

Diameter-dependence of aortic hemodynamics: Does size matter?

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Introduction: Flow-sensitive 4D MRI enables for the visualization of hemodynamics in an entire vessel such as the aorta. The physiological properties of aortic blood flow and the alterations in hemodynamics in cohorts of older or smoking patients have been described before [1, 2]. Occasional reports have suggested that there is an interrelation between geometric alterations of the aorta [3]. Also, there is a known effect of altered blood flow on arterial remodeling and atherogenesis [4-6]. Therefore, it was the purpose of this study to evaluate, whether there is a diameter-dependence in aortic blood flow. As a diameter larger than 3.5cm in the ascending aorta is generally accepted as an ectatic aorta, this value was used to subgroup our patient collective.

Methods: Experiments were performed on a 3T MR-system (Somatom TRIO, Siemens, Germany) after written informed consent of all 62 patients and volunteers. Time-resolved (cine) 3-dimensional blood flow measurements covering the entire thoracic aorta were performed applying 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 1.58-1.69x2.60 - 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 to enable free patient breathing during the acquisition [7].

Data visualization was performed with a commercially available software (EnSight, CEI, Apex, NC, USA) using streamlines, particle traces and vector graphs [8]. 8 cutplanes transecting the aorta were manually placed (fig. 1). The diameter of the ascending aorta (AAo) was used as a reference to subgroup all individuals (≤3.5cm: small aortic diameter group, SAD; >3.5cm, large aortic diameter group, LAD). During visual analysis, datasets were screened for the helicity of blood flow, the development and duration of vortices, the time to peak arterial flow, and the duration of late-systolic and diastolic retrograde flow.

Results: The SAD group consisted of 38 patients (AAo 2.81 ± 0.38cm, age 42.9 ± 18.6 years, 10.0 ± 10.1 kg, 19 female), the LAD group of 24 patients (AAo 3.81 ± 0.39cm, age 62.2 ± 13.4 years, 78.0 ± 9.8kg, 6 female). In the LAD group, there was a significantly higher number of vortices (SAD 1.3 ± 1.1 vs. LAD 2.3 ± 0.8, p<0.0005) and a reduced number of patients with a helical flow pattern (see tab. 1).

With respect to the time to peak arterial flow only cutplanes 1 and 2 revealed a significantly delayed TTP (cutplane 1: SAD 119 ± 33ms vs. LAD 157 ± 49ms; cutplane 2: SAD 123 ± 36ms vs. LAD 167 ± 60ms, for both p=0.0006). The same applied to the duration of backward flow where the LAD group showed significantly prolonged retrograde flow in cutplanes 1 and 2 (cutplane 1: SAD 243 ± 115ms vs. LAD 358 ± 117ms; cutplane 2: SAD 233 ± 122ms vs. LAD 340 ± 119ms; for both p<0.005). All other results showed non-significant differences with a trend towards faster TTP in the SAD group and longer backward flow in the LAD group.

Discussion: By 4D MRI and color-coded visualization the dependency of aortic diameters and hemodynamics has been successfully shown. Obviously, only a small subgroups with respect to the diameter of the aorta were created disregarding the fact that comorbidities such as atherosclerosis, age and smoking may play an equally important role [1,2]. Clearly, a trend towards higher number of vortices in large aortic diameters can be seen. Given the assumption that vortices may induce altered oscillations of the wall shear stress this may be taken as an unfavourable condition. The fact that only cutplanes 1 and 2 show significant delayed TTP and prolonged retrograde flow in the LAD group is not of concern since only the AAo diameter has been used as a subgrouping variable. Further analysis will have to evaluate whether subgrouping of diameters in the arch and DAo alter the results.

Nevertheless, our data suggest, that even small changes such as a mildly dilated ascending aorta does have impact on the related hemodynamics. This points further towards the necessity to put more effort on the evaluation of hemodynamic changes caused by geometric alterations, i.e. in pathologies such as aneurysms or stenoses. It is of special note that the cutpoint of our subgroup, namely the diameter of the AAo is still appreciated as normal in terms of aortic geometries but already has an impact on the hemodynamics keeping in mind that there is a close relation between arterial remodeling and altered wall shear forces [4-6].

References: 1. Bogren, JMRI 1999; 2. Kilner, Circulation 1993; 3. Frydrychowicz, JCAT 2007; 4. Glagov, NEJM 1987; 5. Langille, Science 1986; 6. Davies, Physiol Rev 1995; 7. Markl JMRI 2007; 8. Buonocore, MRM 1998

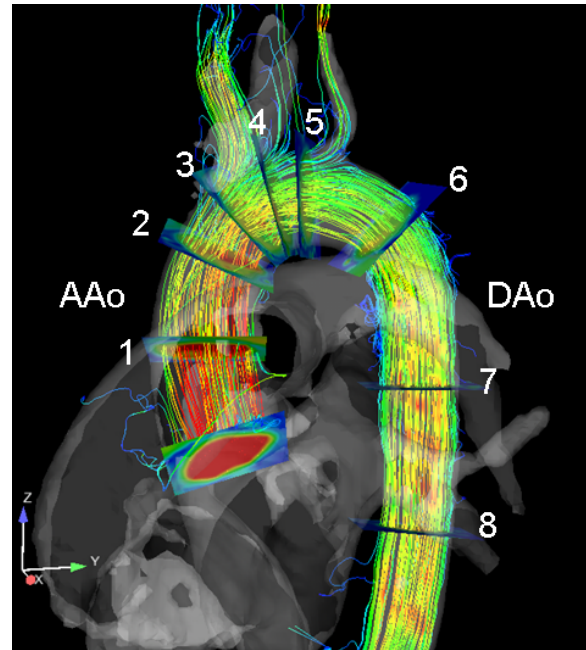


Fig. 1: Streamline visualization of the thoracic aorta at peak systole. Absolute measured velocities are color-coded. 8 cutplanes for visual analysis of hemodynamics were manually placed transecting the aorta.

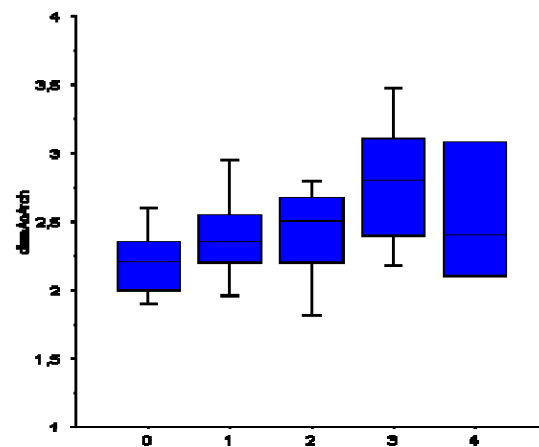


Fig. 2: Diameter-dependency of the number of vortices. Limited by the number of aortae with higher numbers of vortices, there is an overtrend towards higher number of vortices in patients with large AAo diameters.

	AAo	Helicity in % (n)	TTPavg
SAD (n=38)	2.81 ± 0.38cm	75.7% R-handed (28) 5.4% L-handed (2) 18.9% None (7)	131 ± 26ms
LAD (n=24)	3.81 ± 0.39cm	36% R-handed (9) 4% L-handed (1) 60% None (15)	144 ± 34ms

Tab. 1: Summary of results of the small aortic diameter group (SAD) and the group with large aortic diameters (LAD). The averaged TTP did not vary significantly but showed the overall trend towards longer TTP in the LAD group.