

# Analysis of Aortic Hemodynamics after Treatment for Coarctation Using Flow-Sensitive 4D MRI at 3T

A. Frydrychowicz<sup>1</sup>, D. Hirtler<sup>2</sup>, R. Arnold<sup>2</sup>, A. Berger<sup>1</sup>, A. F. Stalder<sup>1</sup>, J. Bock<sup>1</sup>, A. Harloff<sup>3</sup>, M. Langer<sup>1</sup>, J. Hennig<sup>1</sup>, and M. Markl<sup>1</sup>

<sup>1</sup>Diagnostic Radiology, Medical Physics, University Hospital Freiburg, Freiburg, Germany, <sup>2</sup>Pediatric Cardiology, University Hospital Freiburg, Freiburg, Germany, <sup>3</sup>Neurology, University Hospital Freiburg, Freiburg, Germany

**Introduction:** Aneurysm formation or re-stenosis constitute life-threatening secondary complications after therapy in aortic coarctation which may occur years and decades after initial surgery [1]. The identification of patients at risk for the development of such pathologies is of high interest and requires a detailed understanding of the link between vascular malformation and altered hemodynamics. Since flow-sensitive, time-resolved 3D examinations (flow-sensitive 4D MRI) have become available and enable for a large volumetric coverage [2], alterations in aortic flow patterns can be detected further up- and downstream the aorta. Analysis is not restricted to the pathology as in single-slice 2D acquisitions. Initial results from coarctation and aneurysm formation have recently been reported as case reports [3]. It was the aim of this study to evaluate the hemodynamic alterations in aortic blood flow before (n=2) and after (n=22) coarctation repair by flow-sensitive 4D MRI at 3T in a larger collective of 24 patients and to compare findings to results in 25 volunteers with normal aortic geometry (aortic diameter aorta  $\leq 30$ mm).

**Methods:** 24 patients (16m, 8f; age  $16.8 \pm 7.3$  years;  $56.6 \pm 18.6$  kg BW) were included in the study. Flow-sensitive 4D MRI was performed in 22 patients 11.6  $\pm$  4.7 years after coarctation repair (12 resection end-to-end and end-to-side, 5 Waldhausen-, 2 Vosschulte-procedures, 3 angioplasties). The remaining 2 patients did not yet receive therapy. Experiments were performed on a 3T MR-system (TRIO, Siemens, Germany) after written informed consent. For comparison, data from 25 normotensive volunteers (14m, 11f, age  $38.6 \pm 16.9$  years,  $70.1 \pm 10.8$  kg BW) without geometrical alterations of the aorta from a previous study were included as a reference [4]. Data acquisitions covered the entire thoracic aorta in a sagittal oblique 3D slab and were performed using an eight channel body coil and an rf-spoiled gradient echo sequence with interleaved 3-directional velocity encoding (BW = 450 Hz/pixel, flip angle = 7-15°, TE / TR = 2.6-3.7 ms / 5.1-6.1 ms,  $v_{enc} = 1.5$  m/s, spatial resolution = (2.0-2.9 x 1.6-1.7 x 2.2-3.0) mm<sup>3</sup>, temporal resolution = 40.8-48.8 ms). Measurements were prospectively gated to the ECG cycle and utilized a previously reported adaptive navigator technique to enable free patient breathing during the acquisition. Data analysis included the calculation of a 3D phase contrast (PC) MR angiography from the 4D MR data which was combined with spatially co-registered color-coded 3D blood flow visualization (EnSight, CEI, USA) [5]. In a consensus reading, flow helicity, development of flow vortices (local flow direction changes), existence of retrograde flow and sites of accelerated blood flow were analyzed. Data was evaluated compared to findings of the 25 normal volunteers.

**Results:** Echocardiography in patients revealed a normal EF ( $68.1 \pm 7.2\%$ ) and a regular aortic bulb ( $27.9 \pm 4.6$  mm). The site of the coarctation showed a relative stenosis or post-operative re-stenosis with a diameter of  $13.5 \pm 4.3$  mm (range 6-10 mm) and a post-stenotic dilatation of  $25.0 \pm 10.4$  mm (range 18-37 mm).

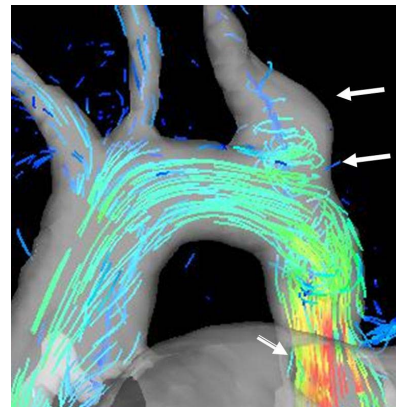
Right-helical systolic aortic blood flow in the ascending aorta (AAo) was seen in all but two patients similar to findings in volunteers (right-handed helix in 21/25 subjects, left-handed helix in 1 subject). Compared to finding in the 25 healthy volunteers, the following specific alterations in blood flow characteristics were found:

- Increased or additional helix formation (overall forward or retrograde circular aortic flow movement inside the entire aortic lumen) in 21/24 patients.
- Clearly detectable flow acceleration across the operated/stenosed localization of the coarctation in all patients irrespective of the level of re-stenosis (see figures 1 and 2).
- Significant ( $p < 0.01$ ) increase in the number of vortices found in the thoracic aorta ( $2.7 \pm 1.0$  (range 1-5) as compared to  $0.8 \pm 1.1$  (range 1-3) in normal controls.
- Vortices in the branch of at least one of the supra-aortic vessels, most often in the left subclavian artery (15/24 patients, see Fig. 1) which were complete absent in volunteers
- Considerable increase of vortex formation in the post-stenotic region in 21/24 patients while similar flow patterns in the DAo were only found in 7/25 healthy controls
- Rarely detectable physiological late-systolic retrograde flow (7/24 patients) while late diastolic retrograde flow in the AAo was present in all 25 normal subjects

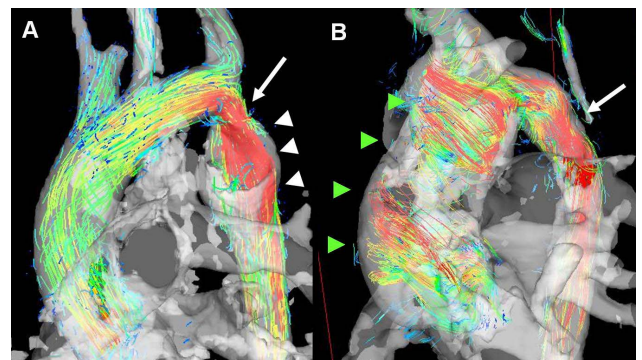
**Discussion:** The results provide insights into the nature of aortic hemodynamics before and after different repair strategies for aortic coarctation. Particularly flow changes not directly associated with the site of the stenosis such as enhanced flow helices and vortices in the subclavian artery were unexpected. These findings underline the complexity of the hemodynamic consequences of the disease. Although the cohort is heterogeneous, there are signs of common impaired flow patterns that may facilitate secondary complications such as aneurysm formation. The development of vortices at the site of the branching of the left subclavian artery observed in large number of patients may result in reduced shear forces at this site which are known to promote endothelial remodeling and thus may facilitate aneurysm formation. Indeed, the observed cohort of patients included patients representing different stages of such aneurysm growths with early onsets of dilatation as in figure 1 and 3A and potentially flow mediated larger aneurysmal structures as in figure 3B. Although a multifactorial etiology of secondary alterations is likely, our findings offer a first hint towards the significance of in-vivo evaluation of blood flow for the diagnosis or follow-up of the development of aortic pathology.

**Acknowledgements:** Deutsche Forschungsgemeinschaft (DFG), Grant # MA 2383/4-1, Bundesministerium für Bildung und Forschung (BMBF), Grant # 01EV0706.

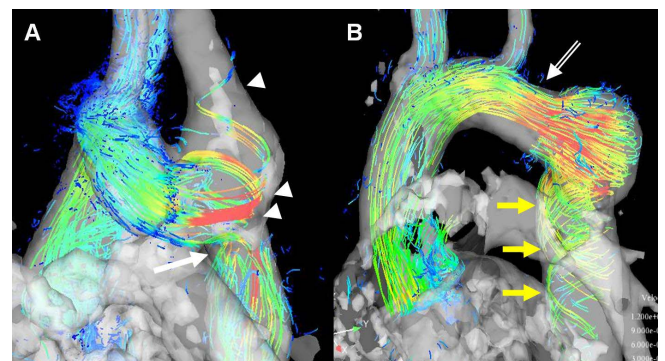
**References:** 1. Oliver JM *J Am Coll Cardiol* 2004; 44:1641-1647. 2. Wigström L, et al. *MRM* 1999;4:793-799. 3. Frydrychowicz A, et al. *J Cardiovasc Magn Reson* 2008;10(1):30. 4. Frydrychowicz A, *ISMRM* 2008. 5. Markl et al, *JMRI* 2007;25:824-831.



**Fig. 1:** 3D-PC-MRA in conjunction with 3D flow visualization in the aortic arch. Vortical flow patterns at the branching site of the left subclavian artery (white arrows) were observed in 15/24 patients. This finding could be observed irrespective of the treatment. The patient also showed typical flow acceleration over a mild re-stenosis (lower white arrow).



**Fig. 2:** Flow patterns in a 35yo man (A) with re-stenosis after treatment and (B) in a 15yo girl, 15 years after end-to-end resection of coarctation. Findings include marked flow acceleration at the location of the coarctation (white arrow), increased helicity (green arrowheads) and a post-stenotic aneurysm (white arrowheads). Of note is that in (B) a vortical flow pattern can be seen in the proximal descending aorta and the subclavian artery.



**Fig. 3:** Different stages of aneurysm development. A: flow patterns in a 10yo boy before treatment of an otherwise not symptomatic coarctation. The elongated and curved aortic arch, the vortical flow acceleration (white arrowheads) before the stenosis (white arrow) can clearly be appreciated. B: Hypoplastic arch (white arrow) gives rise to a flow acceleration that develops into a highly vortical flow through a small aneurysm resulting in substantial helical flow in the DAo (yellow arrows).