Direct quantification of turbulent shear stresses by multi-point phase-contrast MRI

Christian Binter¹, Verena Knobloch¹, Andreas Sigfridsson¹, and Sebastian Kozerke¹

¹Institute for Biomedical Engineering, University and ETH Zurich, Zurich, Switzerland

Introduction:

Excessive shear stresses in the cardiovascular system can lead to platelet activation and damage of red blood cells [1]. The effect is, for example, encountered in mechanical heart valves, requiring life-long anticoagulation therapy. Accordingly, the evaluation and improvement of the hemodynamic properties of heart valve prostheses is of great importance. However, the possibilities for an assessment of these shear stresses are limited. Previous methods including Laser Doppler and Hot Film Anemometry are not feasible in-vivo and are therefore restricted to phantom measurements.

Phase-Contrast (PC) velocity measurements have been widely accepted for acquiring time-resolved 3D velocity vector-fields. Beyond quantification of coherent blood flows, the PC encoding principle may also be modified to enable assessment of kinetic energy stored in incoherent flows giving rise to velocity fluctuations [2]. Moreover, the quantification of turbulent shear stresses is also feasible but the measurement suffers from its susceptibility to noise [3].

In this work, a robust method to determine turbulent shear stresses using generalized PC-MR imaging is presented. It is applied to in-vitro measurements of artificial heart valves and in-vivo data of healthy volunteers.

Theory:

A velocity vector field v can be decomposed into two parts, the mean velocity field V and the fluctuating field v': v=V+v'. The stresses in a fluid due to turbulent velocity fluctuations can be described by the Reynolds stress tensor, defined as $\rho v'_i v'_j$ with ρ denoting fluid density and i, j = 1, 2, 3 denoting directions. Assuming a Gaussian velocity distribution, the elements on the main diagonal of the tensor correspond to the variance σ^2 of the velocities in a voxel for every direction, and can be interpreted as normal stresses. The off-diagonal elements correspond to turbulent shear stresses. In Phase-Contrast MR velocity measurements, velocity fluctuations lead to a signal attenuation depending on the first gradient moment k_{y} and σ^{2} , making it possible to quantify the

variance of the fluctuating velocities. The comprehensive signal model is given in eq. 1 [2]. The trace of the Reynolds stress tensor is termed Turbulent Kinetic Energy (TKE), a direction-independent measure for the energy stored in the velocity fluctuations. For the shear stresses, an additional measurement along the bisecting axis of two directions i and j, resulting in the variance σ^2_{i+j} , has to be performed. The off-diagonal elements are then calculated following eq. 2 [3]. Having determined all elements of the stress tensor, a principal stress analysis yields the principle normal stresses $\delta_1 > \delta_2 > \delta_3$. The maximum shear stress τ_{max} and TKE can then be calculated using eqs. 3-4 [4].

$$S(k_v) = S_0 e^{-\frac{\sigma k_v}{2}} e^{-ik_v V}$$
 eq. 1

2, 2

$$v'_{i}v'_{j} = \sigma_{i+j}^{2} - (\sigma_{i}^{2} + \sigma_{j}^{2})/2$$
 eq. 2

$$\tau_{max} = \frac{\delta_1 - \delta_3}{2} | TKE = \frac{\rho}{2} \sum_{i=1}^3 \sigma_i^2 \text{ eqs. 3-4}$$

Materials and Methods:

3D PC flow measurements with 3 different encoding velocities in all 6 directions (main and bisecting axis) were combined with a Bayesian analysis method adapted from [5]. Multiple encoding velocities lead to a larger dynamic range and smaller errors [6]. The turbulent kinetic energy and the maximum shear stresses in every voxel were determined according to eqs. 3-4.

In-vitro measurements were performed using a home-built pulsatile flow phantom to study a biological Transcatheter Medtronic CoreValve (Medtronic Inc., Minneapolis, MN, USA). The heart rate was set to 67 beats/min, and stroke volume was 82 ml. In-vivo data were acquired in 5 healthy volunteers.

All data were acquired on a 3T Achieva system (Philips Healthcare, Best, The Netherlands) with cardiac triggering and navigator gating. The voxel size was 2 mm isotropic, and temporal resolution was 33-37 ms. Employing 8-fold undersampling and k-t PCA reconstruction, the nominal scan time was 15 min.

Results:

The measured shear stresses and velocity maps for the artificial valve are shown in Fig. 1a-c. Highest shear stresses were found to be around 300 Pa, occurring at the border of the jets. Peak flow velocity was 150 cm/s. The peak shear stresses in all healthy volunteers ranged from 151 to 174 Pa, with average levels of 69±15 Pa. Exemplary shear stress and TKE maps are presented in Fig. 1d-e.

Discussion:

In this work an approach for a detailed assessment of the turbulent flow field has been presented. It has been demonstrated that in vivo measurements of turbulent shear stresses are feasible. In vivo data of healthy volunteers show that the physiological level of shear stress is clearly below the threshold for cell damage of 400 Pa [1], while shear stresses occurring distal to valve prostheses may approach these limits. By obtaining time-resolved shear stress and velocity maps it would also be possible to quantify the exposure time of red blood cells to damaging shear stress levels, leading to a more comprehensive picture about the load conditions.





Fig. 1: a) Streamline visualization of the velocity field distal to the CoreValve, the maximum shear stress map (b), and turbulent kinetic energy map (c) during systole. d) Maximum shear stresses in a healthy volunteer during systole, e) map of the turbulent kinetic energy (same time point).

References:

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