Pressure and volume unloading with pulsatile circulatory support during high-risk percutaneous revascularization

B. Bastos M.[1], JJ. Schreuder[1], J. Daemen[1], CA. Den Uil[1,2], NM. Van Mieghem[1]
(1) Erasmus Medical Center, Interventional Cardiology, Rotterdam, Netherlands (2) Erasmus Medical Center, Department of Intensive Care Medicine, Rotterdam, Netherlands

Background: Percutaneous Mechanical Circulatory Support (MCS) may protect the myocardium and reduce the risk of major adverse events. Clinical data on Left Ventricular (LV) unloading by pneumatically driven percutaneous devices is currently scarce.

Hypothesis: describe the unloading pattern produced by a pulsatile MCS on the LV.

Methods: an 80 years old male with COPD with ischemic cardiomyopathy was admitted with acute coronary syndrome with pathological Q waves in the inferior leads. TTE evidenced infero-posterior akinesia with 30% Ejection Fraction. Coronary angiography showed CTO on the RCA, 90% stenosis on the LM, 90% on the LAD and 90% also on the LCX. The case was considered non-suitable for surgical revascularization. MCS was indicated by heart team consensus with PulseCath iVAC2L™ (PulseCath BV, Amsterdam, The Netherlands). Stents were deployed on the LCX, LAD, and LM. Hemodynamical data was collected with pulmonary catheterization and a conductance catheter in the LV.

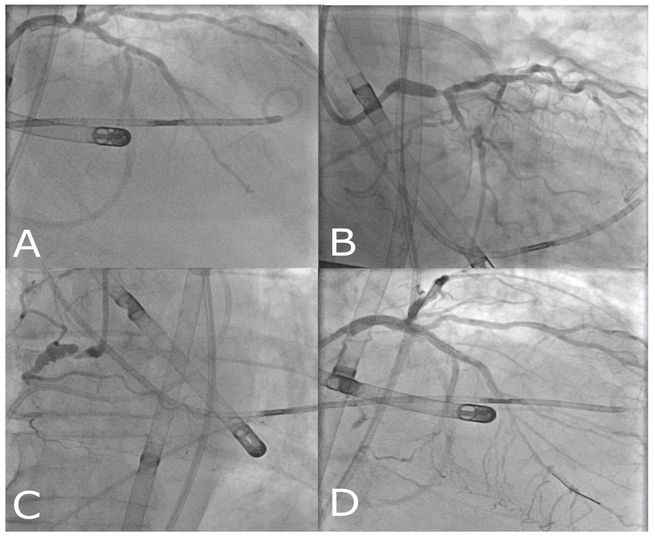

Figure 1: (A) Angiographic projection showing severe stenosis in the Left Main (LM) and Left Anterior Descending (LAD). (B) Severe stenosis in the Left Circumflex (LCX) and LM. The LAD shows diffuse disease and irregularities. (C) Chronic total occlusion in the Right Coronary Artery (RCA). (D) Post-procedural Left Anterior Oblique projection showing the revascularized LAD and LCX.

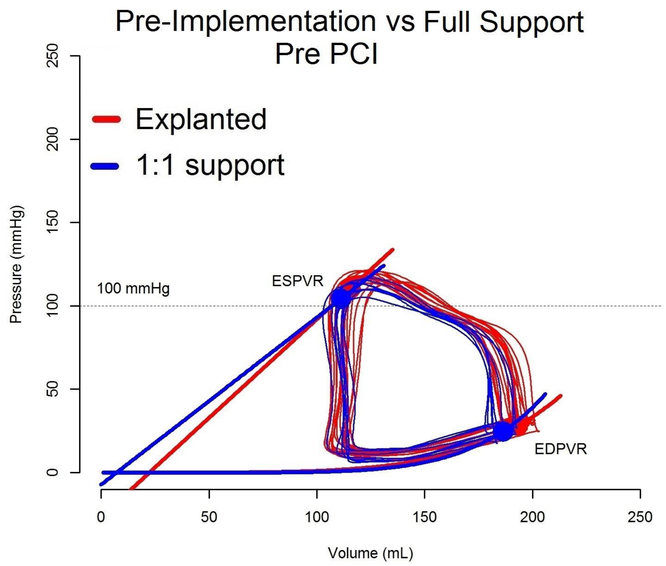

Figure 2: Sets of pressure-volume loops measured before the PCI. The loops show the moments prior to implantation of iVAC2L (red) and after a short period of 1:1 support (blue), indicating nearly unchanged ESPVR and EDPVR. The loops show incipient shifting to the left and downward.

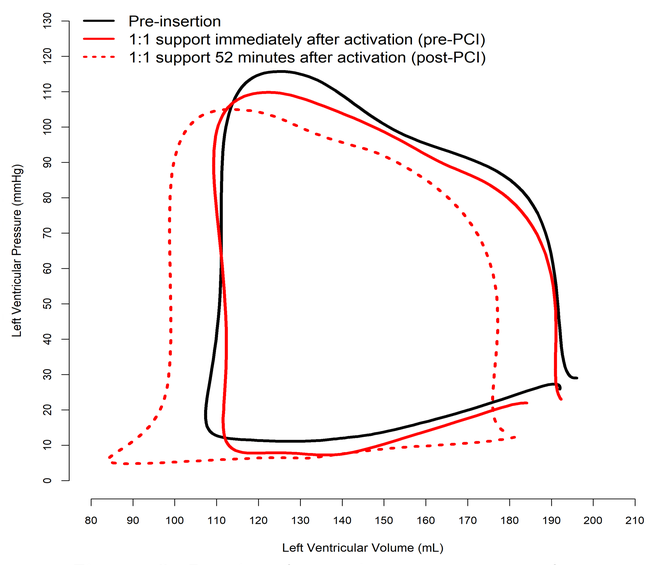

Figure 3: Pre-implantation pressure-volume loops recorded at baseline (pre-insertion), immediately after activation and after the PCI with 52 minutes post-activation. Note progressive shift to the left and downwards.

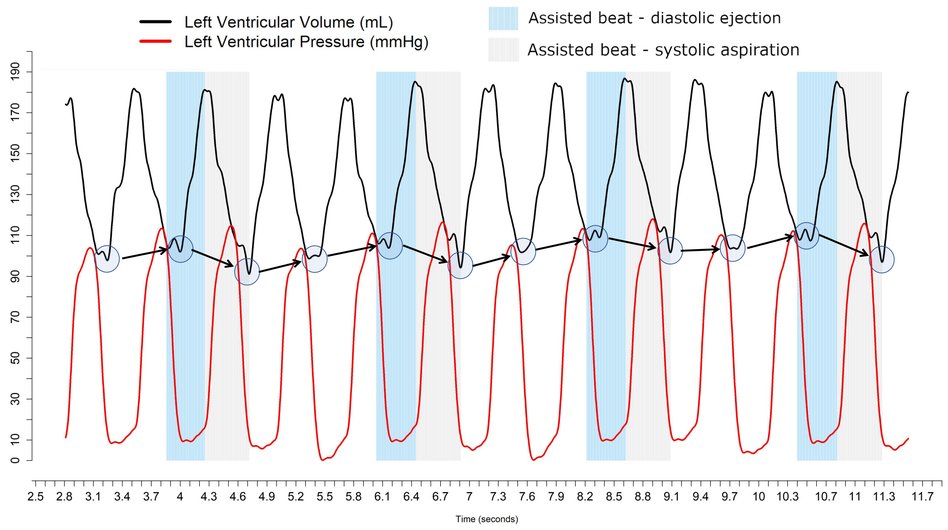

Figure 4: Left ventricular pressure and volume waveforms plotted against time during 1:3 support demonstrating reduced ESV after iVAC aspirates blood in assisted systoles. Progressive increase in ESV and ESP is observed in the two unassisted beats when iVAC is inactive (circles and arrows).

Results: Under 1:1 and 1:3 assist, the level of support was 1.36 and 0.62L/min respectively. After activating iVAC2L, the PV loops were shifted to the left. The pressure-volume Area (PVA: 11504.05±383.36 vs 11251±572mmHg.mL) decreased and also Stroke Work (SW: 6598±191 vs 5931±489mmHg.mL). Pressures and volumes decreased at end-systole (ESP: 110.44±2.39 vs 104.38±3.66mmHg; ESV: 115.50±4.97 vs 111.25±3.41mL) and end-diastole (EDP: 28.12±2.62 vs 24.77±3.90mmHg; EDV: 193.25±5.15 vs 186.23±4.76mL). Contractility was mostly unchanged (End-systolic Elastance, Ees: 1.19±0.05 vs 1.00±0.13mmHg/mL; V100: 106.73±6.00 vs 107.22±4.75mL; +dP/dtmax: 835.31±29.90 vs 849.69±48.37mmHg.s-1), and also lusitropy (Tau: 41.50±3.23 vs 41.50±3.67ms; β: 5.28±0.88 vs 5.36±1.70; V30: 192.84±9.44 vs 195.19±11.62mL). The afterload improved (Effective Arterial Elastance, Ea: 1.51±0.07 vs 1.46±0.10mmHg/mL; End-systolic Wall Stress, WSes: 110.62±2.39 vs 104.54±3.67mmHg) as well.

<u>Conclusion</u>: MCS unloaded the LV and optimized the PVA, with positive effects also over the afterload. Further insights will be released in the PULSE trial (Clinicaltrials.gov NCT03200990).

Disclosure: B. Bastos M. works for PulseCath B.V. and is Affiliated Researcher at the Erasmus MC. JJ. Schreuder works for CD Leycom and is Affiliated Researcher at the Erasmus MC. NM. Van Mieghem is Clinical Advisor for PulseCath B.V. The other authors have nothing to disclose