

Arterial Input Function Theoretical Calibration for First-Pass Quantitative Cardiac Perfusion Studies

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Introduction

The conversion of signal (S) to absolute Gadolinium-DTPA concentration [Gd] for first-pass contrast-enhanced quantitative perfusion studies continues to be problematic. We have developed an in vivo theoretical calibration method to convert the T1-weighted image signal to [Gd], which relies on rapidly measuring T1 during each heartbeat interval, and then converting the T1 values to [Gd] values using the known relaxivity of Gd. However, preliminary studies have indicated that for large [Gd] concentrations, the left ventricular cavity (LV cavity) signal curve (arterial input function or AIF) undergoes distortion or full saturation (clipping) due to nearly full or full recovery of blood magnetization, thus limiting the usefulness of the first-pass method for estimating absolute perfusion. We can ensure that the AIF is not saturated by deriving it from low resolution images acquired with short recovery delay (TD) [1], and we can convert the unsaturated AIF to an undistorted [Gd]-t curve with a theoretical method that eliminates the need for in vitro empirical calibration measurements.

Materials and Methods

We acquired serial T1-weighted images (Fig.1) with a modified fast gradient echo-echo planar imaging (FGRE-EPI) pulse sequence [2] with an interleaved center-out k-space acquisition, after a bolus injection (0.1 mmol/kg) of Gd-DTPA (Magnevist, Schering) at a rate of 6 ml/s. At the beginning of the scan, prior to the contrast arrival, we acquired a proton density-weighted image at each slice location, which we used to normalize each T1-weighted image. The normalization enables us to eliminate the equilibrium longitudinal magnetization and the receive coil profile. Considering longitudinal magnetization evolution only, we derived a theoretical expression (S(T1)) that relates the signal of the T1-weighted images obtained with such a sequence to T1 and to the known imaging parameters. T1 weighting was produced by playing a 90° saturation B1-insensitive rotation RF pulse (BIR-4) and by allowing partial recovery before the k-space read-out during the delay TD. We used the S(T1) expression to map average pixel signal values for selected regions of interest (ROIs) onto corresponding T1 values, and then converted the T1 values to [Gd] values using equation 1 [3].
$$\frac{1}{T_1} = \frac{1}{T_{1int}} + \gamma \cdot [Gd] \quad (1)$$

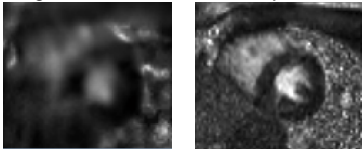


Fig. 1 Examples of low (left) and high resolution T1-weighted images; midventricular short-axis image

[Gd] = 0 (intrinsic blood T1), and γ is the relaxivity of Gd, taken to be equal to 3.8 L/mmol·s (data sheet from Magnevist, Schering AG). In order to eliminate the blood signal saturation problem, we rapidly acquired a low resolution image during the TD delay of each T1-weighted image acquisition. This low resolution image (phase encode lines = 24) has its own short TD_{LR} delay (TD_{LR} = 21.6 ms), which allows some signal recovery, but is short enough to prevent full longitudinal magnetization recovery even at high [Gd], where T1 can be of the order of 20 ms. The high resolution (phase encode lines = 88) T1-weighted images are acquired with TD = 46.9 ms. We performed in vivo imaging in a 3T MR Scanner (Trio, Siemens), using a phased-array RF coil. Other imaging parameters relevant for the theoretical modeling are: imaging flip angle = 10° for the T1-weighted images and 3° for the proton density-weighted image, TR = 6.7 ms and 4.2 ms for the high and low resolution images respectively, echo train length of 4. The images were processed in Matlab (The MathWorks, Natick, MA). For both low resolution images and high resolution images, we selected uncontaminated LV cavity ROIs and calculated the signal at each heartbeat interval as the average pixel intensity. The theoretical curve that relates S to [Gd] is shown in Fig. 2 for the low and high resolution images (generated with the corresponding image parameters).

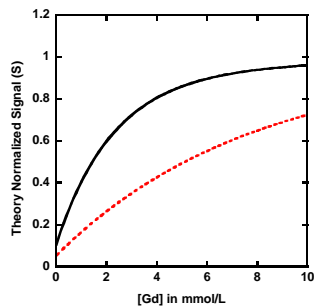


Fig. 2. S-[Gd] theoretical curves for short TD image (--- red) and long TD image.

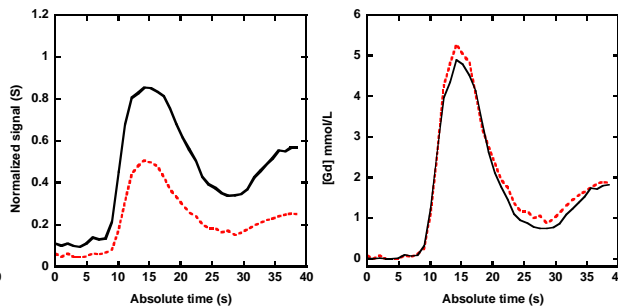


Fig. 3. Measured S-time curves for short TD image (--- red) and long TD image (left); resulting [Gd]-time curves for short TD image (---red) and long TD image.

Results

The image quality of the low resolution image is sufficient to enable the selection of an uncontaminated LV-cavity ROI from a mid-ventricular short-axis image. Fig. 3 shows the AIF curves derived from the short TD_{LR} image and the long TD image (left), and the corresponding arterial input [Gd]-time ([Gd]-t) curves calculated with our theoretical calibration method. Note that the real [Gd] course in the cavity is identical for both cases, since these images are acquired in the same study and during the same heartbeat intervals. The curves are plotted against absolute acquisition times extracted from the dicom header of the images.

Discussion

For the Gd dosage used here, we do not operate in the fully saturated regime of the S-[Gd] curves, and thus we are able to calibrate the signal for both the short TD_{LR} and long TD image, and obtain similar arterial input [Gd]-t curves (Fig. 3). However, the [Gd]-t curve resulting from the long TD image shows some distortion. This is due to the flattening of the corresponding theoretical S-[Gd] curve when the normalized signal surpasses 0.8, which enhances the susceptibility of the [Gd] estimates to image signal variations due to noise. However, for larger Gd doses, the AIF obtained from the long TD will be clipped due to complete signal saturation, and we would not be able to recover the [Gd] values in such a regime, where the S-[Gd] curve is horizontal. To avoid this, we propose to use the arterial input [Gd]-t curve derived from the short TD_{LR} images, as it is reliably calculated from an unsaturated AIF. When inputting the [Gd]-t curves to a deconvolution tracer kinetics model, the short TD_{LR} arterial input [Gd]-t curve can be combined with the large TD myocardium [Gd]-t curve, since they are properly rescaled. This will provide correct arterial input and enhanced SNR myocardium tracer-time courses, which should improve our ability to perform a reliable deconvolution. The tracer kinetics model deconvolution also effectively eliminates γ , and thus using the true γ is not critical. We conclude that this method should be useful for quantitative perfusion studies, since it can provide rapid absolute serial [Gd] in vivo estimates.

References

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