Coregistration of Wall Shear Stress and Plaque Distribution within the Thoracic Aorta of Acute Stroke Patients

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Introduction: Changes in shear forces acting on the vessel wall characterized by wall shear stress (WSS) or oscillatory shear index (OSI) can lead to the development of aortic plaques. Critical parameters (low absolute WSS and high OSI) seem to play an important role in flow mediated atherogenesis [1, 2].

Recent studies have shown that the combination of flow-sensitive 4D-MRI with an optimized data analysis strategy (segmental WSS vectors in freely selectable analysis planes) allows for the assessment of the distribution of normal aortic wall parameters [3-5]. However, the precise relationship between individual plaque location and wall parameter distribution is poorly understood. It was our aim to evaluate the topology of WSS distribution in 94 acute stroke patients with advanced atherosclerosis of the aorta. Moreover, for n=144 complex plaques with thickness ≥4mm, a one-to-one comparison of plaque location and wall parameter distribution was performed.
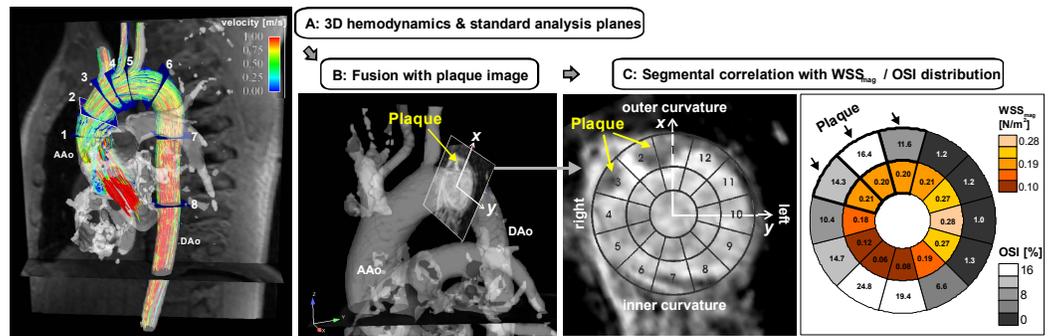

Fig. 1: Strategy for the evaluation of wall shear stress parameter distribution in the aorta (A) and at the site of complex plaques (B, C). **A:** 3D-PC-MRA was used to position 8 standardized analysis planes in the ascending aorta (AAo, 1-3), aortic arch (4-5), and descending aorta (DAo, 6-8) for the calculation of segmental WSS_{mag} and OSI. **B:** For 144 complex plaques, images depicting maximum plaque thickness were fused with the 4D flow data. **D:** A standardized 12 segment model was employed for a direct one-to-one correlation of plaque location and wall parameters.

Methods: 94 consecutive acute stroke patients (67.5±8.6 years) with aortic plaques ≥3mm thickness as detected by transesophageal echocardiography were examined using a 3T MR system (TRIO, Siemens, Germany). 3D GRE imaging with isotropic resolution (~1mm^3) and full coverage of the aorta was performed to detect complex plaques which were found in 74 of 94 patients (figure 3). Flow-sensitive 4D MRI using ECG and respiratory gating was employed to acquire 3D hemodynamics (3-directional velocity encoding, *venc*=150cm/s, temporal/spatial resolution of 40.8ms/1.7x2.0x2.2mm^3) [3].

Flow-sensitive 4D MRI data permitted the calculation of a phase-contrast angiography, 3D flow visualization (EnSight, CEI, USA) and definition of eight standardized analysis planes (fig. 1 A). Regional wall parameters were estimated by interpolating the local velocity derivative on the segmented vessel wall using b-splines. Their time-averaged magnitude (WSS_{mag}) and inversion over the ECG cycle (OSI) were recorded [4]. For complex plaques a reformatted 3D GRE imaging slice at the site of the atheroma was combined with the 4D MRI data via image fusion (fig. 1B). A 12-segment model of the vessel lumen circumference was employed to directly correlate the exact segmental distribution of the plaque with the distribution of WSS_{mag} and OSI (fig. 1C).

Results: The average wall parameter distribution for all 94 patients is shown in figure 2. Critical wall parameters (low WSS_{mag} and high OSI) were concentrated at the left inner curvature of the ascending aorta, near the supra-aortic branches, and the inner curvature of the descending aorta. The most pronounced exposure to critical wall parameters was at the right inner curvature of the proximal descending aorta (yellow circles, figure 2). The presence of low WSS_{mag} typically mirrored that of high OSI which was supported by a significant relationship between low WSS_{mag} and high OSI (*r*=0.47, p<0.001).

Noticeably, the distribution of atherogenic wall parameters was near typical locations of atherosclerotic lesions but did not resemble the plaque distribution in our patient cohort (figure 3). The one-to-one correlation of plaque location and wall parameters revealed a consistent relocation of critical wall parameters by 90° or 180° in segments adjacent to the location of complex plaques (figure 4).

Discussion: The analysis of 3D hemodynamics and plaque location provided detailed insights into hemodynamic wall parameter topology. However, the location of atherogenic WSS did not exactly match with the location of complex plaques in our cohort. The low incidence of plaques in the ascending aorta despite distinct regions with low WSS_{mag} and high OSI points to a different impact of critical wall parameters compared to other aortic segments. Our data suggests, that during the progression of atherosclerosis critical wall parameters relocate to neighboring segments of the region most affected by the disease. The identification of critical wall shear stress in patients with aortic plaques may thus be useful to predict future growth of the atheroma. Future studies are warranted to correlate plaque progression and changes in wall parameter distribution.

References: 1. Malek AM, et al. Jama 1999;282(21):2035-2042 **2.** Cheng C et al. Circulation. 2006;113:2744-2753 **3.** Markl M et al. J Magn Reson Imaging. 2007;25:824-31. **4.** Stalder AF et al. Magn Reson Med. 2008;60:1218-31. **5.** Frydrychowicz A, et al. J Magn Reson Imaging 2009;30(1):77-84.
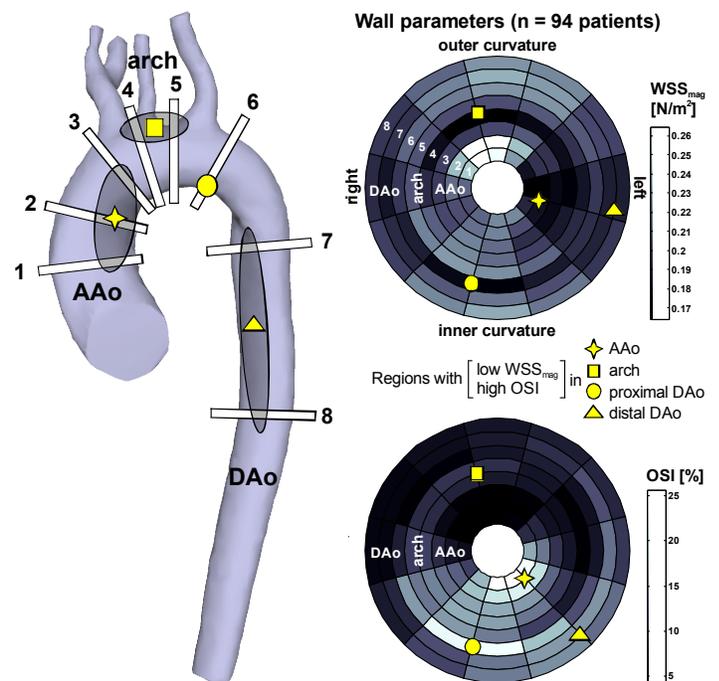
Fig. 2: Averaged distribution of mean WSS_{mag} (top right) and OSI (lower right) in 12 segments along the vessel circumference for the ascending aorta (AAo, planes 1-3), the aortic arch (planes 4-5) and the proximal descending aorta (DAo, planes 6-8). For WSS_{mag} (OSI), darker (brighter) gray scales highlight the presence of atherosclerotic low WSS_{mag} (high OSI) as indicated by the yellow symbols.
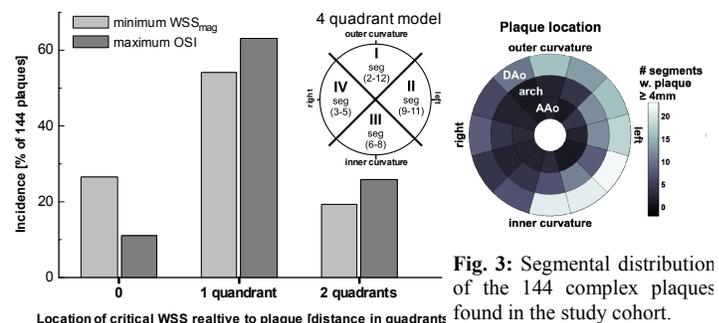

Fig. 3: Segmental distribution of the 144 complex plaques found in the study cohort.

Fig. 4: Individual comparison of the segmental location of complex plaques and critical wall parameters using a 4 quadrant model. In 73% of the complex plaques critical wall parameters for both WSS_{mag} and OSI were shifted by 90° (1 quadrant) or 180° (2 quadrants) to wall segments adjacent to the atheroma.