Local Arterial Input Functions in DCE-MRI

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Introduction: Dynamic contrast-enhanced MRI (DCE-MRI) is a physiological imaging tool used clinically to aid the diagnosis and treatment monitoring of a variety of diseases. Pharmacokinetic parameters can be estimated by fitting DCE-MRI data to one of many mathematical models. The two-compartment model used here describes the concentration of contrast agent (CA) in tissue with respect to three parameters as well as the CA concentration in the plasma through the relationship:

\[ C_e(t) = K_{tr} C_p(t) \ast e^{-k_{ep} t} + V_{p} C_p(t) \]

where \( K_{tr} \) and \( k_{ep} \) are the transfer constant and rate constant respectively, \( \ast \) is the convolution operator, \( V_p \) is the blood plasma volume fraction, and \( C_p(t) \) is the concentration of CA in the blood plasma [1]. In many circumstances, the blood plasma concentration or arterial input function (AIF) is not easily measured, either due to small arterial lumen size when compared to voxel size, or due to the lack of any artery adjacent to the imaging region of interest. We and others have developed “blind” and “semi-blind” deconvolution algorithms for estimating the AIF directly from measured tissue curves. The blind estimation algorithm has been shown to provide good results with simulated and real data over a wide range of SNR. However, the input functions returned from tissue activity curves can be delayed and dispersed as compared to those measured directly from neighboring vasculature. Other researchers have shown that the input functions may change as the ROI moves away from major arteries into smaller vessels in the brain, and that locally measured AIFs may provide more accurate parameter estimates [2]. This work tracks changes in the locally estimated AIF in other regions of the body to support the hypothesis that CA delivery to diseased tissues may be more dispersed than that measured in the arteries.

Method: DCE-MRI data was obtained from 12 patients diagnosed with various soft-tissue sarcomas. Data was acquired on a Siemens 1.5T scanner using a FLASH 3D sequence with 20° flip angle, and TR ranging from 2.81-3.59 ms and TE ranging from 1.13-1.44 ms. 20mL of Omniscan was administered intravenously with a dose of 0.1-0.2 mmol/kg of body weight. Slices were selected from the data sets that contained regions of diseased tissue as well as arterial voxels. Multiple equally-sized regions of interest were defined across the data from the artery to the sarcoma tissue, as shown for one representative data set in Fig 1. The alternating minimization blind estimation technique as described in [3] was then applied to each of the individual regions of interest to obtain a single estimated AIF from each region. The model used in the blind estimation was modified from that described in [3] to contain a single gamma-variate curve and a sigmoid curve to describe the AIF. This was done to simplify calculation and speed up the estimation process. The simple model also served to reduce the likelihood of fitting noise with low SNR curves.

Results and Discussion: As seen in Fig. 2, as the blind estimation mask moves away from the artery into the tissue, the bolus peak height of the AIF decreases and the width increases. These trends continue as the mask moves further from the artery into the sarcoma, suggesting that the local concentration input to the sarcoma may be different than that measured in the artery. Similar results were obtained from other data sets. Studies in [3] show that kinetic parameters estimated from a more dispersed AIF have slightly higher \( K_{tr} \) and \( V_p \) values. A physiologically dispersed input function may be required to obtain more accurate parameter estimates. One of the open questions in blind estimation is the question of a global scaling constant. The method used here assumes that the CA concentration is the same in arteries and tumor vasculature after several minutes and thus scales the AIF by the concentration values over the last few data points. This scaling method may result in the differing bolus heights seen in Fig. 2.

References: