

LAGRANGIAN HEMOLYSIS MODELING IN PATIENT-SPECIFIC AORTIC BLOOD FLOW ALTERED BY AORTIC VALVE STENOSIS

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Introduction

The presence of aortic valve stenosis (AS) leads to an alteration of supra-avalvular flow patterns. These pathological hemodynamics are hypothesized to induce elevated membrane stresses on red blood cells (RBCs). In order to elucidate the time and location of highest damage induced by AS, a Lagrangian-based blood damage analysis, commonly carried out for blood-contacting medical devices, was implemented in patient-specific 4D Flow MRI-based CFD models for a group ($n = 3$) of healthy subjects and a group ($n = 3$) of patients suffering from severe AS.

Methods

Computational models of subject-specific aortic geometries were created using in-vivo medical imaging data. Temporally and spatially resolved boundary conditions taken from 4D Flow MRI measurements were implemented and particles seeded at the aortic orifice inlet throughout the cardiac cycle. After validating the in-silico results with the in-vivo measurements, the occurring RBC damage was quantified for both the pathological and physiological flows using established Lagrangian power-law formulations. Here, the acting shear stresses on individual particle tracks representing the movement of RBCs through the aortic arch were integrated over time. These insights were used to determine the extent and hemodynamic cause of flow-induced cell damage in AS.

Results

The overall feasibility of the 4D Flow MRI-based CFD simulation was proven with excellent agreement between the in-vivo and in-silico velocity fields on cross-sectional planes throughout the aortic arch and an overall correlation coefficient R^2 of 0.9 (Figure 1).

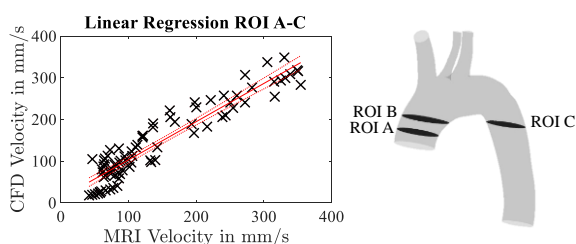


Figure 1: Linear correlation between MRI and CFD velocities on ROI A-C for an AS case.

The subsequent hemolysis analysis showed elevated exposure times and shear stress values on RBCs in the AS cohort, compared to the healthy group. This also resulted in higher hemolysis values, as shown in Figure 2. Further, the mean exposure time of erythrocytes in physiological aortic flow is marked in red, as reference. This underlines the longer exposure times of RBCs during AS caused by highly disturbed blood flow.

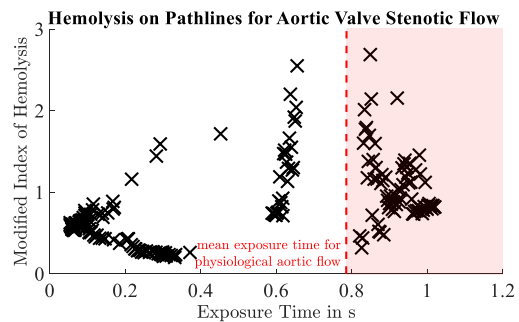


Figure 2: Hemolysis values for a selection of aortic stenotic pathlines and their corresponding exposure duration. The index of hemolysis is multiplied by 10^7 to aid the visualization (MIH).

Finally, the time and location of highest damage were identified to occur during late systole/deceleration, in the pathological turbulent flow regions within the bulk flow of the ascending aorta.

Discussion

In summary, a methodology to generate a 4D Flow MRI-based numerical fluid simulation model of AS flow within the aortic arch was developed and validated. Turbulent flow features within the free stream of the decelerating ascending aortic jet have been identified as the most prominent contributors towards RBC damage. Future work will validate the results in a larger cohort size and investigate the potential restoration of physiological flow structures through clinical interventions.

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