Flow-sensitive 4D MRI for the analysis of aortic hemodynamics and wall shear stress: results form healthy volunteers and follow-up in aortic stenosis.

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Introduction: The feasibility to acquire time-resolved 3-directional and 3-dimensional blood flow data, to visualize the hemodynamic properties of the aorta, and to derive vessel wall parameters such as the wall shear stress, the frictional force the blood imposes on the endothelial layer, has been shown before [1]. From animal models it is well known that there is an association between low OSI, low WSS and the development of atherosclerosis [2]. Also, both WSS and OSI have been correlated to arterial remodeling [3, 4].
Flow-sensitive 4D MRI implies a complete coverage of an entire vessel such as the aorta. Thereby, data analysis is not only restricted to the pathology but can also elucidate alterations in the blood flow properties further up- or downstream. Therefore, we aimed to analyze aortic hemodynamics in volunteers and compare data to findings in a patient with high-grade aortic stenosis. Since the patient was scheduled for dilatation of the aortic stenosis we hypothesized that alterations in hemodynamics and derived vessel wall parameters should at least gradually improve.

Methods: Experiments were performed on a 3T MR-system (Somatom TRIO, Siemens, Germany) after written informed consent of all 11 volunteers and a 13 year-old patient with severe aortic stenosis who was scheduled for percutaneous transarterial dilatation and implantation of a stent (see fig. 1). 3D blood flow measurements covering the entire thoracic aorta were performed applying flow sensitive 4D-MRI using an eight channel body coil and an rf-spoiled gradient echo sequence with interleaved 3-directional velocity encoding (BW = ± 480 Hz/pixel, flip angle=15°, TE / TR=3.67 / 48.8 ms, venc = 1.5 m/s, spatial resolution = (2.71 - 2.93 x 1.58 - 1.69 x 2.60 - 3.0) mm³, temporal resolution = 48.8 ms). The measurement was prospectively gated to the ECG cycle and utilized a previously reported adaptive navigator technique [5] to enable free patient breathing during the acquisition.
Data visualization was performed with a commercially available software (EnSight, CEI, Apex, NC, USA) using streamlines, particle traces and vector graphs. Also, cutplanes transecting the ascending and descending aorta were manually placed. The cutplanes were exported to a MatLab (The Mathworks, USA) based analysis tool to segment the vascular lumen and calculate the WSS. During visual analysis, datasets were screened for the helicity of blood flow, the development and duration of vortices, the duration of late-systolic and diastolic retrograde flow. According to [1] vectorial WSS was calculated for 12 segments along the vascular circumference of each cutplane using B-splines.

Results: The results from both the patient measurement and the volunteers are presented in tables 1 and 2. Obviously, the mean WSS of the ascending aorta of the patient was markedly reduced in comparison to the healthy volunteers. In the proximal descending aorta, closer to the stenosis, only a moderate reduction of the WSS was observed. After dilatation the WSS values in the ascending aorta gradually improve towards normal values (see fig. 2). Note that WSS alterations were most prominent at in inner curvature of the AAo, indicating that local geometric changes, i.e. stenosis in the DAo, can induce changes in far distant regions, i.e. pathological wall shear forces in the AAo.
During visual analysis, only a single vortex was observed in the volunteers whereas the patient presented with 2 vortices of more that 348ms duration. After therapy, there was also a reduction of the number of vortices. In agreement with physiological hemodynamics, the volunteers showed right-handed helical systolic blood flow. However, in the patient no helicity was observed before and directly after therapy. Only in the 9-month follow-up, right-handed helical flow patters were restored.

Discussion: The analysis of hemodynamics in healthy volunteers was already shown and confirmed physiological flow pattern [6]. Also, the degree of WSS in free selectable cutplanes transecting the aorta was successfully calculated. However, the relevance of these normal values which were in close agreement with previously published values from 2D measurements and computational analysis has still to be evaluated. With the herein presented follow-up in a case of a severe stenosis we were able to underline the possibilities of the method: Obviously, changes in hemodynamics and related vessel wall parameters can be detected in the presence of a geometrical alteration of the aorta. Further, the effect of the treatment was successfully monitored. Finally, the 4D nature of the methodology is fully exploited since these effects were observed in a region not directly affected by the pathology and might have easily been missed by a cine-2D examination. Thereby, we contribute in this single case analysis to the assumption of von Kodolitzsch and Oliver [7, 8], that the development of aortic aneurysms in coarctation may in part be hemodynamically triggered. However, this hypothesis will still have to be proved by data from a larger cohort of patients.

References: 1. Stalder, ISMRM 2007; 2. Cheng, Circulation 2006; 3. Langille, Science 1986; 4. Glagov, NEJM 1987; 5. Markl JMRI 2007; 6. Frydrychowicz, JCAT 2007; 7. von Kodolitzsch, JACC 2002; 8. Oliver, JACC 2004

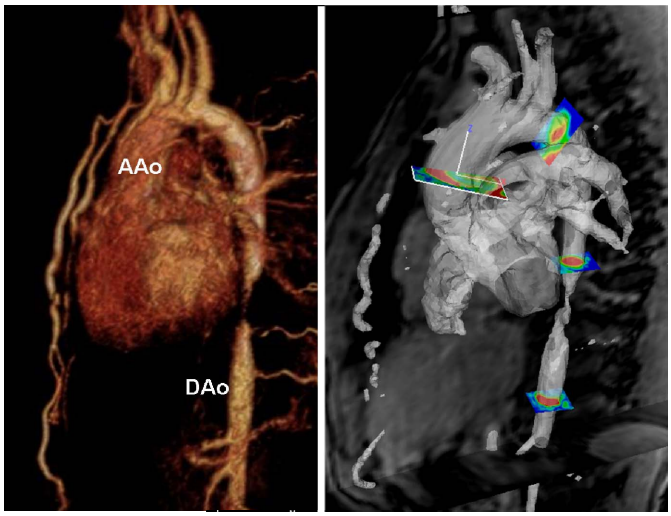

Fig. 1: CE-MRA (left) and phase-contrast MR-angiography (right) in a 13-year-old boy with a severe stenosis in the descending aorta. Manually positioned cutplanes were exported from EnSight to a MatLab-based analysis tool in order to derive the wall shear stress.

	Vortex duration	Mean WSS AAo [N/m²]	Mean WSS DAo [N/m²]	OSI AAo	OSI DAo
Volunteers (n=11)	348ms	0.191±0.06	0.158±0.025	4.1±1.1%	5.4±1.7%
Patient					
- before	>338ms*	0.140	0.158	10.2	2.5
- 5mo	>432ms*	0.127	0.237	10.8	0.0
- 9mo	365.8ms	0.160	0.300	8.5	0.2

Tab. 1: Comparison of hemodynamic values of the volunteers and the patient before, 5 and 9 month after therapy.

	Diameter AAo	Vortex number (n)	Helical flow	Retrograde flow (n)
Volunteers (n=11)	2.4±0.2cm	0 (10) 1 (1)	R-handed (11)	moderate (10) none (1)
Patient				
- before	3.0cm	2	none	none
- 5mo		1	none	none
- 9mo		1	R-handed	none

Tab. 2: Comparison of hemodynamic properties of the volunteers and the patient before, 5 and 9 month after therapy (continued).

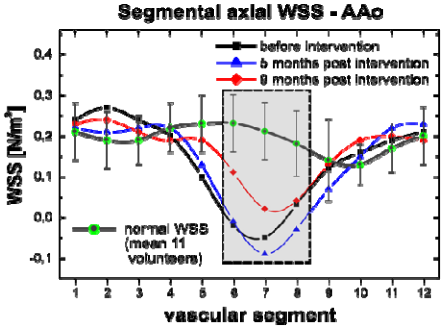

Fig. 2: Graphical display of the alterations of WSS in the patient before and after therapy in comparison to healthy volunteers. Segment 1 is situated on the outer aortic curvature counting anti-clockwise to segment 7 at the inner curvature.