In-Vivo Quantification of Turbulent Velocity Fluctuations

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Introduction

Phase-contrast (PC) magnetic resonance imaging (MRI) is a powerful tool for quantifying blood flow. Due to spatial averaging over the voxel and temporal averaging over the duration of the bipolar gradient, the conventional PC-MRI velocity mapping method measures only mean velocities. Consequently, this method is not suitable for assessing turbulent velocity fluctuations which are essential in understanding the pathophysiology of several cardiovascular diseases. Recently, an in-vitro study has shown that the PC-MRI signal magnitude can be used to measure the intra-voxel velocity standard deviation (SD) in turbulent flow [1]. In the present study, we utilize this method in-vivo and present a novel approach to quantify mean and fluctuating blood flow.

Methods

Using Reynolds decomposition, the average kinetic energy of the flow (KE) can be statistically separated into the mean kinetic energy (MKE) and the turbulent kinetic energy (TKE) according to $\mathrm{KE} = \mathrm{MKE} + \mathrm{TKE} = 0.5\rho(\sum_{i=1}^{3}\overline{U_i}^2 + \sum_{i=1}^{3}\overline{u_i^2})$, where ρ is the density, $\overline{U_i}$ is the mean velocity and u_i is the velocity fluctuation in direction i [2]. The velocity fluctuation reflects the irregularity and randomness of turbulent flow and is therefore interpreted as a property of the turbulence. The intensities of the turbulent velocity fluctuations in different directions, which are required to compute the TKE, were measured using a PC-MRI SD acquisition [1, 3]. The mean velocities were measured using standard PC-MRI velocity mapping.

A 60-year old female patient was scanned using a clinical 1.5 T scanner (Philips Achieva, Philips Medical Systems, Best, The Netherlands). The patient was 46 years status post aortic coarctation repair with end-to-end anastomosis and had signs of restenosis in the anastomotic area distal to the left subclavian artery. Retrospectively cardiac gated 3D PC-MRI data were acquired using three-directional interleaved velocity encoding. Navigator gating was used to suppress respiratory effects and the velocity data was subsequently corrected for background errors. Imaging parameters are summarized in table 1. For anatomical orientation a 3D contrast-enhanced MRA was acquired.

Table 1 – Overview of Imaging Parameters

Acquired quantity	FOV	Matrix size	TR	TE	VENC	Flip angle	Temporal resolution
Velocity	272 x 272 x 60 [mm]	80 x 80 x 20	5.4 [ms]	3.0 [ms]	3.5 [m/s]	8 [°]	64.8 [ms]
SD	288 x 288 x 54 [mm]	96 x 96 x 18	5.1 [ms]	3.0 [ms]	1.4 [m/s]	8 [°]	61.2 [ms]

Results

The MRA showed a constriction at the site of the anastomosis with well-developed collaterals. Aortic blood flow at peak systole is shown in Fig. 1 where streamlines (left) reveal a flow jet distal to the anastomosis. The TKE map (right) provides information on the energy content of the turbulent velocity fluctuations, which are greatest in the surroundings of the flow jet. Figure 2 shows the MKE and the corresponding TKE in the descending aorta over the cardiac cycle at the location indicated by the arrow in fig. 1.

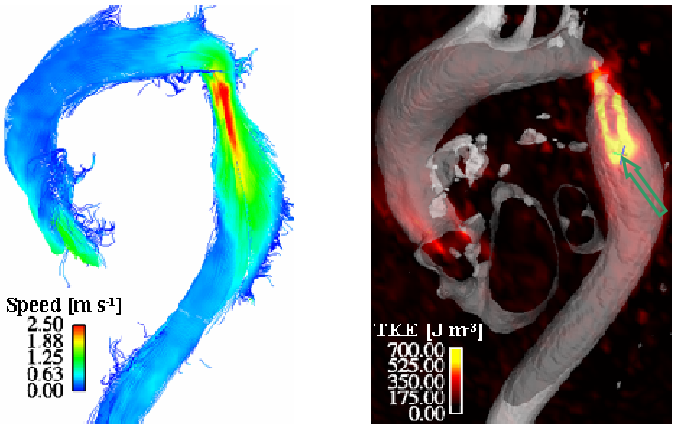

Fig. 1. Visualization of aortic blood flow at peak systole. Left: 3D streamline visualization. Right: 2D plane showing TKE together with a semi-transparent 3D iso-surface rendering of the contrast-enhanced MRA data.

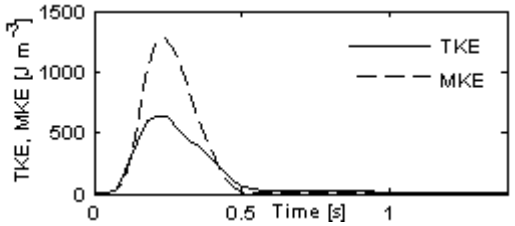

Fig. 2. Plots of MKE (dotted line) and TKE (solid line) over time during the cardiac cycle.

Discussion

These results demonstrate the feasibility of this method for in-vivo quantification of the intensity of turbulent velocity fluctuations. In the immediate vicinity of the stenotic anastomosis the flow has the highest velocities but the TKE is low. In contrast, the surrounding areas are impacted by considerably greater TKE. An advantage of using the TKE as a measure of the turbulent velocity fluctuations is that it can be compared with the MKE as exemplified in fig. 2.

Visualization of blood flow using velocity data from conventional PC-MRI (fig. 1, left) is valuable when studying mean flow behavior, but may miss the turbulent flow fluctuations which have important effects on mixing, vascular remodeling, and atherogenesis. To be able to quantify fluctuating blood flow in-vivo would be of outmost value in improving our basic understanding and detection of disordered flow, and greatly assist in follow-up after surgical or medical intervention.

References

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