4D Flow MRI: Analysis of Aortic Hemodynamics after Valve-Sparing Aortic Root Replacement with an Anatomically Shaped Sinus Prosthesis

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Target audience: MR Physicists; Physicians: Radiologists, Cardiologists, Cardiac and Vascular Surgeons; Biomedical Engineers

Background and Purpose: The DAVID procedure is a standard valve-sparing technique for surgical repair of aortic root aneurysms. Traditionally a straight tubular graft is implanted altering normal root geometry. The aortic roots sinuses, however, are not preserved. Physiologically, vortices forming within the aortic sinuses play an essential role in valve function minimizing leaflet stress[1] and transvalvular pressure gradients[2], and stabilizing leaflets in open position during systole[3]. Hence, the lack of sinuses in tubular grafts results in frequent contact of the valve leaflets with the prosthesis wall[4] and higher leaflet stress.[1, 5] The anatomically shaped sinus prosthesis (Uni-Graft® W SINUS, Braun) with its three separate sinuses promises to preserve aortic root hemodynamics. Therefore, it was the purpose of this work to compare aortic hemodynamics in patients with a physiologically shaped sinus prosthesis (SP) with straight grafts (SG) and healthy volunteers (Vol) by means of 4D Flow MRI.

Methods - MRI scans: 15 patients after David procedure (12 SP [1f, 55±15y], 3 SG [1f, 51±13y]) and 15 age-matched healthy volunteers [13f, 51±11y] were examined at 3T (Philips Achieva) with a 20 channel body surface coil after IRB approval and written informed consent. A retrospectively ECG-gated 4D phase contrast-sequence with adaptive respiratory gating, V_{enc}=180cm/s and an isotropic resolution of 2.6mm, interpolated to 2mm, SENSE (R_{eff} = 2.1) and Cartesian acquisition mode was acquired. Contrast agent (Gadovist®, Bayer HealthCare, 0.1 ml/kg body weight) was given in all patients and 2 volunteers. The cardiac cycle was reconstructed to 20 phases resulting in an effective temporal resolution of 35-61ms depending on each individual's heart rate (49-87/min). Scan time was 13±4min.

Data analysis: Employing GTFlow (v2.1.4, GyroTools, CH) the vessel wall was rendered and blood flow was visualized by streamlines and particle traces color-coded with respect to the acquired velocities. Offline data processing included aliasing correction in 5 datasets using PhaseUnwrappingTool (v1, Fraunhofer MEVIS, GER). Presence and extent of ***sinus vortices*** were graded on a 0-3 scale; the orientation of their rotation axis was recorded as orthogonal or parallel to the vessel wall. ***Secondary flow patterns***[6] (vortices, secondary helices) were assessed in the entire thoracic aorta. Factors known to influence hemodynamics such as ***aortic geometry*** (form[7, 8]: round, gothic, cubic; presence of kinking; diameter) and ejection fraction of the left ventricle (LVEF) were assessed.

Results. In patients with SP ***sinus vortices*** were predominantly medium or maximum (grade 1: 22%, 2: 50%, 3: 28%) compared to small- and medium-sized vortices in volunteers (grade 1: 64%, 2: 33%, 3: 0%), (p<0,05). Although more pronounced, sinus vortices were physiologically configured with a rotation axis aligned parallel to the vessel wall (SP: 83%, Vol: 98%). SG displayed no (22%) or small, abnormally rotated vortices (78%) with a rotation axis orthogonal to the vessel wall (100%).

In the ascending aorta, ***secondary flow patterns*** were considerably more frequent in patients (SP n=1.6±0.8, SG n=1.7±0.6 per subject) than in volunteers (n=0.3±0.5, p<0.05). Almost no secondary flow patterns derived from the aortic arch (SP n=0.3±0.7, SG n=0.0±0.0, VOL n=0.0±0.0, n.s.). In the descending aorta typically a vortex or secondary helix developed in the ductus diverticulum (SP n=1.1±0.3, SG n=1.3±0.6, VOL n=0.9±0.8, n.s.), consistent between groups. There were significant more vortices in patients (SP: n=2.2±0.7, SG: n=2.3±0.6) than in volunteers (n=0.5±0.6, p<0.05) whereas there was no difference in the number of secondary helices (SP: n=0.8±0.8, SG: 0.7±1.2, Vol: n=0.7±0.6).

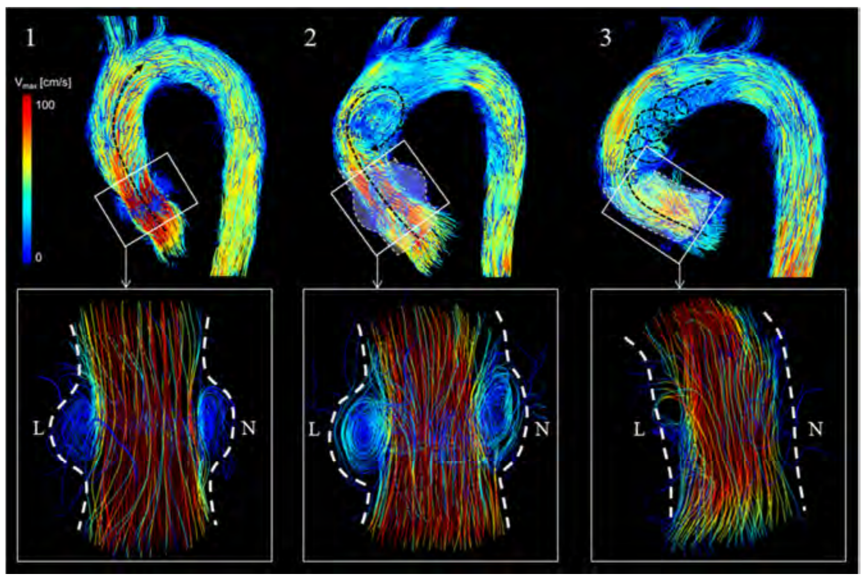

Fig 1. Typical presentation of (1) a volunteer (f, 53y), (2) a patient with sinus prosthesis (m, 60y) and (3) a patient with a straight graft (m, 41y). Upper row shows particle traces, lower row streamlines color-coded according to the acquired flow velocities. Note physiological flow in (1) and near physiological sinus vortices in (2), whereas (2) and (3) demonstrate markedly increased secondary flow patterns in the thoracic aorta.

Volunteers typically presented with a round arch (13/15) whereas patients exhibited mostly cubic and gothic ***geometries*** (SP: 10/12, SG: 3/3). In 14/15 patients there was a kinking of the ascending aorta (kinking of prosthesis itself - SP: 5/12, SG: 1/3; kinking at distal anastomosis - SP: 8/12, SG: 2/3) with at least one secondary flow pattern developing in direct spatial relation. Relative aortic dilatation at the distal anastomosis was observed in every patient (SP: 0.7±0.3cm, SG: 0.7±0.4cm). LVEF of patients (SP: 58±7%, SG: 57±7%) was lower than in volunteers (65±3%, p<0.05).

Discussion and Conclusion. 4D Flow MRI confirms near-physiological hemodynamics in the aortic root in patients with a sinus prosthesis as opposed to straight grafts. In our study cohort, prosthesis implantation is associated with altered aortic geometry and hemodynamics distal to the graft. Confounding factors not statistically tested for in this study may be seen in postprosthetic dilatation as well as altered compliance, which is subject to ongoing analyses. In consequence, curved grafts[9] may overcome these limitations and might prove beneficial to long-term results after graft implantation avoiding vasculopathy.

References. 1. Grande-Allen KJ et al., J Thorac Cardiovasc Surg. 2000: 119(4). **2.** Pisani G et al., J Thorac Cardiovasc Surg. 2013: 145(4). **3.** Caro CG et al.: The Mechanics of the Circulation. Cambridge University Press. 2012. **4.** Fries R et al., J Thorac Cardiovasc Surg. 2006: 132(1). **5.** Beck A et al., J Heart Valve Dis. 2001: 10(1). **6.** Kilner PJ et al., Circulation. 1993: 88(5 Pt 1). **7.** Frydrychowicz A et al., Eur Radiol. 2012: 22(5). **8.** Ou P et al., J Am Coll Cardiol. 2007: 49(8). **9.** Frydrychowicz A et al., Interact Cardiovasc Thorac Surg. 2009: 9(2).