the selective acquisition of changes localized to the capillary networks. This demonstrates a possible advantage of the dynamic ADC contrast over the BOLD contrast mechanism in functional imaging.

References: 1. Gangstead and Song, Magn Res Med 48:385-388, 2002; 2. Song et al, NeuroImage 17:742-750, 2002.