On the influence of respiratory motion in quantitative myocardial perfusion MRI

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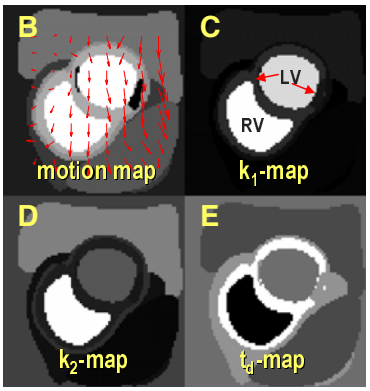
INTRODUCTION Pixel-based analysis of myocardial perfusion using first-pass MRI has the advantage of using the full underlying spatial resolution of the images, but requires accurate motion compensation in the presence of respiratory motion to maintain the true spatial resolution. Numerous motion compensation strategies have been proposed (e.g. breath-holding and image registration), yet little is known about how accurate such approaches should be in order to obtain reliable perfusion measurements. For instance, it is a well-known problem that breath-holding has relatively low reproducibility, which increases the risk of image misalignments between rest and stress examinations. It remains elusive, however, to what extent this affects measurements of myocardial perfusion. Another common problem is that the models employed for correcting image misalignments are typically too simple to adequately resemble the physiological motion of the heart. For example, image registration is fundamentally limited to in-plane correction, and it is often (falsely) assumed that the motion of the heart is described by a global translation. Therefore, the purpose of this work was to investigate the influence of insufficient respiratory motion correction in pixel-based quantification of myocardial perfusion. In particular, we examined the effect of through-plane misalignments and whether insufficient correction might leave subendocardial perfusion defects undetected.

METHODS To study in detail the influence of respiratory motion a realistic and flexible 3-D model of the heart was developed. The model was constructed from an actual first-pass data set acquired during free breathing (see figure 1) and consists of 3 closely spaced short-axis slices, each of which is characterised by four parametric maps: A motion map, which is used to model in-plane motion, and three maps containing the parameters of a one-compartment perfusion model:

$$s_t(t) = k_1 s_{AIF}(t) \otimes e^{-(t-t_d)/k_2} \qquad \text{(Eq 1)}$$

where $s_t(t)$ is the simulated signal in a given pixel (without respiratory motion), $s_{AIF}(t)$ is the arterial input function, k_1 represents perfusion, and t_d and k_2 are time constants. Simulations were performed only for the central slice, which contains a subendocardial and a transmural perfusion defect, as depicted in figure 1A and 1C. The parameter maps for the central slice are shown in figure 1B-1E.



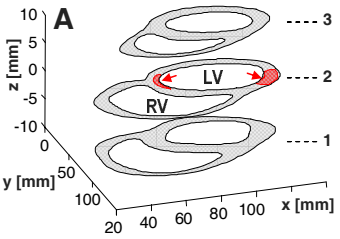


Figure 1. The model of the heart consists of 3 slices (**A**). Simulations are performed for the central slice, which contains two infarctions. The adjacent slices are used for modelling through-plane motion. Subfigures **B-E** are the parameter maps used for modelling in-plane motion (**B**) and perfusion (**C-E**).

Respiratory induced in-plane motion was simulated by warping the central slice according to the motion vector map shown in figure 1B. To change the amplitude of the motion, the map was multiplied by a scalar value. Through-plane motion was modelled as a simple partial volume effect along the z-axis. For instance, to model a 7 mm downwards shift of the heart, the motion corrupted signal at position x would be $s_{corrupted}(x) = 0.7*s_3(x)+0.3*s_2(x)$, where the subscripts indicate the slice number. The arterial input function (s_{AIF}) and simulated myocardial signal curves with no motion ($s_{no\ motion}$), in-plane motion ($s_{in\text{-}plane\ motion}$), and through-plane motion ($s_{through\text{-}plane\ motion}$) are shown in figure 2. The signal distortions in the presence of motion will cause errors when calculating perfusion.

The following simulations were performed: Simulation 1: Through-plane translation of the heart in the range -10 to 10 mm. Simulation 2: In-plane translation of the heart in the range -10 to 10 mm along the motion vector located in the centre of the left ventricle (see figure 1B). Simulation 3: In-plane deformation of the heart according to the motion map depicted in figure 1B but with subtraction of the translation found in simulation 2 (i.e. there is no translation of the heart in this simulation). Simulation 1 and 2 resemble the misalignments that may occur for instance between successive breath-holds. Simulation 3 resembles what happens when image registration is confined to a (rigid) translational model, while the heart undergoes non-rigid deformation. The data was modelled as if acquired during a perfect breath-hold, but for different diaphragmatic positions (or motion amplitudes). This does not fully simulate free-breathing data acquisition, but significantly simplifies the process of interpreting and comparing the results. Finally, for each simulation k_1, k_2, and t_d were estimated in all left ventricular pixels by a non-linear fit to Eq 1.

To illustrate the influence of through-plane misalignments in-vivo, k_1 was calculated for the same patient in *free-breathing* data sets acquired *with* and *without* prospective slice-tracking [1]. Both data sets were acquired during the same scan, and residual in-plane translation of the heart was manually compensated. Thus, the majority of the differences between the two data sets must be due to through-plane motion (which is corrected with prospective slice-tracking but not without).

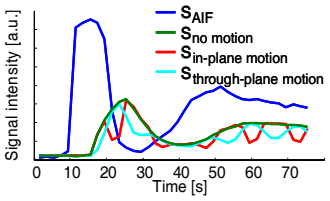


Figure 2. The arterial input function used for all simulations (s_{AIF}) and simulated signal curves for the same myocardial pixel location.

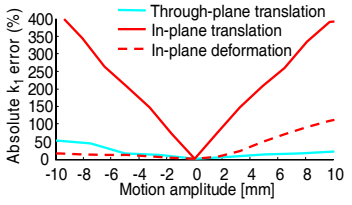


Figure 3. The absolute error of k_1 in the left ventricle (in percent) for each of the three simulations.

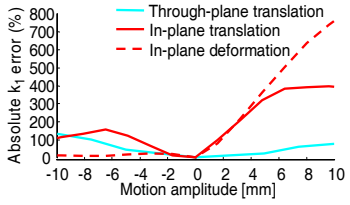


Figure 4. The absolute error of k_1 in the sub-endocardial perfusion defect (in percent) for each of the three simulations.

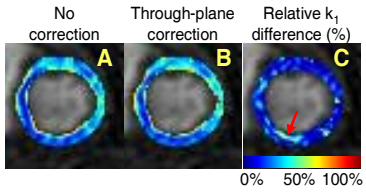


Figure 5. In-vivo maps of k_1 with (**B**) and without (**A**) through-plane correction, and the relative difference of k_1 in percent (**C**).

RESULTS The absolute errors of k_1 are shown in figure 3 for the left ventricle (LV), and similarly in figure 4 for the subendocardial perfusion defect. As expected, the errors increase as the motion amplitude is increased. The asymmetric nature of some of the curves is due to the geometry of the heart. According to figure 3, the peak error for the LV is approximately 400% for in-plane translation, 100% for in-plane deformation, and 50% for through-plane translation. These errors become considerably larger when looking at smaller regions of the myocardium, such as the subendocardial perfusion (cf. figure 4). The in-vivo results are shown in figure 5. As illustrated in figure 5C, the average difference is not that large. However, there is a small region of the subendocardium for which the difference is about 60%.

DISCUSSION AND CONCLUSION A framework has been presented for simulating the influence of respiratory motion of the heart in measurements of myocardial perfusion using first-pass MRI. Overall, in-plane translation is by far the primary source of error. It is relatively straight forward, however, to correct this type of motion by applying dedicated image registration algorithms. Non-rigid deformations and through-plane movements, on the other hand, are not as easily removed and may induce significant errors. In particular, for the simulated subendocardial perfusion defect (cf. figure 4), errors of k_1 may reach a level of more than 100% even for the relatively small motion amplitudes presented in this study. This observation is supported by the in-vivo data. With errors of this magnitude, small subendocardial perfusion defects are easily left undetected. Therefore, our main conclusion is that non-rigid deformations of the heart and through-plane motion should be taken into account when performing pixel-based analysis of myocardial perfusion. As suggested, a possible way to reduce through-plane motion (and at least some of the myocardial deformations) is to use prospective slice-tracking [1]. We are currently investigating this approach in more detail.

REFERENCES [1] Pedersen et al., Proc ISMRM 2005 p. 512.