Conversion of the MR signal intensity to Gd-DTPA concentration in contrast-enhanced MR renography

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Introduction

Quantitative determination of in-vivo Gd-DTPA concentration is required in many studies involving perfusion, tracer kinetics and tissue permeability. This task is often accomplished by designing an acquisition protocol in which signal intensity is linear with Gd-DTPA concentration over a clinically relevant range (1, 2). We have developed and validated a more general method for calculating Gd-DTPA concentration from the MR signal intensity (SI) and applied it to dynamic contrast-enhanced 3D renography. Because the method is based upon the phantom and in vivo measurements of signal enhancement over a wide range of T1, it may be applicable to any pulse sequence and is free of assumptions of linearity between SI and concentration.

Methods:

Ten patients underwent MR renography and ^{99m}Tc-DTPA renal scintigraphy with gamma-camera on the same morning. For dynamic contrast MRI, all patients were injected with 4ml of Gd at a concentration of 0.5mmol/ml and imaged at 1.5 T using a fast, interpolated 3D gradient echo sequence (TR/TE/flip angle=2.84/1.05/12°), acquisition time 3 sec, that was repeated up to 20 min following injection. The low dose was selected to minimize T2 effects of concentrated gadolinium in the renal medulla and collecting system. Concentrations of Gd-DTPA were calculated using the relationship between the pre- and post-contrast relaxation times $T1_0$ and T1: $1/T1=1/T1_0+cR$, where c is the concentration of Gd-DTPA and R is the relaxivity of Gd-DTPA. Pre-contrast T1 values were measured using single breath-hold inversion-recovery prepared True FISP sequence (3). Post-contrast T1 values were determined using the relationship between SI and T1 derived from a patient measurements(Fig.1): SI = gf(T1), where g is a scaling factor that depends on such factors as system gain, coil sensitivity, position, etc. It has been confirmed that various tissues follow the same form of the function f(T1) by performing in vivo perfusion measurement with multiple T1 mapping scans repeated at different times after the injection of contrast (Fig 1). We compared our approximation to the frequently used relative signal change method, computed as $RS_{app} = m(S-S_0)/S_0$, where S_0 is the pre-contrast SI and $m = |f'(S_0)|$ (Fig 2). The kidney MR images were segmented (medulla, cortex and ureter) and concentration estimates were computed using both methods. Scintigraphy was performed using a similar imaging protocol following injection of 10 mCi Tc99m-DTPA. Radionuclide concentrations were calculated in the kidneys and aorta from the gamma camera count rate after calibration using a known source and after correcting for background and tissue attenuation (4). The geometric factors involved in these calculations were derived from individual subject structural MRI. A three compartmental model of the kidney was used to calculate single kidney glomerular filtration rate (GFR) using each concentration estimation technique and results were compared with radionuclide calculations.

Figure 1

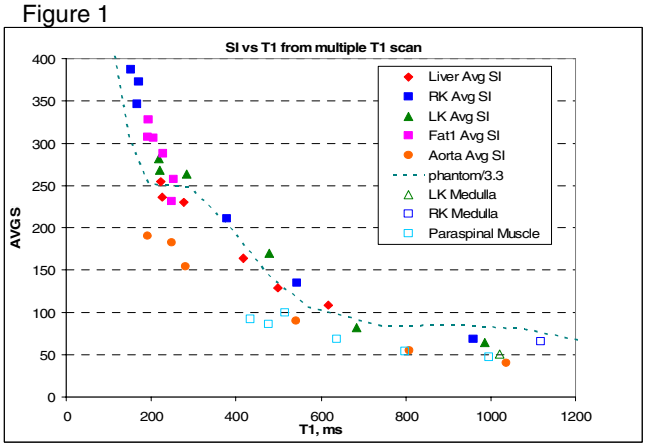


Figure 2

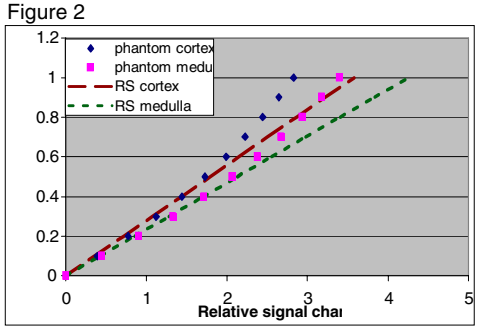


Figure 3

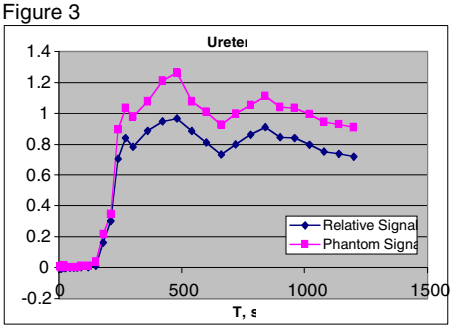


Figure 4

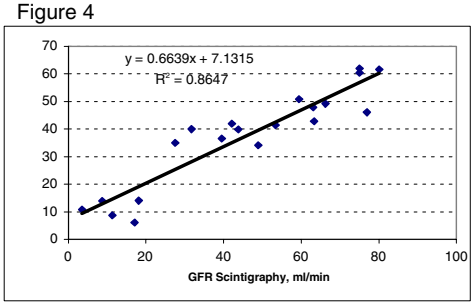
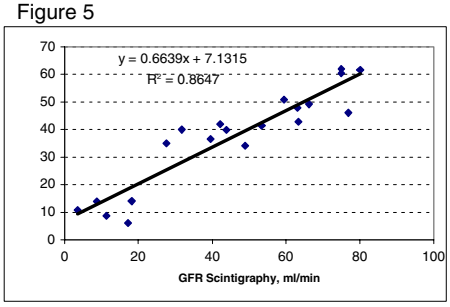


Figure 5



Results

At higher concentrations, the approximation RS_{app} results in an underestimation of Gd-DTPA concentration (Fig.2). For our GRE sequence in higher concentration regions such as the collecting system, there is approximately a 20% error with RS_{app} compared to our calibration method, shown in one representative subject (Fig.3). There is no significant difference between the GFR estimates based on the two Gd conversion methods (Fig.4) for the 4 ml of Gd-DTPA administered.

Discussion

The calibration method has the potential to improve the accuracy of Gd-DTPA concentration, with greater improvements for larger administered doses. (Fig 2) and in regions of concentrated contrast, such as in distal tubules and collecting system. Quantification of these regions is particularly important in the assessment of tubular pathology such as acute tubular necrosis in transplant dysfunction.

References

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