A FLUID-STRUCTURE INTERACTION SIMULATION FRAMEWORK TO DISTINGUISH BETWEEN TRUE AND PSEUDO-SEVERE AORTIC STENOSIS

Sabine Verstraeten (1), Martijn Hoeijmakers (2), Frans van de Vosse (1), Wouter Huberts (1)

1. Eindhoven University of Technology, The Netherlands; 2. ANSYS, The Netherlands

Introduction

Aortic stenosis (AS) is a pathophysiological narrowing of the aortic valve, leading to symptoms such as chest pain, fainting and even heart failure. Valve replacement therapy is recommended for patients with severe AS, i.e., a peak aortic jet velocity ≥ 4 m/s, a mean transvalvular pressure drop > 40 mmHg, and an aortic valve area (AVA) < 1 cm^2 [1]. However, 50% of the patients [2] have a small AVA (< 1 cm^2) together with a small pressure drop (< 40 mmHg). In these cases, the aortic stenosis might be the reason for patient's complaints but the underlying cause might also be related to the inability of the heart to generate a sufficient high cardiac output flow. This low flow leads to a low pressure drop that prevents the aortic valve from fully opening. To accurately diagnose stenosis severity in these cases, dobutamine is administered to increase heart rate, left ventricular contractility, and valvular flow. If the AVA remains small, but the pressure drop exceeds 40 mmHg, it is classified as a true severe AS, and valve replacement is recommended. If the AVA now exceeds 1 cm^2 and the pressure drop remains small, it is a pseudo-severe AS, and valve replacement will not be effective. Unfortunately, for 30% of the low gradient AS patients this diagnostic approach is not possible because the flow increases insufficiently upon Dobutamine [2]. Patient-specific fluid-structure interaction (FSI) simulations of the aortic valve [3] can improve the diagnosis as it can impose a higher flow independent of the patient's cardiac function. In this study, we aim to create a modelling framework that ultimately can be used for such FSI evaluations.

Methods

The modelling framework must be able to: (1) generate a volume mesh of a patient-specific valve geometry; (2) handle contact between the leaflets; (3) handle pressure inflow and outflow boundary conditions. The framework was set up in LS-DYNA, using a generic aortic valve geometry from *dynaexamples.com.* The volume mesh was automatically generated by the fluid solver, and updated each iteration accoring to the Arbitrary Lagrangian Eulerian approach to deal with mesh movement (Requirement 1). To prevent the leaflets from intersecting each other, a single-surface, penalty based contact algorithm was used (Requirement 2). Typical, left ventricular and aorta pressure signals were prescribed at, respectively, the inflow and outflow boundaries of the fluid domain (Requirement 3). The rest of the framework's settings are summarized in Figure 1.

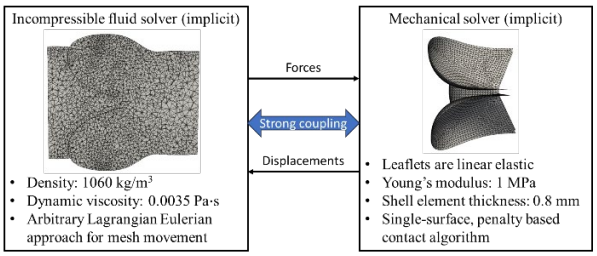

Figure 1: Settings of the FSI simulation framework.

Results

The presented framework was implemented successfully, and simulations of a healthy aortic valve were done. Three snapshots of the simulated cardiac cycle are shown in Figure 2. The maximum velocity during systole was around 2 m/s, and the mean transvalvular pressure drop was 4 mmHg, consistent with healthy individuals [2].

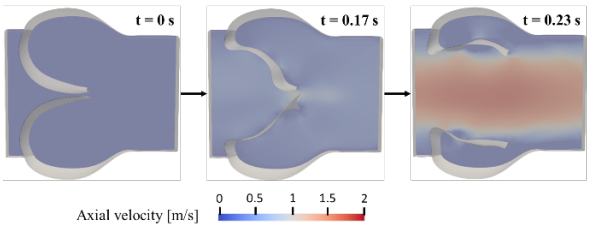

Figure 2: Snapshots at three different moments in the cardiac cycle. The colors indicate the fluid velocity in axial direction, on the mid plane of the geometry.

Discussion

The presented framework fulfills the three requirements defined and allows for effectively simulating valve dynamics, providing detailed fluid dynamic information throughout the cardiac cycle. The model framework is now ready for applying it to patient-specific geometries with corresponding boundary conditions, and establishing a method to estimate patient-specific material properties. Future work focuses on simulating dobutamine responses by prescribing pressure signals with a higher frequency and amplitude, ultimately leading to a diagnostic tool for distinguishing between true and pseudo-severe AS.

References

1. Nishimura et al., Circulation, 129.23: e521-e643, 2014.
2. Clavel et al., Eur. Heart J. 37.34: 2645-2657, 2016.
3. Govindarajan et al., Ann Biomed. Eng.: 1-11, 2023.

Acknowledgements

We acknowledge the European Union's Horizon 2020 research and innovation programme (grant agreement No 101017578) for their financial support.