Improved MR Perfusion Quantification at 1.5T and 3.0T

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Introduction: Quantification of MR perfusion data presents several obstacles relating to delay and dispersion of the contrast (1). The appropriate selection of an arterial input function (AIF), the addition of a correction factor derived from the venous output function (VF) and the use of circular singular value decomposition (cSVD) techniques minimized some of these difficulties. We present data from both 1.5 Tesla and 3.0 Tesla MRI scanners utilizing various corrections to achieve more accurate measures of CBV and CBF.

Materials & Methods: A retrospective study of patients receiving MR perfusion weighted imaging as standard of care for cerebrovascular diseases including stroke and carotid artery steno-occlusive disease is presented. A total of 18 patients (15 Female/3 Male) with an average age of 62.4±4.7 SEM($\sigma^2/\sqrt{n}$) years were analyzed. The studies were performed on both GE 1.5 Tesla (n=8) and 3.0 Tesla (n=10) MRI scanners (GE Medical Systems, Milwaukee, WI) using gradient-echo echo planar imaging (GE-EPI) with the following acquisition parameters.

Field Strength	Matrix	Slice Thickness	Slices	TR/TE	Flip Angle
1.5 Tesla	128x128	8 mm	9	1.5 s/65 ms	90
3.0 Tesla	128x128	6 mm	12	2.2 s/30 ms	60

Protocols were varied clinically between field strengths to account for variances in T_1 as well as to optimize the number of slices and reduce the slice thickness utilizing the increased signal at 3.0 Tesla. The contrast injection was a double dose (0.2 mM/kg) of gadolinium contrast (Omniscan), (Amersham, Princeton, NJ) injected at 5 cc/s with a 10 second delay. Immediately following, a saline chaser of 25 cc was injected at 5 cc/s.

Analysis of the perfusion data was performed on a Dell 2.4 GHz PC using a software program written in IDL 6.0 (RSINC/Kodak, Boulder, CO). Single voxels were placed by a radiologist (PCS) on the internal cerebral artery (ICA) and sagittal sinus (VF). Characteristic single voxel time intensity curves (TIC) from each field strength are shown in Figure 1. All TIC's were fit with a gamma variate function prior to SVD to eliminate oscillations within the calculation. Signal intensities were converted into relative concentrations assuming values of unity for R2 and TE. The AIF was scaled by the ratio of the areas of the AIF to the VF, creating a venous correction factor (VCF) [Eq 1]. Lin, et.al. proposed a similar correction factor normalizing the VF of each patient to the mean of the group (2). CBV was calculated using the accepted factor accounting for blood density and hematocrit [Eq 2]. CBF was calculated using circular singular value decomposition (cSVD) methods proposed by Wu, et.al. [Eq 3] (3). Data was zero filled to twice the number of time points prior to cSVD and a 20% cutoff value was used on the W-matrix. CBF was calculated using the maximum of the residual function, R(t) [Eq 4]. The MTT was taken as the ratio of CBV to CBF according to the central volume theorem.

$$VCF = \frac{\int VF(t)dt}{\int AIF(t)dt}[1] \quad CBV = 0.705\frac{ml}{g}\frac{\int C_m(t)dt}{VCF\int AIF(t)dt}[2] \quad C_t(t) = CBF \cdot VCF \cdot AIF(t) \otimes R(t)[3] \quad CBF = R_{\max}\frac{CBV}{\int R(t)dt}[4]$$

Results: A reduction in CBV and CBF due to the VCF was found to be 54.0±5.1% (SEM) at 1.5T and 30.0±4.7% (SEM) at 3.0T. These were found to be statistically different using a paired 2-tailed t-test with unequal variances (p=0.02). As both CBV and CBF are identically affected by the scaling factor, the MTT remained unaffected. In this study, the VF was always of greater height and area than the chosen ICA which is reflected in the reduction of CBV and CBF. The white matter CBF of all patients was 52.3±8.7 ml/100g/min [12.7 min, 143.5 max]. The white matter CBV of all patients was 5.7±0.6 % [2.8% min, 9.8% max]. The ratio of normal gray matter CBF to white matter CBF was 2.26±0.21 (SEM) which is similar to that as shown in O^{15} PET studies.

Discussion: The decreased VCF seen at 3.0T is caused by either a lower VF area or a higher AIF area than that at 1.5T. We are assuming a lesser partial volume effect for the AIF taken in the ICA in comparison with the MCA (4). The VF taken in the sagittal sinus is also oriented perpendicular to the imaging plane. Although we may hypothesize that the AIF is greater at 3.0T than 1.5T, comparison of the absolute areas of AIF and VOF between these two scan protocols may be precluded due to variances in slice thickness, flip angle, relaxivities and T_1,T_2 values.

Conclusion: MR perfusion weighted imaging at 1.5T and 3.0T was performed on patients as standard of care for various cerebrovascular disease states. The inclusion of a vascular correction factor, the choice of the ICA as an AIF and the use of cSVD all contribute to reduced values of CBV and CBF in standard MR perfusion protocols. The use of these factors leaves the MTT unaffected and may allow more accurate estimates of MR perfusion yielding greater utility and diagnostic value for this technique.

References: 1) Kiselev VG. Magn Reson Med 2001;46:1113-1122. 2) Lin W, Celik A, Derdeyn C, et.al. JMRI 2001;14:659-667. 3) Wu O, Ostergaard L, Koroshetz WJ, et.al. Magn Reson Med. 2003;50(4):856-64. 4) Rausch M, Scheffler K, Rudin M, Radu EW. MRI 2000;18:1235-1243.

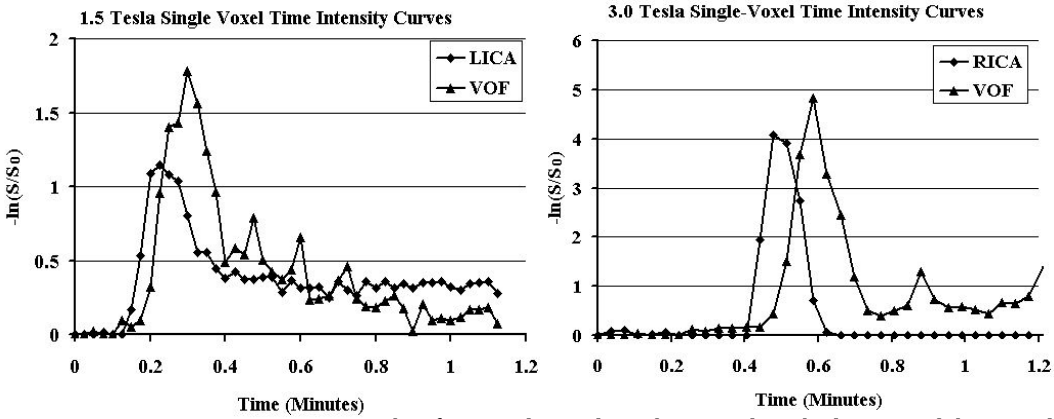

Figure 1: Representative time intensity curves taken from single voxels on the internal cerebral artery and the sagittal sinus.