

Aortic Stiffness following treatment of Severe Aortic Stenosis

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Background: Arterial stiffness is an independent predictor of cardiovascular mortality and can be assessed by CMR measurement of aortic distensibility (AD) and pulse wave velocity (PWV).

Aim: To determine AD and PWV in patients with severe symptomatic aortic stenosis and identify changes following TAVI compared to SAVR.

Methods: Forty-eight patients underwent identical paired 1.5T CMR scans (Intera, Philips or Avanto, Siemens). Brachial artery blood pressure was recorded by Dinamap (Critikon, Tampa, FL, USA). Multi-phase steady state free precession (SSFP) cine imaging (50 phases) was acquired in a axial plane to the thoracic aorta at the level of the pulmonary artery bifurcation (Ascending aorta (AA) and proximal descending aorta (PDA)).

AD (mmHg^{-1}) was calculated following the contouring of the aortic region of interest, Figure 1A (QMass V7.5, Medis, The Netherlands), using the equation; $(\text{Aortic}_{\text{max}} \text{ lumen area} - \text{Aortic}_{\text{min}} \text{ lumen area}) / (\text{Aortic}_{\text{min}} \text{ lumen area} \times [\text{Systolic BP} - \text{Diastolic BP}])$.

Aortic PWV was assessed using identical planning with retrospectively gated, through plane, phase-contrast velocity encoded images (breath-hold, single slice, 10mm thick, 40 phases, typical FOV 350, RFOV 85, VENC 200cm/s). Offline analysis was performed using previously published algorithms implemented in the in-house software PMI 0.4. The velocity encoded images of the AA and PDA were manually contoured to derive velocity-time curves. The distance (mm) between AA and PDA was measured manually from in-plane sagittal-oblique SSFP images of the aortic arch. PWV (m/s) was calculated using the transit-time method (foot-foot delay, Figure 1B).

Results: 27 SAVR patients (age 71.8 ± 7.0 years, 75% male, EuroSCORE II $1.43 \pm 0.44\%$) and 21 TAVI patients (age 81.7 ± 6.3 years, 52% male, EuroSCORE II $6.32 \pm 5.99\%$) were studied before and 6 months following valve replacement. Arterial pulse pressure significantly increased following SAVR (57 ± 19.6 vs. 63 ± 14.6 mmHg, $p < 0.05$) but not after TAVI (68 ± 24.0 vs. 67 ± 21.6 mmHg, $p = 0.91$). AD significantly decreased post SAVR (2.00 ± 1.57 vs. $1.39 \pm 0.69 \times 10^{-3} \text{mmHg}^{-1}$, $p < 0.05$) whereas there was no change observed in the TAVI group (1.68 ± 0.80 vs. $1.76 \pm 0.85 \times 10^{-3} \text{mmHg}^{-1}$, $p = 0.74$).

PWV significantly increased post-SAVR (6.69 ± 5.12 vs. $12.13 \pm 6.22 \text{ms}^{-1}$, $p = 0.01$) whereas there was no change observed in the TAVI group (9.91 ± 9.32 vs. $12.42 \pm 9.24 \text{ms}^{-1}$, $p = 0.23$).

Conclusions: In patients with severe aortic stenosis, SAVR but not TAVI is associated with a significant increase in PWV and decrease in AD at 6 months. This increase in aortic stiffness may be a consequence of the different techniques of valve replacement and has potential long-term implications on cardiovascular mortality.

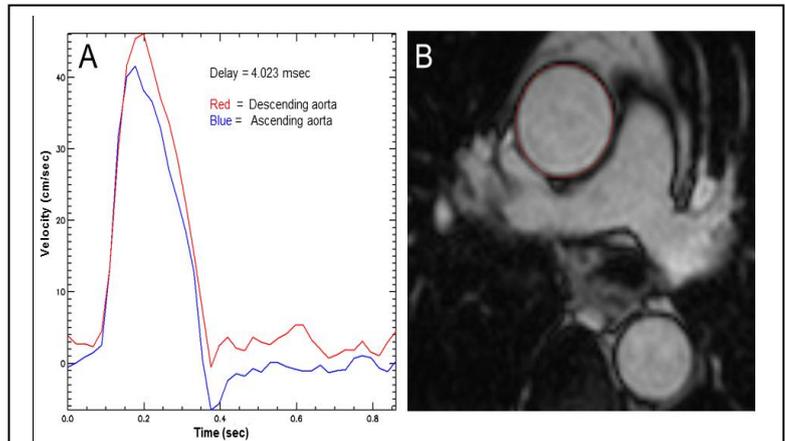


Figure 1: A) Time-Velocity curve derived using PMI software to calculate foot-foot delay (curves are automatically adjusted to accommodate time delay).

B) Aortic cross sectional measurements made by manual planimetry of the ascending aortic endovascular-blood pool interface at minimal and maximal distention.

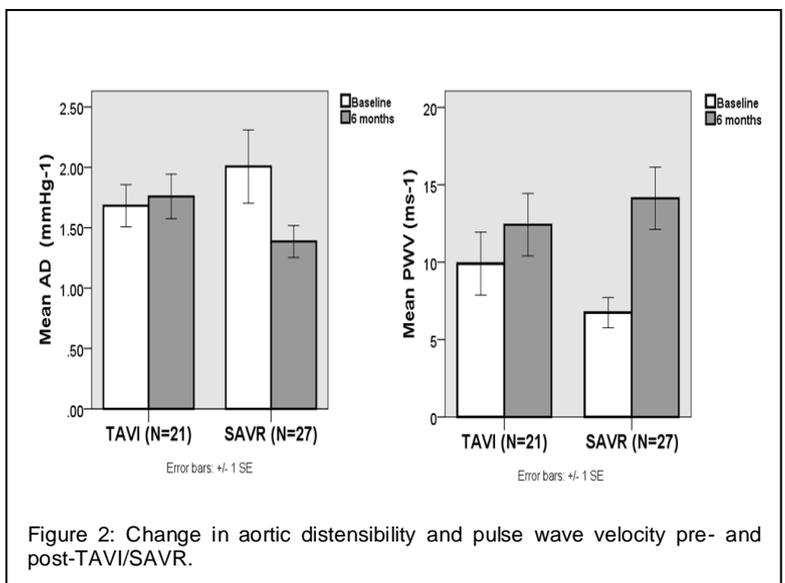


Figure 2: Change in aortic distensibility and pulse wave velocity pre- and post-TAVI/SAVR.