

A novel method to calculate vectorial wall shear stress from 4D phase contrast MRI: analytical and in-vivo validation in the common carotid artery at different resolutions

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Introduction: Wall shear stress (WSS) is the tangential force of flowing blood on the vessel wall. WSS directly influences remodeling of the vessel wall. A fully automated method to calculate time-resolved vectorial wall shear stress (WSS) based on 4D phase contrast MRI (PC-MRI) data was developed and the effect of resolution on WSS calculations was assessed using software phantom simulations and in-vivo measurements of the common carotid artery (CCA).

Material & methods: For the phantom experiments, twenty software phantoms with spatial resolutions ranging from 0.12 mm to 1.2 mm were created. All phantom datasets contained a perfect cylindrical vessel with a parabolic flow profile, center velocity of 100 cm/s, diameter of 6 mm. The theoretical WSS (Poiseuille) in these phantom datasets was 2.13 Pa.

For the in-vivo calculation of WSS, five 4D PC-MRI datasets of the CCA were acquired in a healthy volunteer using a 3T MR system (Philips Healthcare, Best, The Netherlands) and a dedicated eight-channel carotid coil. Imaging parameters were: spatial resolutions 0.4, 0.5, 0.6, 0.7 and 0.8 mm isotropic, field of view 60x60x8 mm, flip angle, 25°, TE/TR 4.3/6.11-7.7 ms, velocity encoding values 30x30x100 cm/s (AP, RL, FH), SENSE factor 1.7, number of heart phases 5 (PPU triggered), scan duration 09:23-1:34 min. Acquired data was corrected for systematic phase offset errors and aliasing artifacts. A level set evolution algorithm [1] was used to segment the vessel wall in the 0.4 mm resolution dataset. All lower resolution datasets (0.5-0.8 mm) were coregistered to the 0.4 mm resolution dataset to correct patient movement in between scans. To compensate for natural variation in blood flow through the CCA, velocities for each acquisition were corrected to match a mean flow of 309 ml/min as measured in the 0.4 mm resolution dataset. The segmented vessel wall surface from the 0.4 mm dataset was used to calculate WSS in all five datasets.

WSS vectors ($\vec{\tau}$) were calculated for each point on the vessel wall: $\vec{\tau} = 2\eta\dot{\epsilon} \cdot \vec{n}$ with $\dot{\epsilon}$ the rate of deformation tensor, \vec{n} the inward normal vector and the blood viscosity ($\eta = 3.2 \cdot 10^{-3}$ Pa s). For each point on the vessel wall, the coordinate system $[x \ y \ z]$ is rotated $[x' \ y' \ z'] = R \cdot [x \ y \ z]$ such that the inward normal vectors align with the z' -axis. The normal vector in the new local coordinate systems becomes $\vec{n} = [0 \ 0 \ 1]$. No flow through the vessel wall was assumed (i.e. $\vec{n} \cdot \vec{v} = 0$), resulting in a shear rate vector of $\dot{\epsilon} \cdot \vec{n} = \left[\frac{\partial v_{xt}}{\partial z'} \ \frac{\partial v_{yt}}{\partial z'} \ 0 \right]$. Velocity values (v_{xt} and v_{yt}) near the wall were calculated using three-dimensional natural neighbor interpolation of the original velocity field. The remaining unknown derivatives in the WSS equation ($\frac{\partial v_{xt}}{\partial z'}$ and $\frac{\partial v_{yt}}{\partial z'}$) were then derived from 1D smoothing splines. The smoothing splines were fitted to the velocities along the inward normals, while enforcing the no-slip condition, i.e. $\vec{v}_{wall} = 0$. The points were weighted according to their distance to the vessel wall in order to compensate for possible partial volume effects in the vessel wall voxels. The WSS vector was then calculated using $\vec{\tau} = \eta \left[\frac{\partial v_{xt}}{\partial z'} \ \frac{\partial v_{yt}}{\partial z'} \ 0 \right]$ and finally the calculated WSS vectors were transformed back to the original coordinate system.

Results/Discussion: The phantom study revealed that increasing resolution resulted in improved approximations of the theoretical WSS as shown in figure 1. Additionally the standard deviation (SD) of the calculated WSS declined with increasing resolution. Figure 1 shows that for the phantom data a resolution of 0.6 mm resulted in 95% of the theoretical WSS. For a resolution of 1 mm the calculated WSS decreased to 83.5% of the theoretical value.

In the in-vivo datasets, the WSS showed a similar increase in mean WSS (averaged over heart phases) for higher resolutions, see figure 2. The standard deviation varied between 0.58 and 0.82 Pa, but did not show convergence towards higher resolutions. This can be explained since a physiological WSS variation due to an asymmetric velocity profile was present in the CCA, see figure 3. Individual heart phases all showed a similar increase in calculated WSS for higher resolutions. Figure 4 shows the coregistered velocity profiles and the resulting WSS values for each acquired resolution.

Conclusion: This work presents a novel method to calculate WSS in-vivo. Software phantom results showed that the calculated WSS converged with increasing resolution, which is in line with earlier research by Cheng [2] and Stalder [3]. This effect of resolution was confirmed in in-vivo measurements of the CCA.

[1] Li et al. IEEE T Image Process; 20(7):2007-16 (2010) [2] Cheng et al. Ann Biomed Eng; 30(8):1020-32 (2002)

[3] Stalder et al. MRM; 60(5):1218-31 (2008)

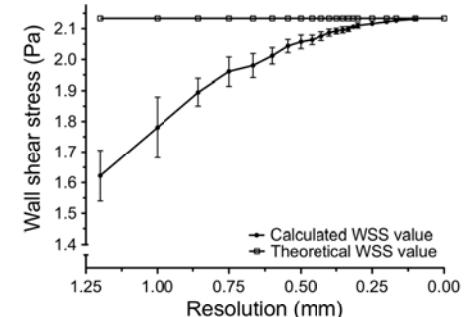
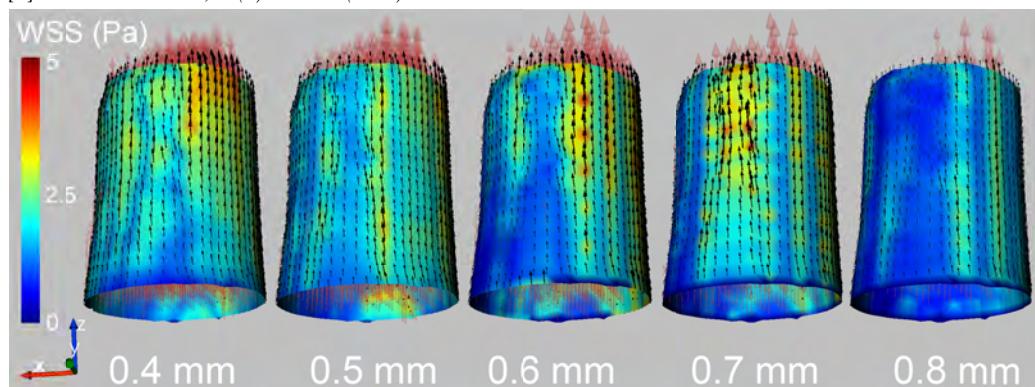


Fig. 1: Mean WSS, SD and theoretical WSS for twenty phantom dataset with different resolution. Vessel diameter 6 mm, center velocity 100 cm/s, theoretical WSS 2.13 Pa.

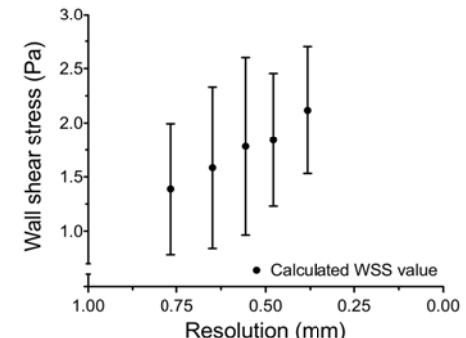


Fig. 2: Mean WSS values and SD for five in-vivo 4D PC-MRI acquisitions at different resolutions.

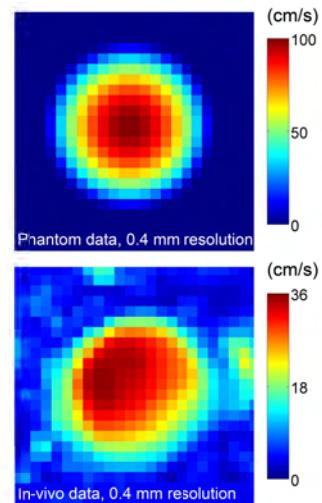


Fig. 3: Velocity profiles in the center of the phantom data (top) and in-vivo data (bottom). Note the asymmetric velocity profile in the

Fig. 4: Calculated WSS values for five in-vivo 4D PC-MRI acquisitions at different resolutions. Red arrows visualize the velocities in systole. Resulting magnitude of WSS is plotted in color on the vessel wall surface. Black arrows depict the direction of the WSS vector.