# In-vivo Systolic Pressure Gradients across the Aortic Root in Patients with a Physiologically Shaped Sinus Prosthesis and Healthy Volunteers Analyzed by 4D Flow MRI

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

**Purpose.** The implantation of grafts in aortic aneurysm surgery is prone to alteration of hemodynamics with yet unknown long-term consequences. The aortic root sinuses play an essential role in minimizing transvalvular pressure gradients[1] and supporting physiologic aortic valve function[2, 3]. In contrast to conventionally used straight grafts, the anatomically shaped sinus prosthesis (Uni-Graft® W SINUS, Braun) promises to preserve aortic root hemodynamics. Invasive *in-vitro*[4] and non-invasive echocardiography[5] studies affirming near-normal peak and mean pressure gradients (ΔP) across the aortic valve [ΔP between left ventricular outflow tract (LVOT) and ascending aorta] are yet to be confirmed by MRI. 4D phase contrast MRI enables to examine temporally and spatially distributed pressure differences non-invasively *in-vivo* and user-interpedently[6]. It was our goal to i) compare systolic pressure differences (ΔP) in patients with sinus prosthesis (SP) and healthy volunteers (Vol) as well as ΔP derived from MRI and echocardiography in patients and ii) to characterize the temporal course of ΔP.

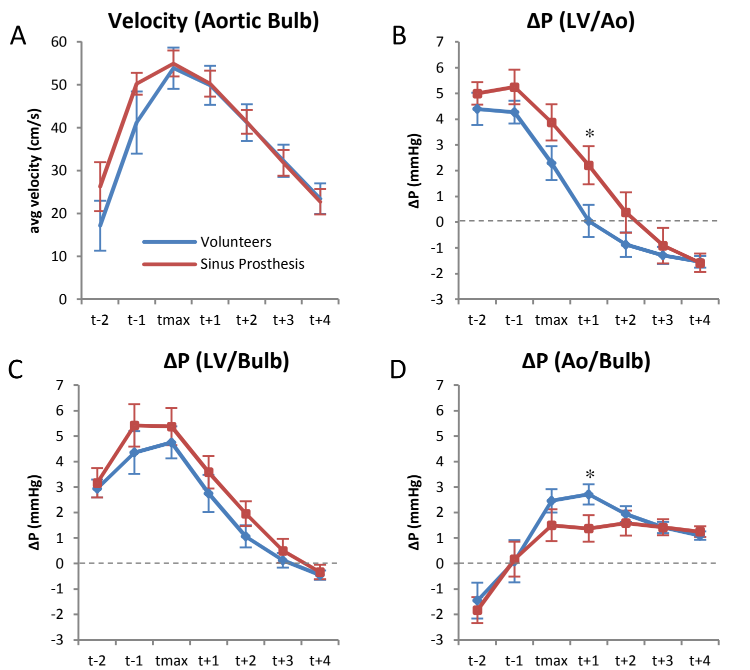


Fig 1. Development of flow velocity (A) and transvalvular pressure gradients (B) with decomposition in its infra- and supravalvular components (C, D) during systole (* significant, α<0.05)

**Methods - MRI scans:** 6 patients with sinus prosthesis (6 male, 55±15y, HR=72±13/min) and 12 age-matched healthy volunteers (1 male, 52±9y, HR=64±10/min) were examined at 3T (Philips Achieva) with a 20 channel body surface coil after IRB approval and written informed consent. Sequence details were: retrospectively ECG-gated 4D phase contrast-sequence, adaptive respiratory gating, $V_{enc}$ = 180 cm/s, acquired isotropic spatial resolution = 2.6 mm interpolated to 2 mm, SENSE ($R_{eff}$ = 2.1). Contrast agent (Gadovist®, Bayer HealthCare, 0.1 ml/kg body weight) was administered prior to the 4D Flow scan in all patients and 2 volunteers. Data were reconstructed to 20 phases resulting in an effective temporal resolution of 35-61 ms depending on each individual's heart rate (49-87/min).

**Echocardiography in patients**. As a secondary endpoint, 4D Flow MRI-derived ΔP were compared to data from echocardiography (Vivid 7, GE) during a follow-up visit in which mean and maximum pressure gradients were estimated by transthoracic echocardiography using the modified Bernoulli equation.

**Data analysis:** Aorta and LVOT were segmented using MEVISFlow (v9, Fraunhofer MEVIS, Bremen, Germany). To calculate relative pressure differences on basis of the acquired time-resolved velocity fields the Navier-Stokes equation was solved assuming blood as a viscous, incompressible fluid[6, 7]. ΔP was evaluated at peak systole ($t_{max}$) and contiguous time points ($t_{max-2}$ to $t_{max+4}$). Three points of reference were placed along the centerline in the aortic bulb (B, vena contracta), left ventricle (LV, 4 cm proximal to B) and ascending aorta (Ao, 4 cm distal to B). 3 pressure gradients were assessed: ΔP(LV,B), ΔP(Ao,B), and the transvalvular pressure gradient ΔP(LV,Ao); the relative pressure in the bulb was set to 0 mmHg.

**Results and Discussion.** Physiological changes of pressure gradients could be confirmed in all individuals. ***Peak pressure gradients across the aortic valve*** [ΔP(LV,Ao)] were 5.5±1.0 mmHg in patients compared to 4.8±0.5 mmHg in volunteers (p = n.s.). There was no significant difference to the peak gradients derived from echocardiography in patients (7.1±2.4mmHg, p = n.s.). In comparison to previous studies with echocardiography[5] and invasive measurements in phantoms[4], 4D Flow underestimates peak pressure gradients across the aortic valve (SP: 8.1±3.6mmHg[5], 8.9±1.1mmHg[4]; Vol: 9.3±2.5mmHg[4]), presumably explained by temporal and spatial resolution and spatiotemporal averaging during MRI acquisitions[6]. Except for $t_{max+1}$ there were no significant differences in pressure gradients between patients and volunteers.

Furthermore, 4D Flow MRI was able to characterize the not entirely intuitive ***temporal evolution of transvalvular ΔP*** consistent with normal pressure gradients[8], e.g. derived from invasive measurements[9, 10]: Expectedly, the transvalvular ΔP from LV to Ao [ΔP(LV,Ao), Fig. 1] is positive in early systole with a maximum just before peak systole promoting LV outflow into the aorta. ΔP(LV,Ao) decreases until it becomes negative in the second half of systole between $t_{max+2}$ and $t_{max+3}$, i.e. aortic pressure exceeds ventricular pressure while blood is still being ejected from the ventricle, a potential function of the Windkessel effect or reflected pressure waves.

As part of the ΔP characterization, the ***Venturi effect***, which describes a reduction of pressure when a fluid passes a constricted section of a pipe could be demonstrated at valve level (Fig 2): At peak systole, results revealed increased relative pressures in LV and Ao as compared to the center of the aortic bulb, the location of the vena contracta, i.e. smallest lumen of the aortic valve orifice. Deducting that valve closure is promoted by the Venturi effect would profit from an in-plane analysis of pressure differences. This analysis, however, was not available at the time of the study and will be limited by the acquired spatial resolution.

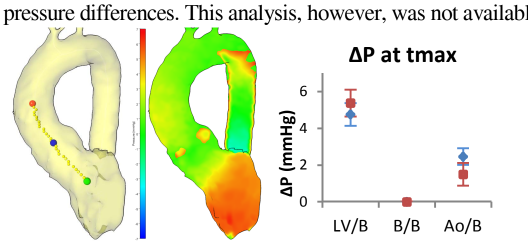


Fig 2. a) Reference points, b) Pressure gradients at peak systole.

**Conclusion.** 4D pressure difference mapping confirmed physiologic pressure differences and their time course across the aortic bulb in healthy volunteers and patients with sinus prostheses. Although an in-plane analysis is lacking, longitudinal pressure differences are in line with the Venturi effect.

**References. 1.** Pisani G et al., ***JTCVS*** 2013: 145(4). **2.** Grande-Allen KJ et al., ***JCTVS*** 2000: 119(4). **3.** Caro CG et al.: ***The Mechanics of the Circulation***. Cambridge University Press. 2012. **4.** Richardt D et al., ***Ann Thorac Surg***. 2010: 89(3). **5.** Schmidtke C et al., ***EJCTS*** 2013: 43(3). **6.** Bock J et al., ***MRM*** 2011: 66(4). **7.** Riesenkampff E et al., ***JACC CV Imaging***. 2014: 7(9). **8.** Mitchell JR and JJ Wang, ***Adv Physiol Educ.*** 2014: 38(2). **9.** Spencer MP and FC Greiss, ***Circ Res.*** 1962: 10(3). **10.** Driscol TE and RW Eckstein, ***Am J Physiol.*** 1965: 209(3).