Pixel-based and model-free quantification of myocardial perfusion using first-pass dynamic contrast-enhanced MRI

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Introduction

First-pass dynamic enhancement MRI has been widely recognized a potential tool to assess myocardial perfusion. Current algorithms of quantifying myocardial perfusion require a pharmacokinetic model involving deconvolution of arterial input function (AIF) and procedures for parametric fitting. Such approach may fail once AIF or regional myocardial signal intensity cannot be fitted satisfactorily with the given function. In this study, we proposed a model-free approach in which myocardial perfusion can be directly quantified based on the initial rise of the signal-time curves in the left ventricular cavity and myocardium. Our approach lies on an assumption that in the early period of the first-pass perfusion Gd-DTPA only flows into but not flows out from the regional myocardium. Therefore, the signal intensity in the regional myocardium *M* in this period is determined by the signal intensity change of the left ventricle as AIF, denoting *V*, delayed time *Td* related to transport of Gd-DTPA from the left ventricle to myocardium, and perfusion rate α:

$$M\ (t + Td\ ) = \int_0^t V\ (\tau\ )d\tau \times \alpha$$

Materials and Methods

Six healthy volunteers received first-pass contrast-enhanced MR studies on a 3T MR scanner (Trio, Siemens, Germany) at rest and dipyridamole–induced stress conditions. Three short-axis planes at basal, mid left ventricular (LV) and apical levels were acquired using SR-Turbo FLASH pulse sequence (TR/TE/FA = 160ms/0.98ms/10°, spatial resolution = 2mm, temporal resolution = 1 R-R interval and the total number of time frames = 80). Right after the scanning started, Gd-DTPA (0.05mmol/kg) was injected via left antecubital vein at a rate of 4 ml/sec. The stress study was performed approximately 10 min after the rest study. Vasodilator (dipyridamole, 140μg/kg^{-1}/min^{-1}) was first infused intravenously via right antecubital vein for 4 min and the image acquisition began at the 7th min when the maximal vasodilation was achieved. After semi-automatic registration of images to correct respiratory effect, pixel-based regional myocardial signal intensity profile *M* and left ventricular signal intensity profile *V* were derived. The optimal perfusion rate α and delayed time *Td* were determined in a range of *Td* between 0.0~10.0 sec and α between 0.0% ~ 6.0% per sec by least square method to fit the observed *M* at each pixel.

Results

As illustrated, the pixel-based analysis of α and *Td* for each layers can be visualized in color scale. To average data from six healthy adult volunteers, α was found not different in anterior, lateral, posterior, and septal areas in both rest and stress states. On the other hands, *Td* at rest was highest in the posterior wall (2.6±0.8 sec), followed by the lateral (1.7±0.6 sec, P<0.05 as compared with posterior area)and septal walls (1.8±0.8 sec, P=0.05), and lowest in the anterior area (1.2 ±0.2 sec, P<0.05). This difference disappeared at stress state (Td=0.9±0.3, 1.0±0.3, 0.9±0.3, and 0.8±0.2 sec in anterior, lateral, posterior, and septal areas, respectively, P=N.S.).

Conclusion

The presented study provides a quantitative analysis of regional myocardial perfusion status in terms of perfusion rate and delayed time. With this method, we found that the relatively low signal intensity in the posterior wall in early phase of rest perfusion may be due to increased transit time rather than true hypoperfusion.

References

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2. *Nagel E et al, Circulation 2003;108:pp432~437*
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Figure Legend

Fig.1: (A) Image registration to correct respiratory effect. (B) The fitting function between red line (S.I. of LV cavity), and blue line (S.I. of myocardium). (C) S.I. profile of LV cavity (D) S.I. profile of single pixel of myocardium (indicated with yellow spot) and the fitting result.

Fig.2: Left, Illustration of pixel-based myocardial perfusion regarding α and *Td* in both rest and stress states. Right, α were not different in both rest and stress states in four areas. But *Td* at rest was highest in the posterior wall, followed by lateral and septal walls, and this difference disappeared after stress.