Quantified Delayed Enhancement in Myocardial Infarction using Free Breathing Saturation Recovery SSFP

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Introduction - T1-weighted imaging during the delayed enhancement phase 10-20 minutes after administration of contrast agent (CA) is clinically established to measure the spatial extent of scar tissue in myocardial infarction (1,2). However, these methods do not provide a quantitative measure of the contrast agent distribution, and require careful selection of sequence parameters to obtain diagnostic results. Quantitative T1 mapping methods such as Look-Locker type sequences (2-4) can overcome this limitation, but require breath-holding (2-5), and provide no means to cope with cardiac motion or only insufficient spatial resolution. We present a high resolution T1 mapping method allowing the quantification of contrast agent concentration during free breathing.

Methods - All experiments were performed on a 1.5T clinical MR scanner (Achieva, Philips Medical Systems) equipped with a cardiac coil array. An ECG-triggered, navigator-gated saturation recovery sequence as sketched in Fig. 1 was employed. To provide a reliable navigator signal, a spatially selective saturation pulse (S) sparing the right hemi-diaphragm (navigator location) was applied. A 3-component composite pulse (90° spatially selective pencil beam, 180° refocusing pulse, and 90° nonselective secant saturation pulse) was employed for this purpose (cf. Fig. 1). A 2D SSFP sequence (α=60°, TR/TE=4.2/2.1 ms, 32 readouts per cardiac cycle, resolution 0.89×1.25×8mm) was used for data readout in late diastole, where a varying delay TS between presaturation (S) and image acquisition provided n=10 images with different T1 weightings. A 3-parameter exponential model was fitted to the data to obtain T1. The absolute amount of contrast agent in tissue was quantified according to Eq. 1, where r_1 denotes the relaxivity of the CA (Magnevist, Bayer Schering, r_1=4.95mmol^{-1}s^{-1}). It was assumed that the r_1 is equal in myocardial tissue and blood (6). Spatial maps of the CA distribution were acquired *in vivo* in 5 patients with chronic myocardial infarction approximately 15 minutes post contrast injection of 0.2mmol/kg. The scan time was 2-3 minutes during free breathing.

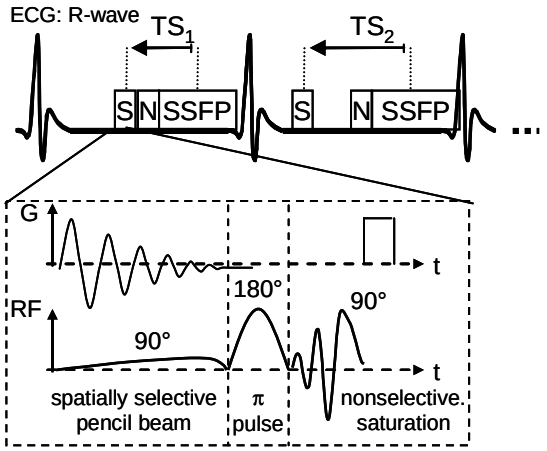

Fig 1. ECG-triggered, navigator (N) gated saturation recovery sequence used for the T1 measurement. A spatially selective saturation pulse (S) is performed with different delays TS from the image acquisition.

$$c = \frac{T1^{-1}_{nonviable} - T1^{-1}_{viable}}{r_1} \qquad \text{[Eq. 1]}$$

Results - Selected *in vivo* results are shown in Fig 2 [a-f], respectively. Artifacts resulting from cardiac or respiratory motion sufficiently suppressed, resulting in good image quality. The T1-weighted images acquired with different saturation delays reveal enhanced areas with myocardial scar tissue (solid arrows, Fig 2 [a-d]). T1 shortening due to the accumulation of CA is quantified in the color-coded T1 map (Fig 2 [e]). The focal concentration of CA in myocardium is shown as a color-coded concentration map overlaid with the anatomy in Fig. 2[f]. A good visual agreement between areas showing enhancement on the T1 weighted image, and areas with accumulation of CA on the concentration map was observed. A higher CA concentration was measured in the infarct core compared to the rim.

Conclusion - Using the present, cardiac and respiratory motion corrected T1 mapping sequence, the spatial distribution of CA can be measured during free breathing, while motion artifacts are suppressed. The sequence provides a submillimeter resolution, which allows differentiating different areas within an infarction. The proposed technique seems to be sufficiently robust and accurate to be used for clinical validation.

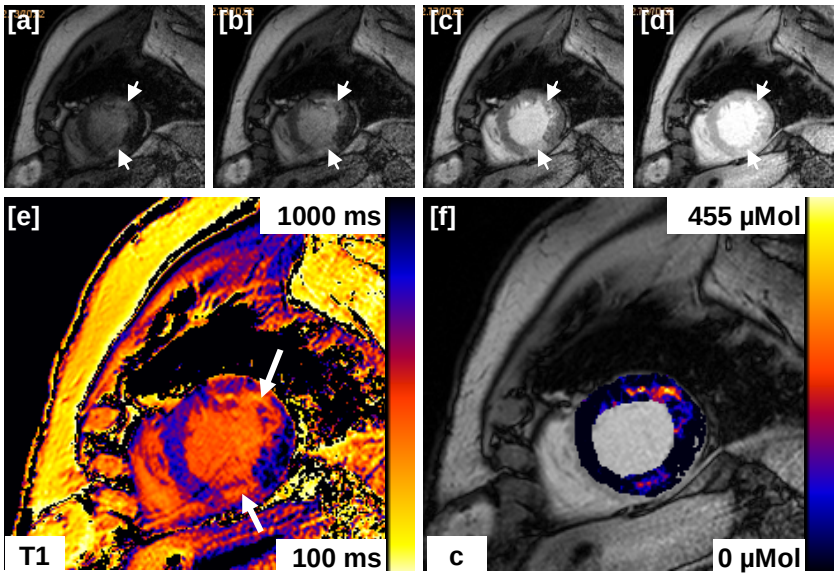

Fig 2. Saturation recovery data acquired with different delays TS [a-d], T1 maps in milliseconds [e], and spatial distribution of contrast agent in µMols. The solid arrows mark the infarct areas.

References
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